TEXT-BOOK

OF

OPHTHALMOLOGY

BY

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AUTHORIZED TRANSLATION FROM THE TWELFTH GERMAN EDITION; COMPLETELY REVISED AND RESET, WITH NUMEROUS ADDITIONS SPECIALLY SUPPLIED BY THE AUTHOR AND OTHERWISE MUCH ENLARGED

BY

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WITH FOUR HUNDRED AND FIFTY-FIVE ILLUSTRATIONS

SEVENTH EDITION REVISED AND RESET

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PREFACE TO THE SEVENTH EDITION

The book has been entirely rewritten and rearranged. In the rearrangement the attempt has been made to follow a logical order, so that the relations between the separate parts of the subject shall be made most clear. Thus ptosis and the motor affections of the iris have been placed under motor anomalies instead of under diseases of the lids and iris; hemianopsia has been placed under disorders of the visual field instead of under diseases of the optic nerve, etc. In the textual changes made, the translator has had the benefit of consultation with Dr. Fuchs and of comparison with the thoroughly revised 13th German edition just prepared by Professor Salzmann. In many places he has drawn on the latter. When he has done so to any great extent the fact is indicated. In other regards the translator assumes the responsibility for the very numerous changes made—changes which are found on practically every page. These include, first, changes in the sections on the anatomy of the eye which are now grouped together, so as to form the initial part of the book. The succeeding portions on the development and physiology of the eye have also received considerable additions and changes.

The chapters on refraction have been entirely remodelled and much augmented. Considerable changes also have been made in the sections on motility and various alterations and additions in the sections on the light sense, color sense, perimetry and functional and objective examination—the attempt having been made to bring these subjects down to date in all particulars. Among the subjects added in these special parts of the book are a fuller discussion of heredity, Von der Hoeve's theory of the origin of cataract and senile macular degeneration, the newer applications of heat and radiotherapy, massage, and hypertonic solutions; notions of the wave theory of light, the theory of prisms, the composition of lenses from prisms, the laws governing the formation of images, Gullstrand's observations on the character of the images in astigmatism and the structure of the lens, the cardinal points of lenses and the eye, a new table of accommodation, the evolution of ametropia, and a more extended consideration of the determination and treatment of refractive errors; complete revision of the sections on color vision, with many additions on the physical basis of color vision, color mixtures, and perversions of color sense; revision of the section on the light sense including Percival's method of light-sense testing; numerous additions on the subject of perimetry (especially the perimetry of glaucoma); complete revision of the sections on fixation and binocular vision with many other changes in the sections on motility; a much fuller presentation of the paths of the pupillary reflexes and of the visual paths, considerable revision and additions to the section treating of the methods

of objective examination including paragraphs on the slit lamp, contact illumination, examination with red-free light, etc.

In the part treating of special diseases almost every page has been revised. Some of the more considerable changes will be found in the articles on trachoma, inclusion conjunctivitis, ulcerative keratitis, ulcus serpens, pannus degenerativus, corneal and scleral ectasiæ, anomalies of the anterior chamber and of the vitreous, the classification of iritis, the description of the varieties of chorioiditis and retinitis, the theory and treatment of glaucoma, and in all parts in the matter of treatment. The part on operations has also been largely revised, the specific additions comprising, among others Francis' method of removing superficial tumors, Wheeler's operation for a new socket and for blepharoplasty, a variety of the other plastic operations on the lids, some of the newer operations for keratoconus and staphyloma, a much fuller description of trephining, the indications for the glaucoma operations, sclerectomy for detachment, fuller description of the Reese resection, Wootton's advancement, the indications for muscle operations, fuller descriptions of the substitutes for enucleation. A number of new illustrations have been added. With all these changes the attempt has been made throughout to preserve the spirit and wherever possible the words of Dr. Fuchs, whose genius gave the work an impress quite unique and whose unerring judgment make it remarkable alike for the breadth and depth of its information and for the balance and co-ordination of its separate parts.

ALEXANDER DUANE.

PREFACE TO THE FIFTH EDITION

In preparing the present edition, which is in several senses a new work, the translator has been placed in a position of peculiar responsibility and difficulty. No new German edition has been issued and none is at present contemplated. When, however, Hofrat Fuchs learned that another American edition was projected, he not only gave his permission for the insertion of such additions as in the translator's judgment might seem desirable, but also with characteristic kindness himself supplied notes of many additions and changes. These, therefore, as well as the many alterations made by the translator, are not contained in any German edition.

The translator with some diffidence has made a number of radical alterations in the arrangement of the text, which, he believes, will make the book more serviceable as a work of reference. The many pages of remarks, in fine print, which were massed as an appendix at the end of chapters or major divisions, and whose considerable value and interest were somewhat obscured by this arrangement, he has split into shorter sections, each placed in direct juxtaposition to the portion of the text with which it was related. Some of the more important items in the fine print he has transferred bodily to the text. He has also transferred to Part V, on operations, the descriptions of various operative methods previously scattered through other portions of the book.

These changes had the approval of the distinguished author. With regard to other changes, however, owing to the difficulties in correspondence entailed by the war, consultation with him was impossible, and these changes the translator has made quite on his own responsibility. In the chapter on motility he has entirely rewritten the section on the diagnosis of ocular paralyses, and in the articles on heterophoria, squint, and nystagmus has consolidated with the original text the matter which in the former edition he had himself added as an appendix. While, as always, the fact that matter is thus added or radically altered is indicated by the use of brackets and the initial D, so that there should be no misapprehension as to the authorship, the translator wishes in justice to the original author to make clear the fact that the latter is not responsible for these changes nor for any errors that may unwittingly have been made.

The progress of ophthalmology has necessitated numerous and important changes in all parts of the book. Probably of most importance to the American reader are the additions in the chapters on glaucoma, diseases of the retina, and disturbances of motility, and in the sections on refraction, accommodation, and operations. The latter section has not only been rearranged, but considerably added to. Among the many additions

scattered through all parts of the book, may be mentioned the remarks on tuberculin and vaccine therapy, the visual field and color testing, the mapping of scotomata and the blind spot, squirrel plague and eel's blood conjunctivitis, Samoan conjunctivitis, peculiarities of conjunctivitis in the Near East, extragenital gonococcus infection, inclusion blennorrhœa, the etiology of trachoma, blastomycetic dermatitis, superficial linear keratitis, sclerosis of the cornea, the etiology of iritis, sclerosis of the chorioid, suppurative chorioiditis, Elliot's summary of glaucoma theories, retinitis stellata, retinitis exudativa and angiomatosis retinæ, the different forms of retinal degeneration, the varieties of accommodative troubles other than paralysis, and the newer operations.

The translator trusts that these changes, so largely made on his own responsibility, will not in any way serve to detract from the many excellencies of a book which, because of its author's unrivalled experience, knowledge, and judgment, has for twenty-seven years remained a model of its kind.

Alexander Duane.

139 East Thirty-Seventh Street, New York,
March 16, 1917.

PREFACE TO THE FOURTH EDITION

THE translation now presented to the reader has been made from the twelfth edition of Professor Fuchs's celebrated book. Besides the numerous additions and corrections which Dr. Fuchs has inserted in all parts of the book and a number of new illustrations, there has been added a whole new part constituting a general introduction to the work. This valuable addition, comprising some sixty pages of matter, which is nearly all entirely new, considers in an eminently lucid and conservative way the general physiology of the eye, and the pathology, etiology, symptomatology, and treatment of eye diseases as a whole. The helpfulness of this feature of the book in introducing and elucidating the portions which follow and which relate to the diagnosis, pathology, and treatment of the special conditions in turn, will, we believe, be much appreciated by both the student of ophthalmology and the general practitioner.

The present edition, like the previous ones, contains both in text and illustrations numerous additions by the translator. These will be found mainly in the sections on functional examinations, motor anomalies, refraction, and operations. For these insertions (distinguished by being inclosed in brackets and also, when of any length, by being signed with the initial D), the translator is wholly responsible.

In view of the favorable reception accorded to the previous American editions of Professor Fuchs's work in this country, the translator has little hesitation in offering still another, particularly as the German edition upon which it is based represents an essentially improved form of a work which has already proved to be of value to so many—and of a work, moreover, which in matter, scope, and treatment contains so much that appeals both to the professed ophthalmologist and to the general practitioner.

ALEXANDER DUANE.

139 EAST THIRTY-SEVENTH STREET, NEW YORK,
June 1, 1911.

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PART I

ANATOMY OF THE EYE

ANATOMY OF THE EYE

1. In many unicellular organisms—animals and plants, bacteria and protozoa—sensitiveness to light is a property of the body as a whole. In the more highly organized, multicellular animals, sensitiveness to light is no longer a function of the whole body, but is taken over by certain cells, which, in contradistinction to the rest, have retained the property of reacting to light. In its simplest form a visual organ of this sort consists of an epithelial cell connected with a nerve fibre. The epithelial cell has the power of transforming light into another form of energy of such a character that it can be conducted along the nerve fibre to a central nerve organ. Even the eye of man is essentially reducible to the same type of apparatus, which, however, here is developed to the highest perfection. For here, instead of a single sensory epithelial cell, there are an enormous number of such cells, which are united to form the retina and optic nerve. This is the essential part of the eye, and with its formation, too, the development of the eye begins (outgrowth from the rudimentary brain of the primary optic vesicle, which gives rise to the retina). All the other parts of the eye develop later and are designed for protection, for nutrition, or for optical purposes. The exterior fibrous envelope of the eyeball—cornea and sclera—serves to protect the delicate retina; the uvea subserves nutrition; and all of these, together with the lens, help to make a better optical apparatus—the cornea and the lens by the way in which they refract light, and the uvea because of its light-regulating diaphragm, the iris, and because of its dark pigment. To these are added the adnexa of the eye—the lids and lacrimal organs—by which the cornea is sheltered and kept moist so that it shall suffer no impairment of the smoothness and transparency essential for the performance of its optical function—and the muscles and other structures lodged in the orbit, which support the eye and move it in conjunction with its fellow.

TEXT-BOOK

OF

OPHTHALMOLOGY

CHAPTER I

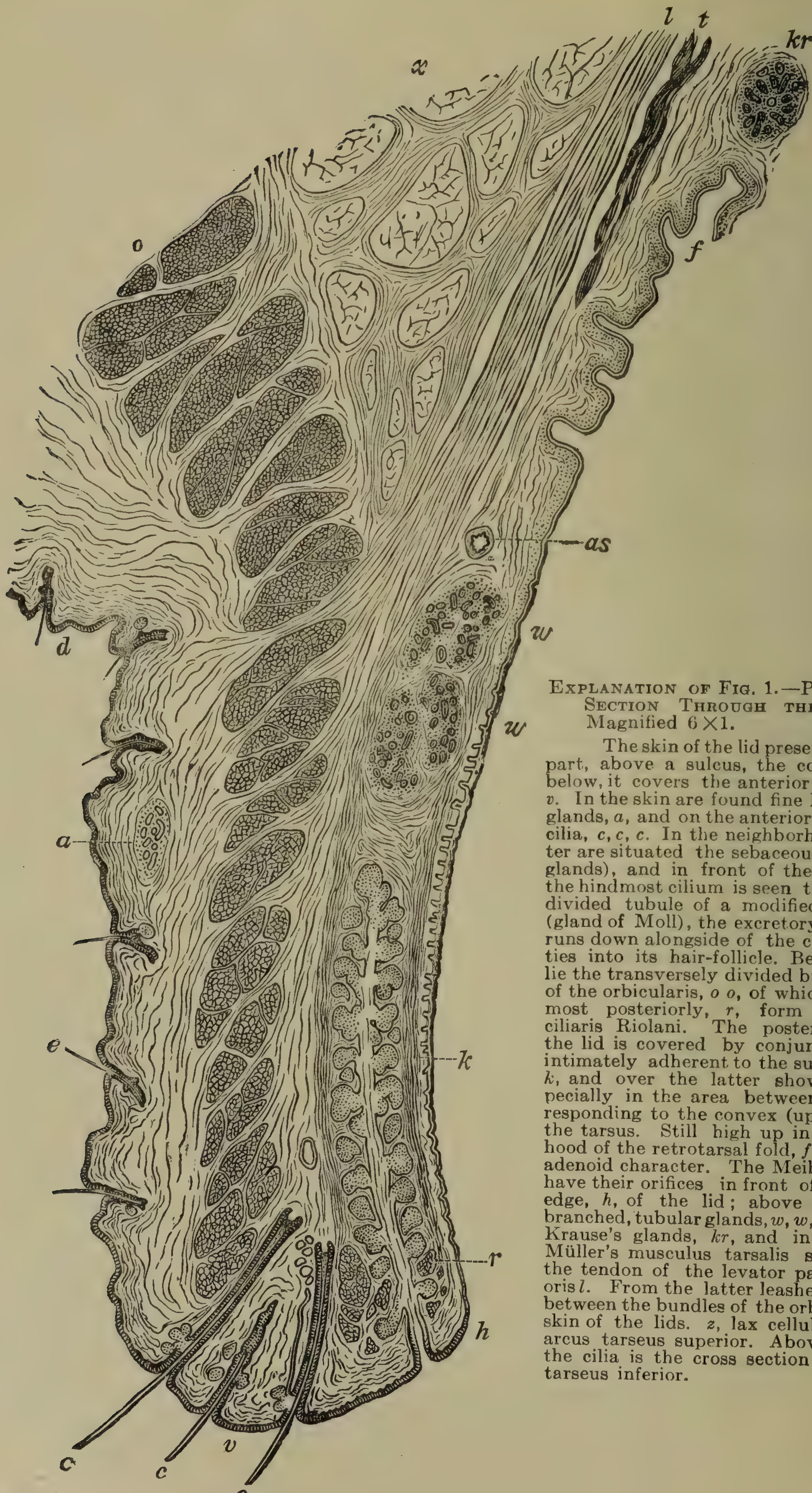
PROTECTIVE ORGANS (ADNEXA) OF THE EYE

I. Lids

2. **Anatomy.**—The lids (palpebræ[1]) are, in origin, folds of the external skin, which push their way over the eyeball to cover and protect it. The boundaries of the upper lid are formed by the eyebrow (supercilium), but the lower lid passes without any sharp line of demarcation into the cheek. The lids bound the palpebral fissure, at the two extremities of which (the angles of the eye) they unite. The external angle of the eye (canthus externus) runs out to a sharp point; when the lids are drawn apart there is put upon the stretch a delicate reduplication of skin (the external commissure), connecting the upper and lower lids in this situation. The inner angle of the eye, on the contrary, presents a horse-shoe shaped notch, at the bottom of which lies the caruncle (see § 9). The mean width of aperture of the palpebral fissure varies with the individual. On an average the fissure opens so far that, with the ordinary way of looking, the upper lid overlaps the upper part of the cornea, while the lower lid leaves the lower margin of the cornea free. The shape and width of the palpebral fissure are of the greatest influence upon the expression of the eye. Eyes which have the reputation of being large and beautiful are generally not really large eyeballs, but eyes with a wide-open palpebral fissure. So, too, the statement that "the eye is small" means for the most part simply that the palpebral fissure is less wide open than usual.

The skin covering the lids is about the thinnest in the human body. As, moreover, it is but very loosely attached to its bed through the medium of a lax and non-fatty connective tissue, it can very readily be made to shift its position. For the same reason it can readily wrinkle up and stretch out again, as the lids open and shut. In old people it is thrown into numerous wrinkles. Because of the ease with which it is displaced, it is readily distorted by scars in its vicinity, so that ectropion cicatriciale is produced. So, too, on account of the laxity of its attachment, it is very apt to be affected by extensive ecchymoses and œdema. It is only in the neighborhood

[1] From *palpare*, to stroke.



EXPLANATION OF FIG. 1.—PERPENDICULAR SECTION THROUGH THE UPPER LID. Magnified 6×1.

The skin of the lid presents in the upper part, above a sulcus, the covering fold, *d;* below, it covers the anterior edge of the lid, *v*. In the skin are found fine hairs, *e e*, sweat glands, *a*, and on the anterior edge of the lid, cilia, *c, c, c*. In the neighborhood of the latter are situated the sebaceous glands (Zeis's glands), and in front of the hair-papilla of the hindmost cilium is seen the transversely divided tubule of a modified sweat gland (gland of Moll), the excretory duct of which runs down alongside of the cilium and empties into its hair-follicle. Beneath the skin lie the transversely divided bundles of fibres of the orbicularis, *o o*, of which those placed most posteriorly, *r*, form the musculus ciliaris Riolani. The posterior surface of the lid is covered by conjunctiva which is intimately adherent to the subjacent tarsus, *k*, and over the latter shows papillæ, especially in the area between *k* and *w* corresponding to the convex (upper) border of the tarsus. Still high up in the neighborhood of the retrotarsal fold, *f*, it acquires an adenoid character. The Meibomian glands have their orifices in front of the posterior edge, *h*, of the lid; above them lie the branched, tubular glands, *w, w*, and still higher Krause's glands, *kr*, and in front of these Müller's musculus tarsalis superior, *t*, and the tendon of the levator palpebræ superioris *l*. From the latter leashes of fibres pass between the bundles of the orbicularis to the skin of the lids. *z*, lax cellular tissue. *as*, arcus tarseus superior. Above the roots of the cilia is the cross section of the arcus tarseus inferior.

of the free border of the lid that the skin is closely united to the subjacent tarsus by rigid connective tissue. This free border of the lid forms a narrow surface which looks downward in the upper lid, upward in the lower lid (*r*, *r*, Fig. 207 A). When the lids are closed, these two surfaces are adjusted to each other with perfect accuracy, so that, with the aid of the lubrication afforded by the secretion of the Meibomian glands, they are able to keep the lacrimal fluid in. When we open the lids forcibly in people affected with lacrimation and blepharospasm, we not infrequently see spurt from the eye a stream of tears which have been kept in by the closed lids—a proof that the closure of the lids is water-tight.

The lines along which the free border of the lids is reflected on to the anterior and posterior surface, respectively, of the lid are called the anterior and posterior margins of the lids (Fig. 1, *v* and *h*); the narrow surface lying between them is the intermarginal strip. The anterior margin of the lid is rounded off, and has jutting from it the eyelashes (cilia), which are arranged in several rows one behind the other. The cilia upon the upper lid are larger and more numerous than upon the lower. The posterior margin of the lid, where the free border of the lid passes into the conjunctival surface of the latter, is sharp. Directly in front of it lies a single row of small puncta, the orifices of the Meibomian glands (Fig. 1; Fig. 2, *m*). Between these and the cilia runs a fine gray line (*i*, Fig. 2) which divides the intermarginal strip into two parts, anterior and posterior. The free border of the lid has the above character as far inward as the spot where the punctum lacrimale is situated, a spot which corresponds to the inner extremity of the tarsus (Fig. 2, *p*, and Fig. 3).

On everting the lids we get a view of their posterior surface, which is covered with the conjunctiva. This is intimately adherent to the tarsus, and, particularly in the upper lid, allows the Meibomian glands situated in the tarsus to show through clearly.

That part of the cornea and of the scleral conjunctiva which ordinarily is not covered by the lids is called the *interpalpebral zone*. Since in this situation the eyeball is deprived of the protection of the lids, it is particularly exposed to many sorts of disorders, and it is therefore important to know its situation. This situation changes according to circumstances, and in the following way: 1. In the ordinary way of looking the interpalpebral zone comprises the entire cornea, with the exception of its extreme upper part, and comprises also a corresponding large triangular area of the conjunctiva on both sides of the cornea. 2. When the eyes are a little screwed together—e. g., when we are walking in the face of the wind or rain or in the midst of smoke—the interpalpebral zone diminishes in size, and at the same time is depressed so as to occupy the lower half of the cornea. The lower lid is raised a little and covers the extreme lower half of the cornea, and the upper lid drops a good deal so that its border lies only a little above the centre of the cornea. Then the interpalpebral zone forms upon the cornea a zone from 4 to 6 mm. in breadth which occupies the lower half of the cornea, with the exception of its extreme lower part, and with which there is connected on either side a very small triangle of scleral conjunctiva. The interpalpebral zone as thus defined is the part which more than any other is constantly exposed to external injuries. Hence in many men we find this portion of the scleral conjunctiva somewhat injected all the time, and later on in life we find it occupied by the pinguecula. In this spot are developed pterygium, zonular opacity of the cornea, and xerosis of the conjunctiva and cornea. In inflammations of the conjunctiva this division of the latter is frequently distinguished by being somewhat more swollen than the rest, or it may even protrude into the palpebral

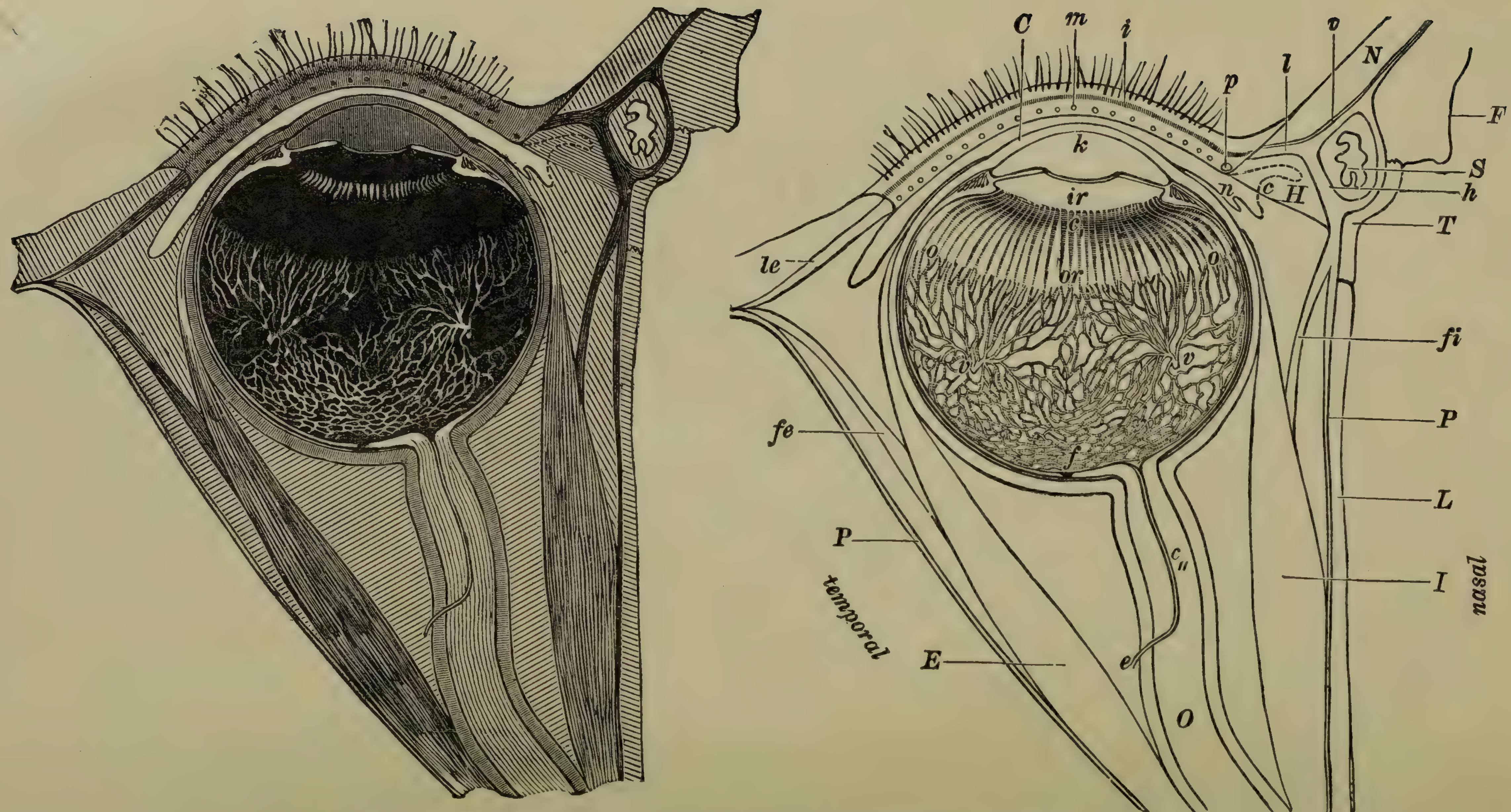


Fig. 2.—Horizontal Section of the Orbit. (Schematic.) Magnified 2 × 1.

The nasal wall of the orbit is formed by the lamina papyracea (os planum) of the ethmoid (*L*), the lacrimal bone (*T*), and the frontal process (*F*), of the superior maxilla. The last two bones bound the fossa sacci lacrimalis, in which lies the lacrimal sac, *S*. The bony walls of the orbit are coated by a periorbita (periosteum), *P*, from which the palpebral ligaments take their origin. The internal palpebral ligament, *l*, divides into an anterior limb, *v*, and a posterior limb, *h*, which together inclose the lacrimal sac. From the posterior limb arise the fibres of Horner's muscle, *H*. *le*, external palpebral ligament; *fi*, and *fe*, the slips of fascia (check ligaments) likewise originating from the periosteum, going to the internal rectus muscle, *I*, and the external rectus, *E*. The skin, *N*, of the dorsum of the nose passes into the lower lid, at whose free border are seen the cilia and the orifices of the Meibomian glands, *m*; between the two extends a gray line, *i*. At the inner extremity of the lids lies the inferior punctum lacrimale, *p*, and farther along in the conjunctival sac the caruncle, *c*, and the plica semilunaris, *n*. From the eyeball, the lower half of which is exhibited, the lens and along with it the vitreous humor have been taken out. The anterior chamber, *k*, the iris, *ir*, and the ciliary body, consisting of the corona ciliaris, c_{I}, and the orbiculus ciliaris, *or*, are visible. Back of the ora serrata, *o*, is the chorioid with its veins which are aggregated into vortices, *v*. *f*, fovea centralis retinæ; c_{II}, central vessels of the optic nerve, *O*, entering it at *e*.

fissure under the form of a transversely placed, very œdematous swelling. 3. When the eye is turned upward in sleep the interpalpebral zone, in case the lids are not completely closed, is displaced, so as to occupy mainly the scleral conjunctiva beneath the cornea, and at most the extreme lower portion of the latter. Affections within the confines of the interpalpebral zone as thus defined are found when the palpebral fissure is kept open during sleep, and hence mainly in lagophthalmus, in which the conjunctiva beneath the cornea is found injected or œdematous, and in which, when the affection is of greater extent, the lowermost division of the cornea also suffers damage. The same turning upward of the eyeball that occurs in sleep takes place also when one winks because of the approach of anything endangering the eye, for which reason injuries by burns and caustic substances affect principally the extreme lower portion of the cornea.

3. Lid Muscles.—In the lids are found two voluntary muscles, the orbicularis (sive sphincter) palpebrarum and the levator palpebræ superioris. The *orbicularis* lies directly beneath the skin of the lid to which it belongs; it is nothing but a flat expanded cutaneous muscle which surrounds the palpebral fissure in the form of a circle. We can distinguish in it two portions, a central and a peripheral. The central portion lies in the lids themselves, and is hence called the palpebral portion (portio palpebralis). Its fibres originate from the internal palpebral ligament, the *ligamentum canthi mediale* (sive *canthi internum*). This is a firm, fibrous ligament (Fig. 2, *l*) which is attached to the frontal process of the superior maxilla (*F*), and lies directly beneath the skin of the internal angle of the eye (Fig. 3). Hence, it is visible even in the living, especially in lean persons with thin skins, if the eyelids are drawn outward, a manœuvre that causes the internal palpebral ligament to project and bulge the skin forward. From the internal palpebral ligament the fibres of the palpebral portion of the orbicularis run in arches over the anterior surface of the two lids, covering them from the free border of the lids to the margin of the orbit, and finally meeting at the outer side of the palpebral fissure. Here they unite by means of a tendinous inscription (the *rhaphe palpebralis lateralis*). Beneath this lies the external palpebral or canthal ligament (*ligamentum palpebrale laterale*) (ligamentum canthi externum) which connects the temporal extremities of both tarsal cartilages with the temporal border of the orbit (Fig. 2, *le;* Fig. 3). The external portion of the orbicularis is the orbital portion (portio orbitalis). It lies outside of the palpebral portion, upon the margin of the orbit and the parts surrounding it.

The *levator palpebræ superioris* arises at the bottom of the orbit from the circumference of the optic canal, and from this point runs forward, lying as it does so upon the superior rectus, with which it is intimately connected by bands of fascia. Spreading out in the form of a fan, its tendon runs down upon the anterior surface of the tarsus of the upper lid (*l*, Fig. 1). Besides this striated levator muscle of the lid, there is also an organic muscle discovered by Heinrich Müller, and called the *musculus tarsalis superior.* The smooth fibres of this arise from between the striated fibres of the levator, along the under surface of which they too run to the upper margin of the tarsus (*t*, Fig. 2). An analogous bundle of smooth muscular fibres is also present in the lower lid, where it lies to the lower side of the inferior rectus, and is attached to the tarsus of the lower lid (*musculus tarsalis inferior* of Müller).

The orbicularis is innervated by the facial nerve, the levator by the oculo-motor, and the two tarsal muscles of Müller by the sympathetic.

The *ligamentum palpebrale mediale* arises from the frontal process of the superior maxilla (*F*, Fig. 2) and first passes straight outward, skirting the anterior wall of the lacrimal sac (*S*). Then it turns round the anterior and external walls of the lacrimal sac and runs backward to the crista lacrimalis posterior of the lacrimal bone (*T*). We accordingly distinguish in the internal palpebral ligament two branches, which meet at the point where it begins to turn. The anterior branch (*v*) is situated directly beneath the skin, and hence is visible in the living subject; at its point of reflection it gives off a process to the upper and lower tarsus respectively (Fig. 3). The posterior branch (*h*), which starts from the point of reflection and extends to the crista lacrimalis, can be brought to view only by dissection. The two branches together with the lacrimal bone (*T*) bound a space, triangular on cross section, in which lies the lacrimal sac, the walls of the latter being united by loose connective tissue with the inner surface of the ligament. Into the external surface of the ligament are inserted the fibres of the palpebral portion of the orbicularis. One portion of the fibres of the latter springs from the anterior, another portion from the posterior branch of the ligament. The latter fibres, whose insertion is in part continued out beyond the posterior extremity of the ligament upon the inner wall of the orbit, are called the *pars lacrimalis musculi orbicularis* (or *tensor tarsi*) or, from their discoverer, Horner's muscle (*H*). The insertion of the fibres of the orbicularis into the internal palpebral ligament is of significance for the conduction of tears. (See § 67).

The fibres of the orbicularis fuse with the anterior surface of the tarsus. In the neighborhood of the free borders of the lid there are some bundles which lie near the inner margin of the lid, partly in front of, partly behind the excretory ducts of the Meibomian glands (*musculus ciliaris Riolani* sive *subtarsalis; r*, Fig. 1).

4. Tarsus; Glands of Lids.—The tarsus (*k*, Fig. 1) forms, so to speak, the skeleton of the lid, giving it rigidity of form and affording it firm support. The tarsus of the upper lid is broader (higher) than that of the lower (Fig. 3). In the tarsus are distinguished a free and an attached (convex) border, and also an anterior and a posterior surface. The fibres of the orbicularis (*o*, Fig. 1) lie upon the anterior surface, while the posterior surface is covered by the conjunctiva. The two extremities of the tarsus are continuous with the external and internal palpebral ligaments. To the convex border of the tarsus is attached a fascia which runs from it to the margin of the orbit, and upon either side is connected with the palpebral ligaments (fascia tarso-orbitalis). Hence, when the lids are shut the orbit is closed in all over anteriorly by fibrous structures which together form the *orbital septum* —namely, the two tarsi in conjunction with the fascia tarso-orbitalis and the two palpebral ligaments (Fig. 3).

The tarsus is called the cartilage of the lids. It is not, however, cartilage, but a very dense fibrillary connective tissue, in which are imbedded the *Meibomian glands.* These are elongated acinous glands which, lying parallel with each other, traverse the tarsus from its attached to its free border. They are longest in the middle of the tarsus, where the latter attains its greatest height, and grow progressively shorter toward the edges of the tarsus (Fig. 3). In their essential character the Meibomian glands are nothing but large sebaceous glands. Like the latter they secrete sebum, which lubricates the edges of the lids. By this the overflow of tears over the free border of the lids is hindered; the closure of the palpebral fissure is rendered water-tight; and, lastly, the skin of the border of the lid is protected from maceration by the tears. Acinous mucous glands are frequently found near the convex border of the tarsus (Fig. 1, *w*, and Fig. 3).

At the free border of the lids there are found, in the vicinity of the cilia, hair follicles and the sebaceous *glands* (here called Zeis's glands) connected with them. Moreover, close to the free border of the lids, sweat glands occur, the structure of which varies somewhat from that of the ordinary sweat glands, for which reason they are known as modified sweat glands or Moll's glands. They empty into the hair follicles of the cilia.

In accordance with its anatomical structure, the lid can be readily divided into *two parts*. The anterior or cutaneous portion contains the skin, together with the cilia and also the fibres of the orbicularis. The posterior or conjunctival portion consists of the tarsus with the Meibomian glands and of the conjunctiva. The two portions are joined simply by loose con-

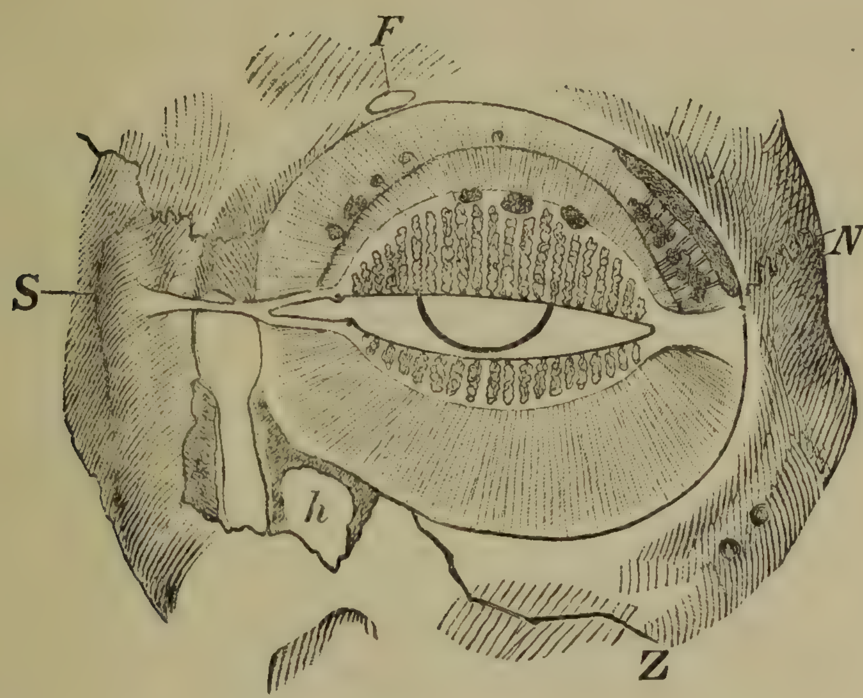


FIG. 3.—SEPTUM ORBITALE AND LACRIMAL SAC. Natural size.

The skin and the muscular fibres of the orbicularis have been removed from the lids and the parts surrounding them, so that the septum orbitale lies exposed to view within the bony circumference of the orbital cavity. The septum orbitale consists of the tarsus, which is broader in the upper lid, narrower in the lower, and of the fascia tarso-orbitalis. The external extremities of the tarsi are attached by the broad, but not very dense rhaphe palpebralis lateralis to the malar bone, somewhat below the suture, *N*, between this bone and the zygomatic process of the frontal bone. The internal palpebral ligament is narrower, but stout; its anterior limb, which is the only one visible in the drawing, runs from the frontal process of the superior maxilla, *S*, outward and divides so as to be inserted into the inner extremities of both the upper and lower tarsi. (At the point of insertion is seen the somewhat projecting papilla lacrimalis.) The fascia tarso-orbitalis, represented in the drawing by the radial lines of shading, runs from the convex border of both tarsi, and from the palpebral ligaments to the margin of the orbit, and together with these parts closes in the orbit in front. The tarsi and the fascia are here supposed to be transparent. Hence, in the former there can be seen the Meibomian glands, which, in consonance with the varying breadth of the tarsus, diminish in height from the centre of the latter to its two ends. Moreover, in the upper lid three acinous glands (cf. Fig. 1, *w*) are visible along the upper border of the tarsus. Still higher up a curved line shows the situation of the fornix conjunctivæ. Upon the fornix, especially in its nasal half, lie the acinous glands of Krause (Fig. 1, *kr*), while in the temporal half of the fornix are found lobules similar in character, but more densely packed, representing the inferior lacrimal gland This adjoins the excretory ducts of the superior lacrimal gland, whose anterior border comes into sight just below the upper margin of the orbit. At the inner and lower margin of the orbit the bone has been chiseled away to show the lacrimal passages. The lacrimal sac lies behind the internal palpebral ligament, its apex rising a little above the latter. The line that in the drawing runs straight upward from the apex of the lacrimal sac to the horizontal suture is the suture between the frontal process of the superior maxilla and the lacrimal bone, upon which two bones the lacrimal sac rests (cf. Fig. 2, *F* and *T*). The lacrimal sac, after undergoing a slight constriction, passes into the nasal duct. To the outside of this is the antrum of Highmore, *h*, which has been opened up and is accessible to view. *Z*, suture between the superior maxilla and the malar bone. *F*, supraorbital foramen.

nective tissue, and so can be very readily split apart. For this purpose we need only make a stab in the gray line that runs between cilia on the one hand and orifices of the Meibomian glands on the other (Fig. 2, *i*). The splitting of the lid into its two layers forms an important part of many trichiasis operations (cf. § 920).

5. Vessels of the Lids.—The *blood-vessels* of the upper lid arise from the arcus tarseus superior and inferior (*as* and *ai*, Fig. 4), from them fine twigs are given off to all parts of the lid. The most vascular portions are the free border of the lid and the conjunctiva.

The veins of the lids are still more numerous and of wider caliber than the arteries. They form, beneath the upper and lower retrotarsal folds, a dense plexus, which even in the living subject can be seen in this situation shining through the conjunctiva of the fornix when the lid is everted. The veins of the lids in part empty into the veins of the forehead, in part into the branches supplying the ophthalmic vein. The latter set, in order to reach to the veins of the orbit, must pass between the fibres of the orbicularis. Hence, permanent contraction of the orbicularis, such as occurs in blepharospasm, may lead to engorgement of the veins, and consequently to œdema of the lids, a result which in fact, we very frequently observe, especially in children with phlyctenular conjunctivitis and coincident blepharospasm.

The *lymphatic vessels* of the lids are abundant, especially in the conjunctiva. Furthermore, lymph spaces of larger size (periacinous spaces) are found about the acini of the Meibomian glands. The lymph vessels of the lids run to the lymph gland in front of the ear, which, consequently, is often found to be swollen in the severer forms of inflammation of the lids, and sometimes also in affections of the conjunctiva (e. g., conjunctivitis gonorrhoica).

II. Conjunctiva

6. Divisions.—The conjunctiva coats the posterior surface of the lids and the anterior surface of the eyeball. It forms a sac, the *conjunctival sac*, which is slit open anteriorly in a line corresponding with the palpebral fissure. In the conjunctiva we distinguish three divisions. That part of the conjunctiva which covers the posterior surface of the lids and which is closely adherent to the tarsus is called the conjunctiva tarsi; that division which coats the anterior surface of the eyeball is the conjunctiva bulbi. The connection between the two is formed by the third division, which we name the retrotarsal fold (conjunctiva fornicis). That region where the conjunctiva is reflected from the lids to the eyeball and which forms the periphery of the conjunctival sac is called the *fornix conjunctivæ.*

7. Tarsal Conjunctiva.—We get a view of the conjunctiva tarsi in the living eye by everting the lids. It has a smooth surface and is intimately and immovably adherent to the subjacent tarsus (Fig. 1, *k*). (It is therefore impossible to cover up losses of substance of the palpebral conjunctiva by performing an operation to draw the adjacent conjunctiva over them, as is often done with the conjunctiva bulbi.) On account of its thinness, the conjunctiva tarsi allows the Meibomian glands, which lie in the tarsus itself, to be seen through it clearly.

The conjunctiva of the lids as well as that of the fornix is covered with a laminated cylindrical epithelium. The mucous membrane proper is of adenoid character—that is, even in the healthy state it contains an abundant quantity of lymphocytes, which notably increase in number with every inflammation of the conjunctiva. The palpebral conjunctiva contains small glands, which are found partly along the convex border of the tarsus (Fig. 1, *w*), partly in the fornix conjunctivæ (Krause's glands, Fig. 1, *kr*). In structure these resemble the lacrimal gland.

In the epithelium of the conjunctiva, particularly in its uppermost layers, are found cells which are undergoing a mucous metamorphosis (*beaker cells*). They occur but

sparsely in the normal conjunctiva, but multiply to a great extent in inflammatory disorders of the latter.

The conjunctiva of the tarsus rises in low papillæ, over which, however, the epithelium passes undimpled so that the surface of the conjunctiva is smooth. This continues so up to the convex border of the tarsus, where not only are the papillæ higher, but the surface of the epithelium dips down between the papillæ, forming sulci so that the papillæ become visible as distinct structures and the surface of the conjunctiva acquires a slightly velvety appearance. The extent to which these papillæ are developed shows great individual variations, and the line between a physiological and a pathological state cannot be sharply drawn. The conjunctiva is exposed to external influences more than any other mucous membrane, and hence in every man attacks of hyperæmia occur in it often in the course of life, and these may ultimately result in a permanent alteration of the membrane. This is probably also the cause of the variations in degree of the adenoid structure of the conjunctiva. Small quantities of plasma cells and small nodules consisting of lymphocytes, are present in the healthy conjunctiva, but these become increased as a result of the state set up by repeated irritation. The like is true of the inconstant, but still very frequently seen tubular depressions lined with cylindrical epithelium which are called, after their discoverer, Henle's glands (Fig. 208, *t*). Furthermore, in middle and advanced life there are found, frequently in the retrotarsal fold, less often in the palpebral conjunctiva, little yellow dots resembling the infarcts of the Meibomian glands (§435). This again is an instance of the new formation of tubular glands, in which develop concretions that are visible through the conjunctiva under the form of yellow dots.

8. Retrotarsal Fold.—The conjunctiva of the retrotarsal fold is brought to view by everting (or doubly everting) the lid (see § 388). It is the most lax portion of the conjunctiva, which is here so abundant that it lies in horizontal folds. This arrangement insures the eye its free power of movement. If the conjunctiva were to pass directly from the lid to the eye, as is sometimes observed in consequence of disease of the conjunctiva, every movement of the eyeball would be transmitted to the lids; and if one of the lids was held still with the finger, the eyeball would be hampered by it in its movements. But the conjunctiva is present in such quantity at the fornix that the eye is able to move in complete independence of the lids, the folds in this region being smoothed out or crumpled together, as the case may be. Appearing through the lower retrotarsal fold are the extensive subjacent plexus of veins and also the white glistening fascia. Its lax character and also its abundant blood supply render the retrotarsal fold particularly liable to great swelling in inflammations of the conjunctiva.

9. Bulbar Conjunctiva.—The *conjunctiva bulbi* covers the anterior surface of the eyeball. It has no aperture corresponding to the cornea but continues, even if with altered character, over the latter. This continuity of the conjunctiva makes it plain to us why morbid processes of the latter do not stop at the margin of the cornea but are continued upon the surface of the latter, as we see very clearly in trachoma and in phlyctenular conjunctivitis. The two divisions of the conjunctiva bulbi are distinguished as the conjunctiva scleræ and conjunctiva corneæ. The *conjunctiva corneæ* is perfectly transparent, and is so intimately adherent to the cornea proper that it must be regarded as the uppermost layer of the latter, and is better treated with the cornea itself (see § 16).

The *conjunctiva scleræ* covers the anterior segment of the sclera in the form of a thin pellicle. It is connected with the sclera by lax connective

tissue (the episcleral tissue) so loosely that it can readily be moved about from side to side upon the sclera. It is only at the periphery of the cornea, where it ends in a sharp edge, the limbus[2] conjunctivæ, that the conjunctiva scleræ is intimately adherent to its substratum. It is very thin and elastic and lets the white sclera be seen through it plainly, thus forming the "white of the eye." There is an area, especially marked in old people, but present even in the young, at the inner and the outer margins of the cornea which contrasts by its yellow color with this whiteness. This has the shape of a triangle with its base at the corneal margin, and projects a little above the rest of the conjunctiva. It is called the interpalpebral spot or the pinguecula.[3]

The pinguecula is so called because of its yellow color, which was formerly referred to disposition of fat in the conjunctiva. What actually is present, however, is a thickening of the conjunctiva, due chiefly to an increase in the number and size of its elastic fibres. Associated with this is the formation of numerous concretions of a yellowish hyaline substance, to which in fact the pinguecula owes its yellow color. As a result of these changes, the conjunctiva in this place becomes less transparent, for which reason the pinguecula appears most prominent when the conjunctiva bulbi is markedly reddened, whether from injection or from extravasation of blood. In this case the pinguecula does not allow the red color of the blood to shine through as plainly as does the adjacent conjunctiva that is not thickened, and the former, therefore, stands out from the red substratum in the form of a light-colored triangle, so that by beginners it is easily confounded with a diphtheritic infiltration of the conjunctiva, or, when the yellow color is pronounced, with a small pustule.

The conjunctiva scleræ is covered with laminated pavement epithelium and contains no glands. At the inner angle of the eye it forms a crescentic duplication, the semilunar fold (plica semilunaris), which represents an abortive remnant of the palpebra tertia (nictitating membrane) of animals. To the inside of the semilunar fold is a small, reddish, nipple-like prominence, the caruncle (caruncula[4] lacrimalis), which occupies the bottom of the horseshoe-shaped excavation at the angle of the eye (Fig. 218, *C*). This is shown to be histologically a small island of skin, containing sebaceous glands, sweat glands, and small glands like Krause's glands and having its surface covered with minute light-colored hairs.

10. Blood-Vessels.—The conjunctiva of the upper lid obtains its blood supply from two arterial arches, the arcus tarseus superior and the arcus tarseus inferior.

The arcus tarseus inferior (Fig. 4, *ai*) lies on the anterior surface of the tarsus close to its lower border. To reach the conjunctiva its branches perforate the tarsus through its entire thickness from before backward, 2 to 3 mm. above the free edge of the lid (Fig. 4, *rp*). The line along which the vessels come out along the tarsus is marked by a shallow furrow (sulcus subtarsalis) on the conjunctival surface of the lid. The arcus tarseus superior (Fig. 4, *as*) lies a little above the upper border of the tarsus upon the fascia tarso-orbitalis, which runs from the tarsus to the margin of the orbit and through which the branches of the arcus superior pass to the conjunctiva. On the lower lid there is but one arterial arch.

The conjunctiva of the eyeball receives its blood-vessels chiefly from the vessels of the retrotarsal fold—the posterior conjunctival vessels (Fig. 5,

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[2] *Limbus*, hem. [3] *Pinguis*, fat. [4] *Caruncula*, dim. of *caro*, flesh.

h and h_1) Furthermore, the anterior ciliary vessels (Fig. 5, c and c_1) take part in supplying the conjunctiva with blood. These vessels come from the four recti muscles (R, Fig. 5) and run under the conjunctiva (through which they are visible, shining with a bluish lustre) until near the edge of the cornea, where they suddenly disappear, since they pass through the sclera into the interior of the eye. But before this happens, they give off branches which end in vascular loops, in the limbus conjunctivæ directly at the margin of the cornea (marginal network of the cornea—Fig. 5, r and Fig. 6). This latter is of great importance for the cornea, which is chiefly dependent upon it for its nutrition. Other branches of the ciliary vessels (anterior conjunctival vessels, Fig. 5, v) run backward in the conjunctiva toward the posterior conjunctival vessels (h h_1) and anastomose with them.

We have therefore in the conjunctiva two vascular systems—that of the posterior conjunctival vessels and that of the anterior ciliary vessels. In inflammations of the eye, one or the other may be injected. (§ 448).

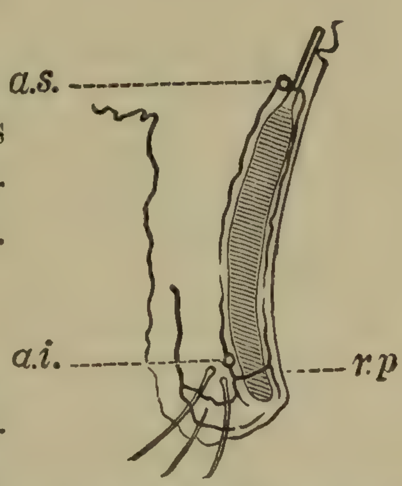

FIG. 4.—ARTERIES OF THE UPPER LID. Magnified 3 × 1.
as, arcus tarseus superior; *ai*, arcus tarseus inferior; *rp*, rami perforantes.

III. LACRIMAL ORGANS

11. Lacrimal Glands.—The lacrimal organs consist of the lacrimal gland and the lacrimal passages.

The *lacrimal gland* (glandula lacrimalis) is an acinous gland with short, branched gland tubules. It consists of two divisions, the larger, known as the superior lacrimal gland, lying in the upper outer angle of the orbit in a depression in the bony wall of the latter, the fossa glandulæ lacrimalis. Excretory ducts of the superior lacrimal gland pass downward and empty into the outer half of the superior fornix conjunctivæ.

The second division of the lacrimal gland—the inferior lacrimal gland—is much smaller, and consists only of one or two lobules, for which reason it is also known as the accessory lacrimal gland. Its lobules lie along the excretory ducts of the superior gland directly beneath the mucous membrane of the fornix (Fig. 3). If the upper lid is everted and at the same time the eye is made to look downward, we often see the conjunctiva of the fornix in the vicinity of the outer angle of the lid pushed forward by a soft mass which is in fact the accessory lacrimal gland. Krause's glands (see Fig. 1, *kr*, and Fig. 3) form a sort of continuation of the lobules of the inferior lacrimal gland along the fornix as far as its inner end. Their structure is that of the lacrimal gland, so that they may be regarded as the ultimate scattered outlying portions of the latter.

12. Lacrimal Passages.—The lacrimal passages begin with the *puncta lacrimalia.* These lie on the free border of the upper and lower lid (upper and lower punctum) and near the inner extremity of the lid at the spot where the tarsus terminates (Fig. 3). They are situated upon small elevations, the lacrimal papillæ (papillæ lacrimales), and form the orifices of the canalic-

uli lacrimales. These latter, starting from the puncta, run at first vertically for a short distance—i. e., in the upper lid run upward and in the lower lid downward—then they bend at a right angle and become directed toward

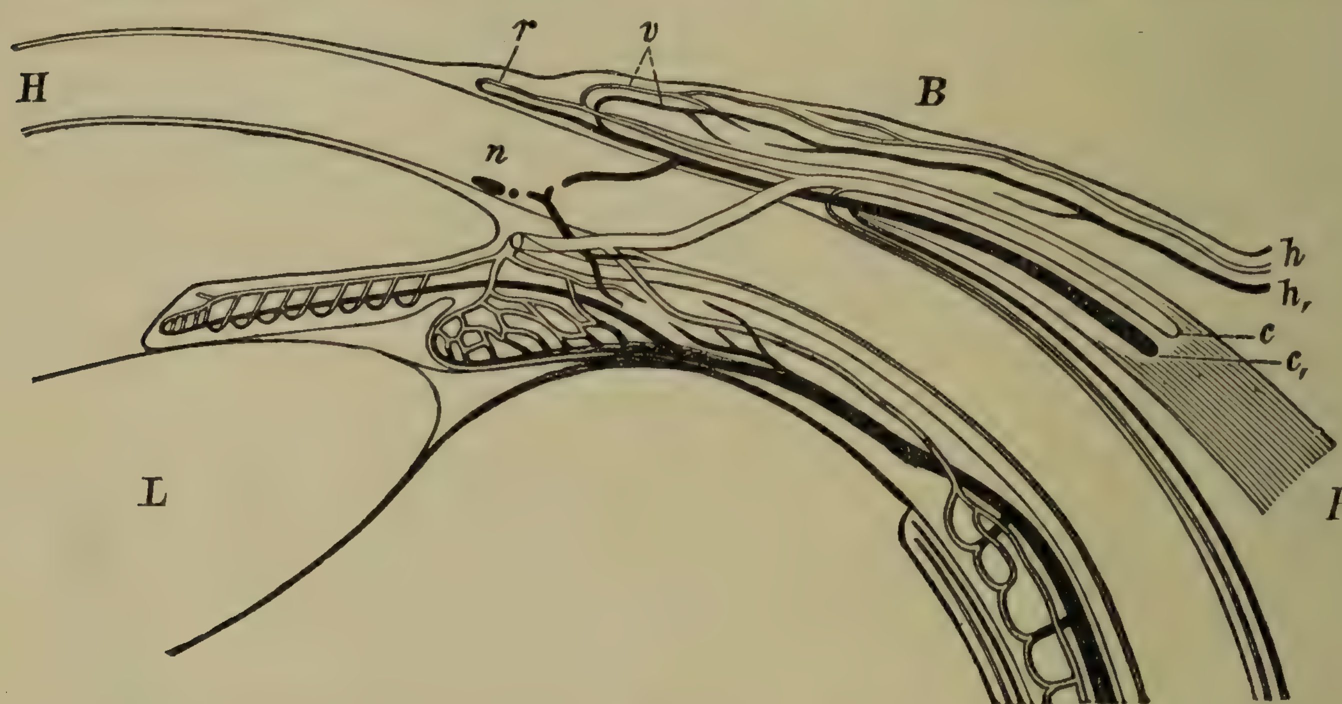

FIG. 5.—BLOOD-VESSELS OF THE ANTERIOR SEGMENT OF THE EYE. SCHEMATIC. (After Leber.)
The posterior conjunctival vessels, h and h_1, communicate with the anterior conjunctival vessels, r, which come to meet them and which are branches of the anterior ciliary vessels, c and c_1; and with the latter form the marginal network in the limbus. n, Schlemm's canal; H, cornea; B, conjunctiva; R, one of the recti muscles; L, lens.

FIG. 6.—MARGINAL NETWORK IN THE LIMBUS. (After Leber.)
The arteries are drawn in light color, the veins dark. In the marginal loops can be distinguished the thinner arterial and the thicker venous segments.

the lacrimal sac. In so doing they first pass behind the caruncle, and converging more and more, at length reach the lacrimal sac. Into this they empty, either separately or after having united to form a short common trunk.

The *lacrimal sac* (saccus lacrimalis) lies in the inner angle of the eye in the cleft (fossa sacci lacrimalis) which the lacrimal bone forms for its recep-

tion. The lacrimal bone bounds the lacrimal sac (*S*, Fig. 2) nasally, while to the front and temporally it is inclosed by the two branches of the ligamentum palpebrale mediale (*v* and *h*, Fig. 2). This relation of the lacrimal sac to the internal palpebral ligament enables us to determine the position of the former—a matter which is of importance when operations are concerned. If by drawing the lids outward we put them on the stretch and so cause the palpebral ligament to project, the lacrimal sac lies behind the latter, and in such a way as to rise just above it by its cupola or fundus (Fig. 3).

At the spot where the cleft of the lacrimal bone merges into the bony canal the lacrimal sac passes into the nasal or *lacrimal duct* (ductus lacrimalis). The point where this transition occurs constitutes the narrowest part of the whole lacrimal channel (Fig. 3), and is therefore particularly liable to the formation of pathological contractions (strictures). From this point the lacrimal duct passes downward and empties into the nasal fossa below the inferior turbinated body. In its downward course the lacrimal duct deviates a little backward and outward from the vertical. Hence, the two lacrimal channels diverge as they go down, the lacrimal sacs being less far apart than are the lower orifices of the lacrimal ducts.

We can represent the course of the lacrimal channel on the living subject by placing a straight sound in such a way as to lie at its upper part upon the middle of the internal palpebral ligament, and below upon the furrow forming the boundary line between the cheek and the alæ of the nose (see Fig. 7). This sound gives precisely the direction of the lacrimal duct (Arlt). If we place a sound in this way on each side of the nose, we see how the sounds diverge as they go down, and we can readily convince ourselves that the degree of divergence differs in different individuals. The divergence, in fact, depends upon the breadth of the root of the nose on the one hand, and upon the breadth of the inferior nasal orifice on the other. These facts are of importance with regard to the operation of sounding the lacrimal duct, in the performance of which the sound must be pushed along in the direction of the duct.

The mucous membrane of the lacrimal sac and that of the lacrimal duct forms one continuous whole. There is, therefore, no sharp dividing line between these two structures. They are mainly distinguished by the fact that the lacrimal sac lies against bone (the lacrimal bone) at one side only, and everywhere else is free, while the lacrimal duct is inclosed on all sides by bony walls. It follows from this that, in engorgement of the lacrimal channels with fluid, it is *only the lacrimal sac which is distended* so as to appear as a visible swelling at the inner angle of the eye. The lacrimal duct cannot be distended; on the contrary, it is the favorite seat of *constrictions*, which again do not occur in the lacrimal sac. The formation of these constrictions is facilitated by the fact that a dense plexus of wide veins, analogous to the venous plexuses beneath the mucous membrane of the turbinated bodies, is interposed between the mucous membrane of the lacrimal duct and the bony wall (Fig. 8). Swelling of these veins alone is sufficient to contract or to close entirely the lumen of the duct.

The lacrimal passages are always filled with a little lacrimal fluid. If air is found in them, it is to be regarded as a pathological condition.

The mucous membrane of the canaliculi is lined with laminated pavement epithelium, that of the lacrimal sac and nasal duct with a double layer of cylindrical epithelium. The mucous membrane at different spots projects in the form of folds into the lumen of the lacrimal passages—a phenomenon which has been described as a formation of *valves*. The largest of these folds is Hasner's valve, at the lower orifice of the nasal duct. This, however, is not a true valve, any more than are the others—that is, not a valve which could close up the lumen of the lacrimal channel. On the contrary, it is

simply a fold produced by the great obliquity with which the nasal duct passes through the mucous membrane of the nasal fossa. Like the other folds of mucous membrane in the lacrimal passages, it is not of constant occurrence.

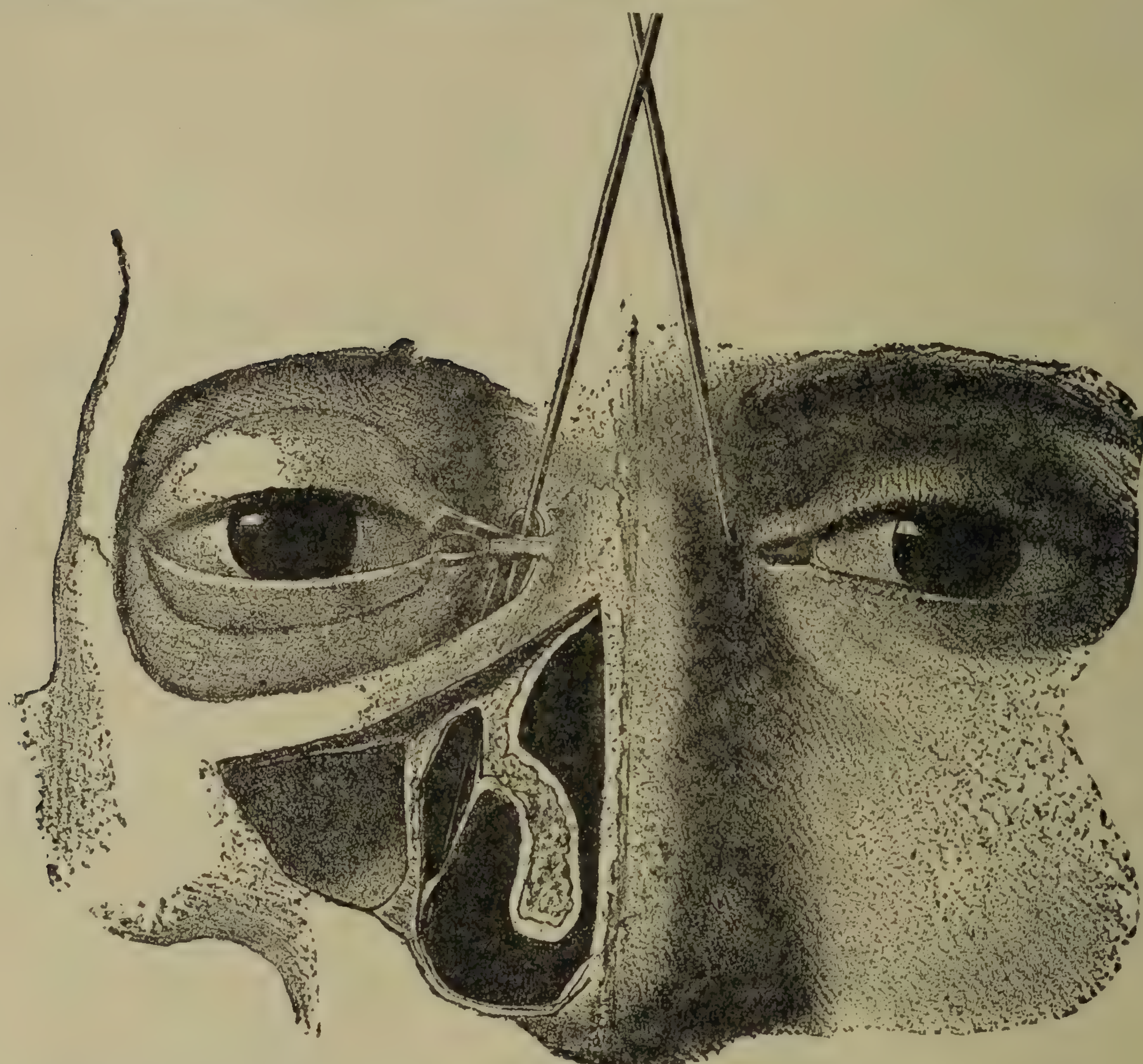

FIG. 7.—SECTION OF CANALICULI, LACRIMAL SAC, AND NASAL DUCT. (De Wecker.) After Norris and Oliver.

FIG. 8.—CROSS SECTION THROUGH THE LACRIMO-NASAL DUCT. Magnified 11 × 1.
The lumen of the lacrimal duct is oblong and lined with cylindrical epithelium. The subjacent mucous membrane contains very many lymphocytes, i. e., it has an adenoid character. The submucous tissue is marked by its great abundance of blood-vessels. Most of these are veins; the few arteries are made obvious in the drawing by their double contour. At *a* lie the acini of a mucous gland, whose excretory duct emptying into the lacrimal duct is not traversed in the section here shown. Succeeding the submucous tissue is the wall of the bony canal; *b* is the side of this canal that is directed toward the maxillary antrum, *c*, the side that is directed toward the nasal cavity.

CHAPTER II

CORNEO-SCLERA

I. Cornea

13. The exterior, protective envelope of the eye is a fibrous capsule, having nearly the shape of a sphere about 24 mm. in diameter. Much the larger portion of this capsule is formed by the opaque sclera; the anterior, more convex portion is the transparent cornea.

14. Gross Anatomy.—Seen from in front, the cornea has the form of a horizontal ellipse, the horizontal diameter, which is 12 mm., surpassing the vertical diameter, which is 11 mm. It is thinner in the centre than at the edges, where its thickness amounts to about 1 mm. Hence it follows that the curvature of the posterior surface is somewhat greater than that of the anterior. The latter has on the average a radius of curvature of 7.5 mm. Since the radius of curvature of the whole eye is more than this—

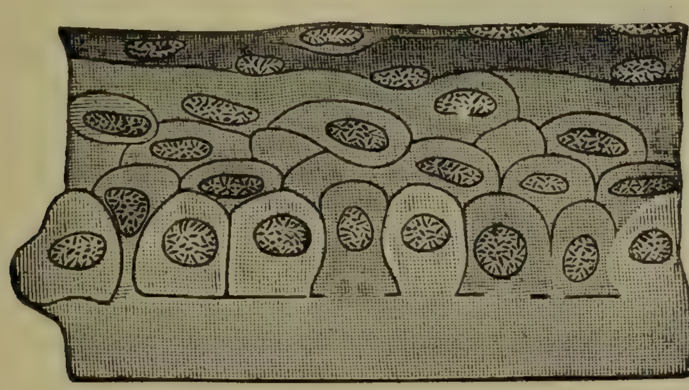

Fig. 9.—Epithelium of the Cornea. (After H. Virchow.) Magnified 660×1.
B, Bowman's membrane; *u* lowest, *m* middle, *o*, uppermost layer of cells.

amounting, in fact to 12 mm.—the curvature of the cornea is greater than that of the rest of the eyeball; the cornea therefore rests on the sclera like a watch crystal. The same comparison also obtains for the method in which the cornea is inserted into the sclera; for, in its posterior layers the cornea extends farther toward the periphery than in the anterior, where the sclera, as it were, laps over the edge of the cornea. The microscope, however, does not show any sharp boundary line between cornea and sclera; on the contrary, the fibres of one pass continuously into those of the other.

The healthy cornea is transparent. Almost all morbid changes of the corneal tissue make themselves known at once by a diminution of this transparency (see § 69). In advanced age, however, a cloudiness makes its appearance even in the healthy cornea (arcus senilis corneæ; see § 596).

While the cornea, viewed from in front, appears elliptical, it is circular if looked at from behind. Hence the elliptical form of the front surface of the cornea is due to the fact that both sclera and conjunctiva overlap it farther above and below than at either side.

15. Minute Structure.—The cornea consists of the following layers:

1. The *anterior epithelium* (Fig. 9; Fig. 10, *E*). This is a pavement epithelium consisting of several layers; the lowermost cells (foot cells, Fig. 9, *u*) are cylindrical, then follow rounded cells (Fig. 9, *m*), and finally flat cells (Fig. 9, *o*).

2. *Bowman's membrane* (lamina elastica anterior, Fig. 10, *B*) is a thin, homogeneous membrane closely connected with the corneal lamellæ lying beneath it. It may be said to represent the uppermost layer of the stroma of the cornea, which has become homogeneous and destitute of cells. From the epithelium it is separated by a sharply defined border; and, under pathological conditions as well as after death, the epithelium separates readily from Bowman's membrane.

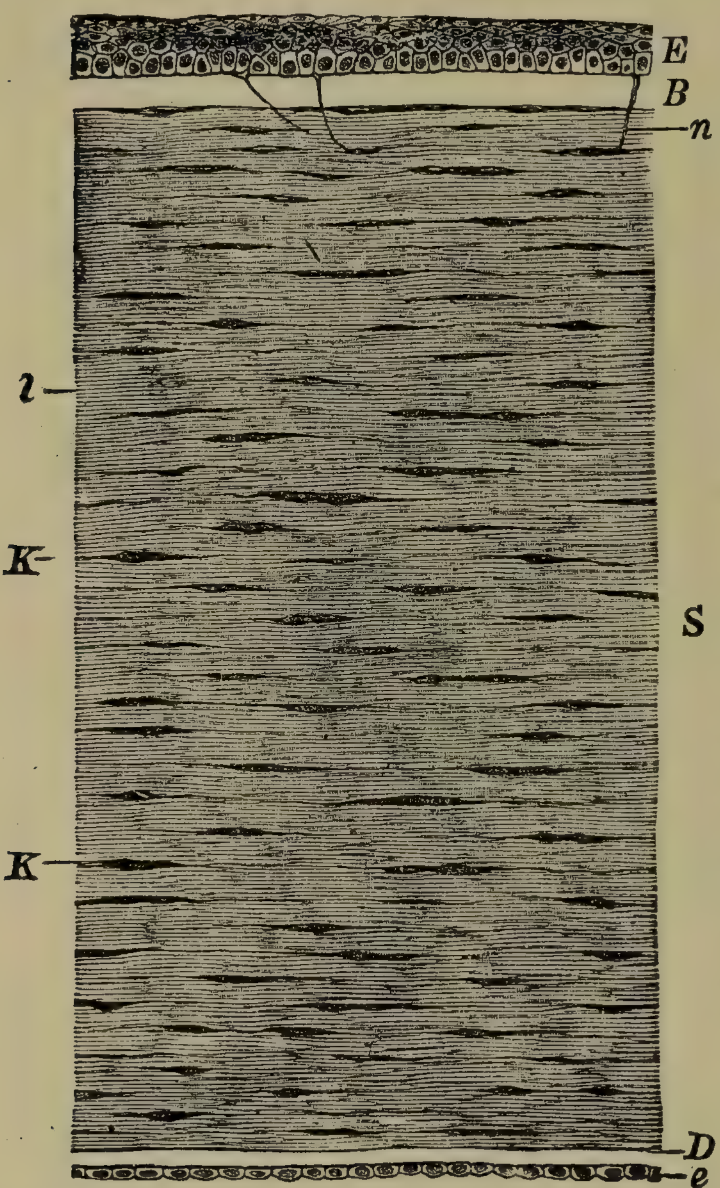

Fig. 10.—Cross Section through a Normal Cornea. Magnified 100×1.

E, anterior epithelium; *B*, Bowman's membrane; *S*, stroma, composed of the corneal lamellæ *l*, and the corneal corpuscles, *K*; D, Descemet's membrane; *e*, posterior epithelium; *n*, nerves extending through Bowman's membrane and the epithelium.

3. The *stroma* (Fig. 10, *S*). This is composed of a fibrillated ground substance and of cells.

The ground substance in its ultimate constitution consists of minute fibres of connective tissue, united by a cement substance into flat bundles. The bundles are so applied to one another that lamellæ (Fig. 10, *l*) are produced; by the arrangement of these lamellæ in layers one above another the cornea is built up. The latter has, therefore, a laminated structure. The individual lamellæ are connected together so that it is impossible to separate the cornea into its lamellæ perfectly and without tearing many fibres. Moreover, there are minute elastic fibres in the cornea, which form a specially dense network in front of Descemet's membrane.

Between the lamellæ lie the cells of the stroma of the cornea, the *corneal corpuscles*, which are of two kinds—nonmotile and motile cells. The former are the *fixed corpuscles* of the cornea. They are cells with a large nucleus and a very flat, protoplasmic cell body, the numerous branched processes of which are connectedwith the processes of adjoining cells so that in this way there is formed a system of connected protoplasmic bodies (cells and their processes, Figs. 11, 12, and 13).

The second variety of cells belonging to the stroma are the *motile corpuscles* of the cornea (wandering cells), which were discovered by Recklinghausen. These are nothing but white blood-corpuscles which have made their way into the cornea and which move about in its system of lymph passages. In the normal cornea they are present in very small amount; but whenever any irritation acts upon the cornea they at once increase considerably in number, since they escape from the network of blood-vessels forming the marginal loops and pass into the cornea. These cells play an important part in inflammation of the cornea.

4. *Descemet's membrane* (*D*, Fig. 10) is a homogeneous, hyaloid membrane which forms the posterior boundary of the cornea. Unlike Bowman's membrane, it is quite sharply separated from the stroma of the cornea, from which also it is chemically different. It is very resistant to chemical reagents, and likewise to pathological processes going on in the cornea. When the entire stroma of the cornea has broken down into pus, we often see the thin Descemet's membrane still for days offering resistance and remaining unimpaired (see § 532).

5. The *posterior epithelium* (endothelium, *e*, Fig. 10). This under the form of a single layer of flattened cells, coats the posterior surface of Descemet's membrane.

16. Divisions of Cornea.—The cornea at its margin abuts against three membranes, the conjunctiva, the sclera, and the uvea (iris and ciliary body). Embryology teaches us that the cornea consists of three superimposed layers, each of which corresponds to one of the membranes adjacent and may be said to represent its continuation over the most anterior portion of the eyeball. The cornea hence consists of three divisions—a conjunctival, a scleral, and a uveal. The conjunctival portion of the cornea (the so-called conjunctiva corneæ) consists of the anterior epithelium and of Bowman's membrane, and also in the marginal portions of the cornea of the most anterior lamellæ of the corneal stroma, which at the limbus merge into the tissue of the conjunctiva (Fig. 261, *L*). The most posterior lamellæ of the corneal stroma and Descemet's membrane, together with the posterior epithelium lining it, belong to the uvea, while the middle lamellæ of the cornea represent the continuation of the sclera (Waldeyer).

In the fully developed eye these three divisions are fused into a common whole, although their community of origin with the adjacent membranes still finds expression under pathological conditions; the conjunctival division suffering most of all in diseases of the conjunctiva, the uveal division in diseases of the uvea.

17. Blood Supply and Nerves.—The cornea contains no vessels. These cease at the margin of the cornea, forming there at the limbus the network of marginal loops which is supplied by the anterior ciliary vessels (see § 10, and Figs. 5 and 6). From the marginal loops the blood-plasma passes over into the stroma of the cornea.

The *nerves* of the cornea arise partly from the ciliary nerves, partly from the nerves of the bulbar conjunctiva. They are very numerous, particularly in the uppermost layers of the stroma, where they form a plexus and then pass forward through Bowman's membrane to form another plexus beneath the epithelium. From this second plexus fibres pass into the epithelium as far as the most anterior layers of the latter (Fig. 10, *n*) and terminate in end organs among the epithelial cells (Collins). The cornea is therefore extremely sensitive to the touch. In the induction of narcosis the reflex squeezing together of the lids that follows from touching the cornea is employed to test the depth of the narcosis, since this reflex act is among those that are the last to disappear. Lesions of the cornea are particularly painful whenever they affect the uppermost layers, which are

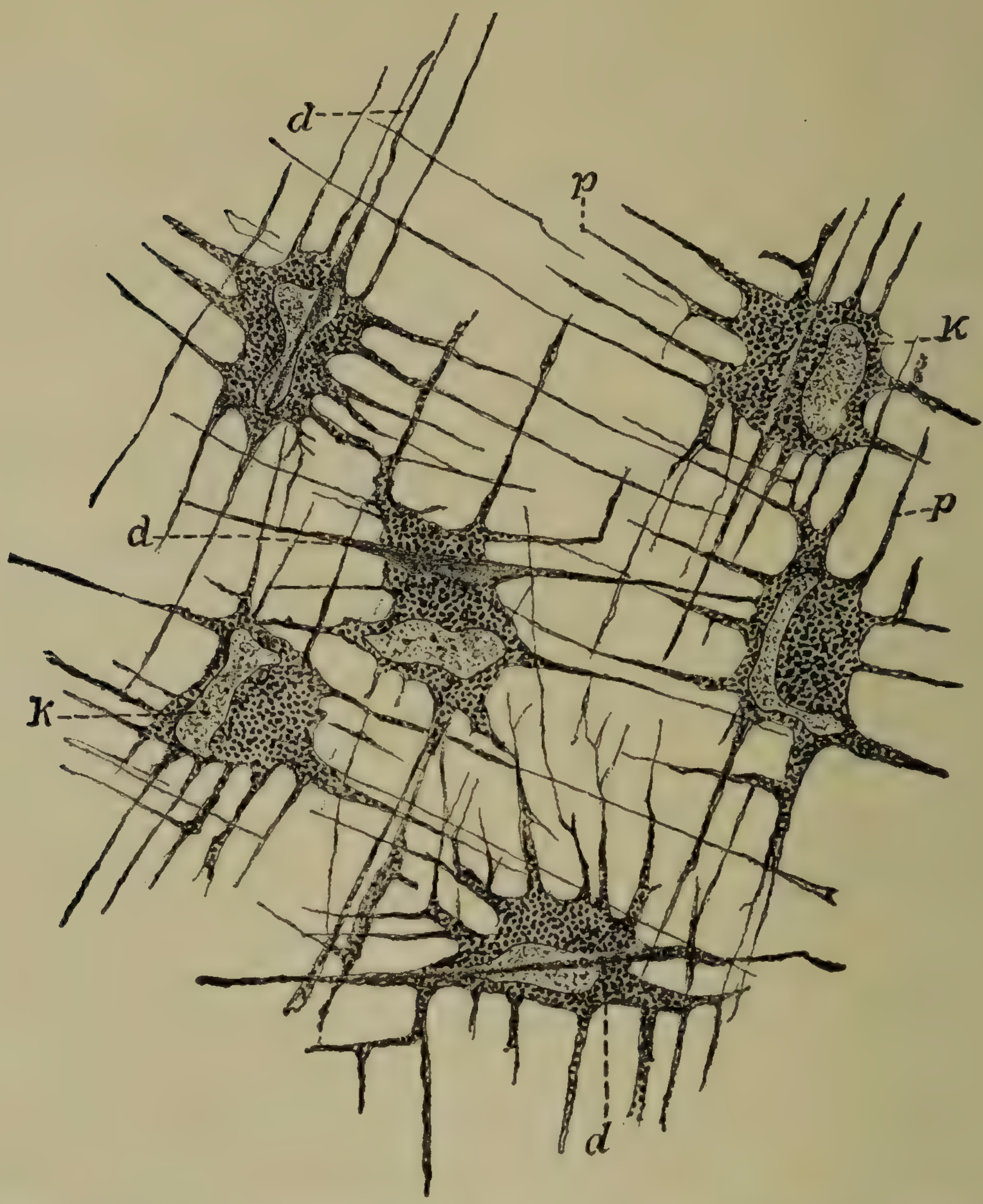

FIG. 11.—FIXED CORNEAL CORPUSCLES OF THE FROG. (After v. Ebner.) Magnified 325×1.

k, nuclei of the corneal corpuscles; *p*, processes of the cells, which either cross each other at right angles or anastomose with each other; *d*, pressure-ridges, so called because the way they are produced is that the cell protoplasm is squeezed into the gaps adjoining the lamellæ, and being compressed there are moulded into a rectilinear, ridge-like shape.

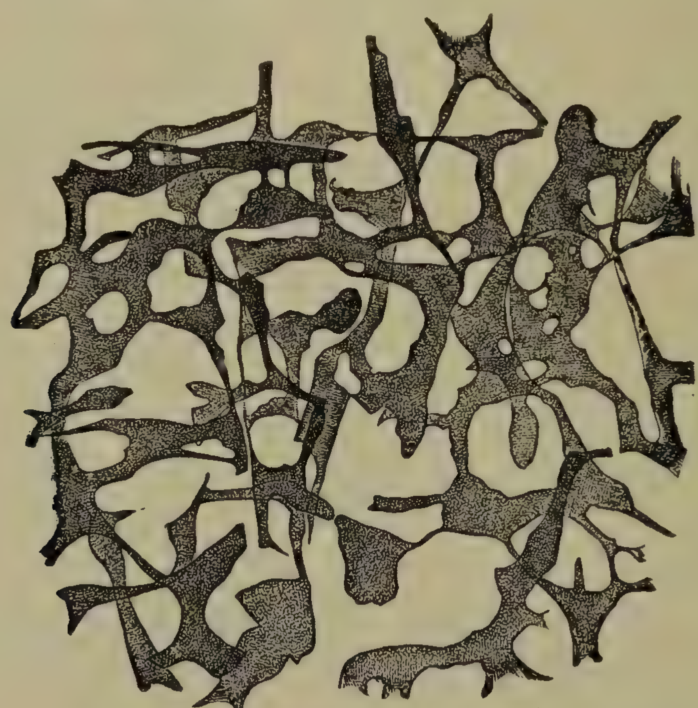

FIG. 12.—CORNEAL CORPUSCLES OF MAN. Magnified 145×1.

The description of corneal corpuscles ordinarily given is taken from the corneæ of animals, and it corresponds to the picture given in Fig. 11 of the frog's cornea. The figure here given is drawn from a section which was made through the cornea of an adult man parallel to its surface, and which was stained while still quite fresh with gold chloride. The section comprises several successive lamellæ and hence shows corneal corpuscles which lie in different planes, those which are drawn of lighter shade being the deeper. The processes of the cells are few and thick; the nuclei of the cells in this preparation are not stained enough to be visible. (Cf. Fig. 13.)

so rich in nerves, as, for instance, in the case of exfoliation of the epithelium, by which the numerous fibres of the epithelial plexus of nerves are laid bare.

II. Sclera

18. Structure.—The sclera[1], or opaque portion of the fibrous envelope of the eye, is thickest in its posterior segment, where it has a thickness of about 1 mm. It gradually diminishes in thickness anteriorly, becoming, however, somewhat thicker in the most anterior segment, because here the tendons of the recti muscles become fused with and reinforce it.

The sclera consists of fine fibrillæ of connective tissue, which are united into bundles. These run, generally speaking, in two directions—from before backward, (meridional fibres), and in a direction concentric with the margin of the cornea (circular or equatorial fibres). Between the bundles are found a few flat cells. The tissues of the sclera and of the cornea are very much alike, and, moreover, at the corneal margin

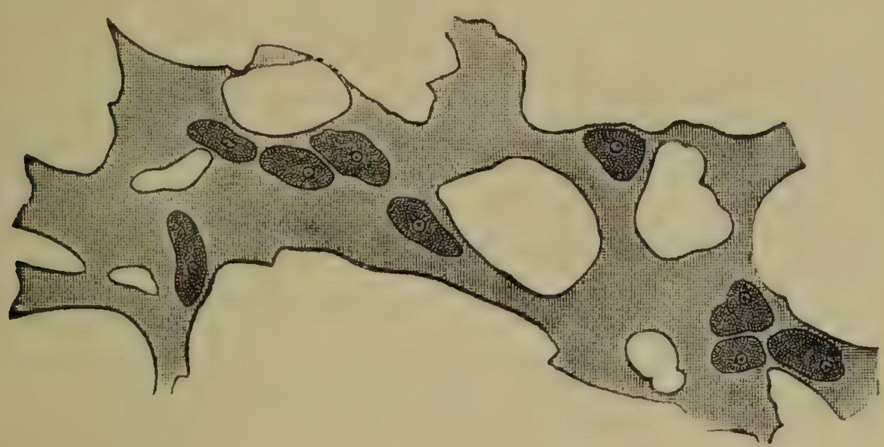

Fig. 13.—Corneal Corpuscles of Man. Magnified 250×1.

From another cornea, also stained while still fresh with gold chloride. The corpuscles represented all lie between the same two lamellæ and consequently in the same plane. They are connected by thick processes with each other and appear to form a single protoplasmic network, since the boundaries between the separate cell territories are not visible. The nuclei lie grouped together. (Cf. Fig. 12).

pass into each other without any sharp line of demarcation. They are chiefly distinguished by the arrangement of the bundles of fibres, which is much more regular in the cornea than in the sclera.

The sclera also contains branched pigment cells, which for the most part are met with only in its deep layers and also along the vessels and nerves that traverse it. In the living eye we often perceive the pigment in the spots where the anterior ciliary veins emerge from the sclera, these spots appearing as small brown dots upon the white membrane. Sometimes there is more marked pigmentation or the sclera looks bluish (see § 634).

The sclera is traversed by vessels and nerves which penetrate into the interior of the eye, but has itself very few vessels. On the other hand, there are numerous vessels contained in the so-called episcleral tissue, that loose connective tissue which envelops the sclera and in the anterior segment of the eye attaches the conjunctiva to it. In the posterior segment of the eye the optic nerve passes through the sclera, which here apparently has an aperture for the passage of the nerve(foramen scleræ). In reality, however, the inner layers of the sclera are continued as the lamina cribrosa through the foramen scleræ (Fig. 36; for more precise particulars, see § 47).

[1] From σκληρός hard.

CHAPTER III

THE INTERIOR REFRACTIVE MEDIA

I. Aqueous

19. The refractive media of the eye consist of the cornea in front, the aqueous filling the anterior chamber (see § 31) behind the cornea, the lens, and the vitreous. The aqueous is a watery liquid containing a trace of albumin and a small amount of salts. Normally these media are transparent, and are so constructed as to refract light rays accurately upon the retina (see Chapter XIII).

II. Lens

20. Gross Anatomy.—The lens (crystalline body, lens crystallina) lies between the iris and vitreous, and, together with the zonula, divides the eye into a smaller anterior and a larger posterior section—the cavity of the aqueous and the cavity of the vitreous. It is transparent and colorless structure of lenticular shape, the anterior surface of which is less, the posterior surface more curved. In the lens we distinguish an anterior and a posterior pole, and the rounded edge, or equator, where the anterior and posterior surfaces of the lens meet. The sagittal diameter (thickness of the lens) amounts in the adult man to 4 mm., the equatorial diameter to 9 mm.

The lens lies within the circle formed by the ciliary processes, but in such a way that its equator is distant about 0.5 mm. from the apices of the processes. The interspace between the ciliary body and the equator of the lens is called the circumlental space. The posterior surface of the lens is embedded in the fossa patellaris of the vitreous. The lens is kept in position by the suspensory ligament, or zonula cilaris.[1]

If after rupturing the zonula we take the lens out of the eye, we find it, in the first place, inclosed in a transparent capsule—the capsule of the lens. If after removing the capsule we try to crush the lens of an elderly man between the fingers, the softer peripheral masses separate, while the harder central portion remains uncrushed between the fingers. The former form the *cortex* the latter the *nucleus* of the lens (see Fig. 308 *r* and *k*). These are distinguished not only by their consistence but also by their color. The cortex is colorless, while the nucleus has a yellowish or brownish hue. The nuclear layers owe their greater consistency and also their coloration to a process which is known as sclerosis, and which consists mainly in a loss of water. The sclerosis begins even in childhood, but advances so slowly that it is not until the age of twenty-five that a distinct, although still small, nucleus is present.

Since sclerosis of the lens fibres is a change due to advancing age, it affects first the oldest fibres—i. e., those that lie in the centre of the lens. By a continuous progress of

[1] Synonyms: Zonula Zinnii, ligamentum suspensorium lentis.

the sclerosis from the centre to the periphery of the lens, the nucleus steadily increases in size as the years go on, and the cortex diminishes in like proportion, so that at length, at a very advanced age, almost the entire lens is converted into nucleus, or is sclerosed. There are many individual differences in this regard, so that persons of the same age have lenticular nuclei of different size. The size of the nucleus is of practical importance in the operation for cataract.

The sclerosed portion of the lens is hard and rigid, incapable of changing its shape. Hence, the further advanced the sclerosis of the lens is, the less able is the latter to make that alternating change in its shape which is requisite for the act of accommodation. For this reason the accommodative power diminishes with advancing age (presbyopia; see § 182).

The nucleus reflects more light than the non-sclerosed part of the lens. Hence, the pupil in elderly people, whose lens has a large nucleus, is no

Fig. 14.

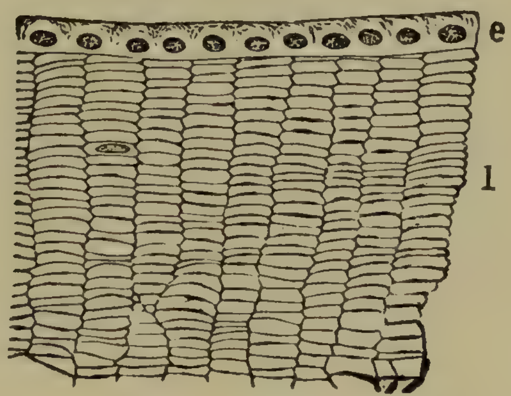

Fig. 15.

Fig. 14.—Capsular Epithelium of the Cortex. (After Rabl.) The epithelium which was left attached to the capsule when the latter was stripped off is seen in surface view. The area represented lies directly in front of the nuclear zone of the lens. In front of the latter the epithelial cells, which further forward are disposed irregularly (*u*), become arranged in regular meridional rows, *m*.

Fig. 15.—Equatorial Section Through a Human Lens. (After Rabl.) *e*, epithelial cells of the anterior capsule; *l*, cross section of the lens fibres which are arranged so as to form radiating lamellæ. In one of these fibres an oblong nucleus is visible.

longer of such a pure black as in youth. It gives a gray or grayish-green reflex (the senile reflex), which by the inexperienced is easily confounded with beginning cataract.

21. Histology of the Lens.—The external envelope of the lens is formed of the *lens capsule.* This is a homogeneous membrane (Fig. 16, *l*), which is thicker upon the anterior than it is upon the posterior surface of the lens. The anterior capsule of the lens is further distinguished by having a single layer of cubical epithelial cells, the epithelium of the lens (*e*, Fig. 16). This plays an important part in the growth of the lens, as the fibres of the latter originate from the cells of the capsular epithelium. If we follow the epithelium of the anterior capsule toward the equator, we see that at this point the epithelial cells, which were irregularly disposed before now get to be arranged in meridional rows (Fig. 14, *m*). Then these epithelial cells become taller and taller, until finally they are converted into long fibres, the fibres of the lens (Fig. 16 *f*). Since the latter have originated from meridional rows of epithelial cells, they are arranged in radiating lamellæ (Fig. 18), which explains why opacities of the lens so often occur under the

form of radial striæ. As the cells become elongated their nuclei recede from the capsule into the interior of the lens, so that a zone is found along the equator, in which there are numerous nuclei lying in the lens substance itself. This *nuclear zone*, as it is called (*k*, Fig. 16, cf. also Fig. 20, *k*), represents that district of the lens in which the growth of the latter takes place. This growth occurs by a process of apposition, new epithelial cells constantly growing out into lens fibres, which are placed outside of and next to the older lens fibres. In this way the lens acquires a concentrically laminated as well as a radial structure. The fibres lying in the centre of the lens are thus the oldest, and the

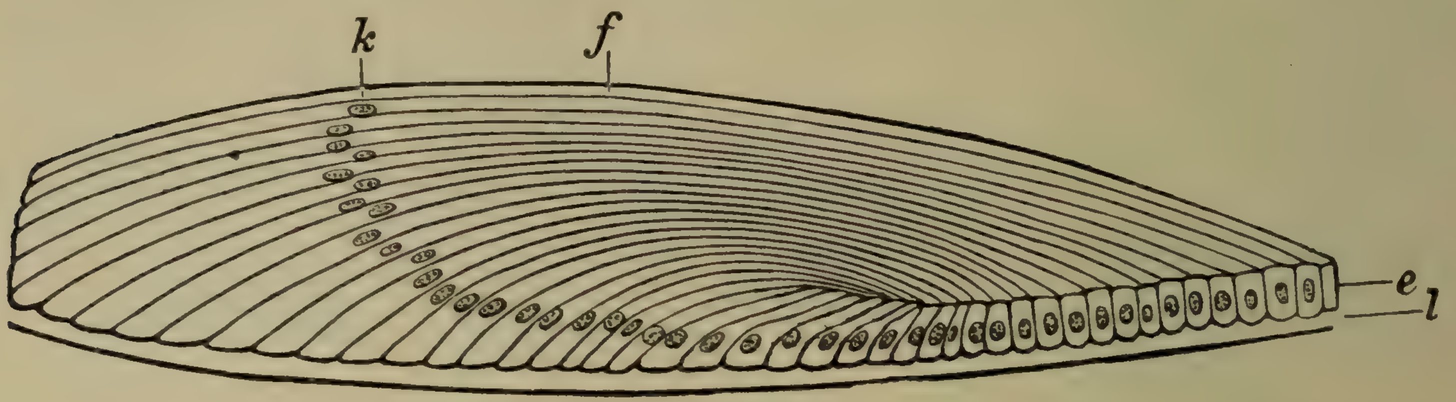

FIG. 16.—NUCLEAR ZONE OF THE LENS. (After Babuchin.)
l, lens capsule. The epithelial cells, *e*, by a process of gradual elongation, grow out into the lens fibres, *f*, with the nuclei, *k*.

most exterior fibres are the youngest. The reason for nuclei not being present outside of the nuclear zone in the interior of the lens is that the nuclei disappear from the older lens fibres.

The lens consist of fibres having the form of long, prismatic, six-sided cords. They are closely applied to each other, and are held together by a cement substance. The fibres begin and end upon the anterior and posterior surfaces of the lens, along lines which radiate from the anterior and posterior poles (Figs. 17 and 18). Here they form a Y-shaped figure—the stellate figure of the lens—which can be recognized even in the living eye in adults by means of lateral illumination. The three rays of the stellate figure branch, and thus divide the lens into a number of sectors (see Fig. 18) whose apices meet in the region of the anterior and posterior poles of the lens. In pathological cases—i. e., in opacities of the lens—the sectors often stand out very distinctly. The fibres of the nucleus are distinguished from those of the cortex by being slenderer and having edges that, owing to the shrinking of the fibres, are finely crenated; there is, however, no sharp line of distinction between the nucleus and cortex.

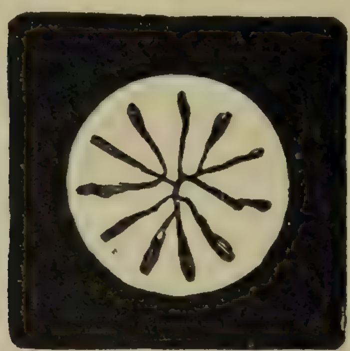

FIG. 17.—STELLATE FIGURE OF THE POSTERIOR SURFACE OF THE LENS. DRAWN FROM A LENS HARDENED IN MÜLLER'S FLUID. Magnified 2×1.

From the posterior pole of the lens start three primary rays, one of which is directed straight downward, the other two inward and upward and outward and upward. These divide into their branches so near their origin, in this case, that the Y-shaped figure is here not at once obvious.

22. Development.—The structure of the lens is easy to understand, when we know its development. The lens springs from the ectoderm, which becomes invaginated so as to form a vesicle (*L*, Fig. 54). Since the coating of cells upon the posterior wall of the vesicle grows out and is used up in the formation of lens fibres (Fig. 54), no such coating is found in this situation later on; hence, the posterior capsule of the lens has no epithelium. By this outgrowth of cells and their transformation into long fibres the vesicle is filled up so as to form a solid sphere. In this sphere each of the newly formed fibres extends from the anterior to the posterior lens capsule (Fig. 55). Similiarly in the adult lens each individual fibre stretches from a ray of the posterior to a ray of the anterior lens-star. The subsequent growth of the lens by apposition continues, as in the case of other epithelial structures, during the entire life. But while in the other epithelial structures (e. g., the epidermis, hair, and nails) the exfoliation of the oldest cells serves to maintain a state of equilibrium, no such ex-

foliation is possible in the lens, which is completely shut in; and in this case compensation takes place by a diminution in volume of the oldest fibres through a process of shrinking (formation of the lens nucleus). This diminution in volume, however, does not fully offset the appositional growth, so that the lens keeps on enlarging even in advanced life. In the sixty-fifth year of life it has about one-third more volume than in the twenty-fifth (Priestley Smith).

23. Zonula Ciliaris.—The zonula ciliaris consists of delicate, homogeneous fibres, which take their origin from the inner surface of the ciliary body, beginning at the ora serrata. The fibres at first keep in contact with the surface of the ciliary body (*z*, Fig. 20), but leave it at the apices of the ciliary processes, and, becoming free, pass over to the edge of the lens (free portion of the zonula; z_1, Fig. 20). As they do this, they diverge so that some go toward the very equator of the lens, others in front of the equator and behind it, to reach the lens capsule, with which they become fused. The space, triangular on cross

A B

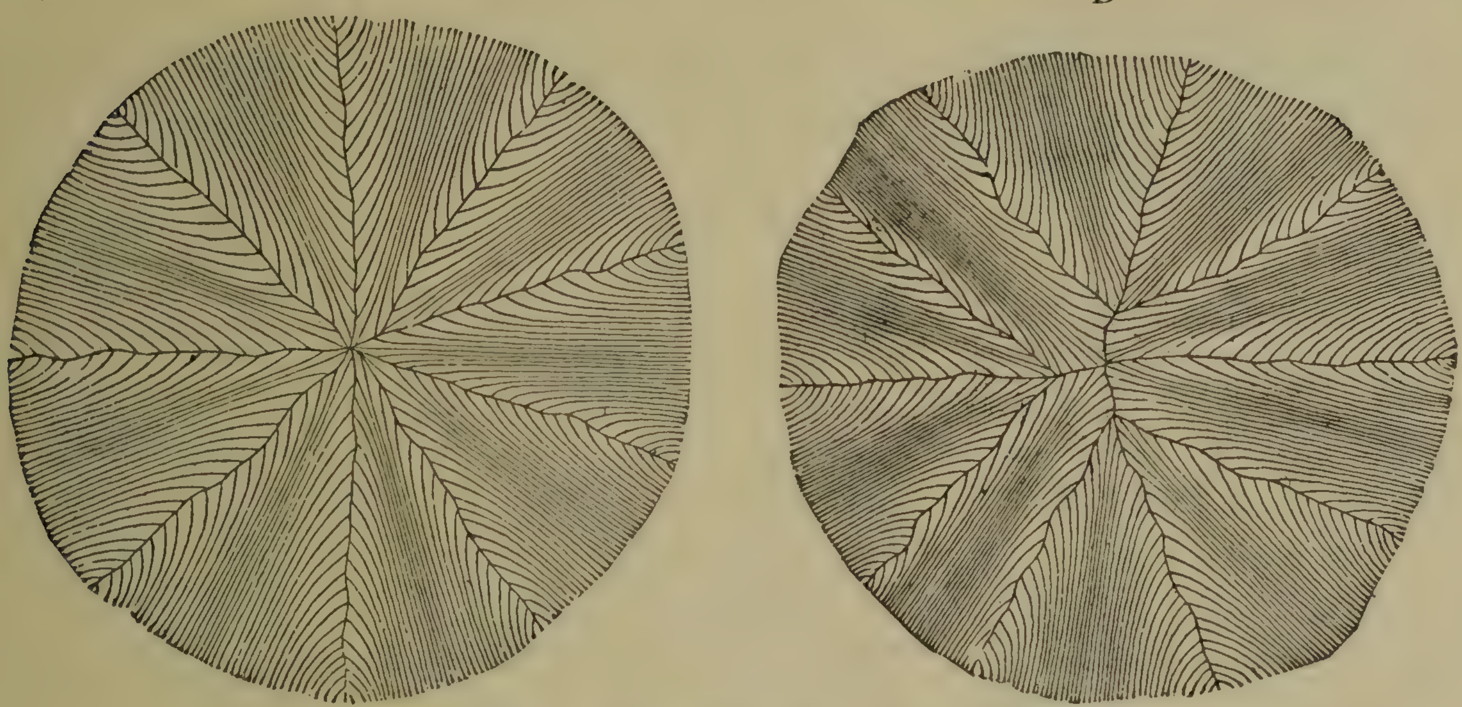

Fig. 18.—Lens-Star. (After Babuchin.) From Norris and Oliver.
A, central portion of anterior lens-star; B, central portion of posterior lens-star. In both figures are seen the lens fibres terminating in the lines of junction of the sectors.

section, included between the fibres of the zonula and the equator of the lens, is called the canal of Petit (*i i*, Fig. 20). It is connected with the posterior chamber by means of slit-like gaps between the separate fibres of the zonula.

III. Vitreous

24. The vitreous (corpus vitreum) is a transparent, colorless, gelatinous mass which fills the posterior cavity of the eye. On its anterior aspect it has a depression (the fossa patellaris), in which rests the posterior surface of the lens. By its other aspects the vitreous is applied to the inner surface of the ciliary body and the retina.

The vitreous is intimately adherent to the pars citiaris (§30) but not to the retina proper. It consists of a clear liquid substance containing cells (vitreous corpuscles) which have a varying (rounded or branched) shape and are found particularly in its outer layers. In its periphery, especially, in the vicinity of the ciliary body, it shows evidence of fibrillation and is enveloped in a homogeneous membrane (hyaloid membrane). Some regard both fibrils and hyaloid membrane as artefacts, but evidence from pathology seems to prove their existence (Lister).

The vitreous in the fully developed eye contains no blood-vessels or at most a remnant of the hyaloid artery. The hyaloid canal, which in the fetus, lodges the hyaloid artery (§ 58) sometimes persists and apparently serves as a lymph channel (§ 78 and cf. § 672).

CHAPTER IV

THE UVEAL TRACT

25. The Uvea.—If we carefully remove the sclera and cornea from an eyeball, we have presented to us the iris, ciliary body, and chorioid in connection. Together these form the middle tunic of the eye, which takes the shape of a sphere, colored dark brown by the pigment which it contains. In front this has a large aperture, the pupil; behind, it has a small one, the opening designed for the transmission of the optic nerve. On account of the similarity of the dark sphere, hanging upon the optic nerve as upon a stalk to a grape (*uva*), the middle tunic of the eye has received the name of *uvea*, and also of uveal tract.

I. Iris

26. Gross Anatomy.—The iris[1] is a disc-shaped membrane, perforated in the centre by the pupil.[2] By its peripheral or ciliary border it springs from the anterior surface of the ciliary body. From this point it stretches over the lens, its central or pupillary border lying upon the anterior capsule, and gliding upon it with the movements of the pupil (Fig. 20). By lying in this way upon the lens, the iris obtains a firm support. Hence, when the lens is absent or has lost contact with the iris, the latter is seen to tremble or vibrate with movements of the eyeball (tremulousness of the iris, *iridodonesis*[3]). Since the umbo of the lens lies farther forward than the spot where the iris originates in the ciliary body, the iris forms a shallow cone, whose apex, directed forward, is cut off short by the pupil. The shallower the anterior chamber becomes through advancement of the lens, the greater is the altitude of this cone; if, on the other hand, the lens is absent, the iris extends in a plane.

In looking at the iris with the naked eye, or, still better, with the magnifying glass, we recognize in it delicate markings, which are formed by elevations and depressions of its anterior surface (relief of the iris, Fig. 19). Sharp and clear in the normal eye, these markings are blurred or absolutely indistinguishable in an inflamed or atrophic iris, so that they constitute an important sign in iridic affections. The markings are chiefly formed by radially directed, projecting ridges, which are nothing but the blood-vessels lying in the stroma of the iris, and running from the ciliary to the pupillary margin. Near the latter they interlace with a ring of circular ridges—the lesser circle (circulus minor) of the iris (*k*, Fig. 19). This latter divides the iris into two zones: that lying to the periphery of the circulus minor is the ciliary zone (*C*); that lying to the central side of it is the much narrower

[1] Iris on account of its rainbow shape, not on account of its color.

[2] Pupilla properly means girl; perhaps so called because in the pupil ones sees a diminutive image of himself reflected from the cornea. So, also, in old German works the pupil is named "Kindlein" (= little child). In Greek, too, the pupil is called κόρη, girl, from which the expressions corectopia corelysis, etc., are derived.

[3] From *iris* and δονέομαι, I vibrate.

pupillary zone (*P*), which is often distinguished from the ciliary zone by a different coloration. Along the circulus minor may be noticed pit-like depressions (crypts, *c*) in the surface of the iris. Similar but much smaller openings in the anterior surface are also present at the periphery of the iris, close to its root; but these are not perceived in the living eye, partly because they are too small partly because they are concealed by the margin of the sclera, which projects in front ot them. It is only in blue eyes, especially in children, that this peripheral perforated zone becomes apparent as a dark, almost black, circle (*p*) close to the root of the iris. The pupillary margin of the iris is seen to be lined by a narrow black fringe (*r*), which stands out with especial prominence in eyes affected with cataract; for it contrasts much more forcibly with the white background of the clouded lens than with the black of the pupil of a normal eye.

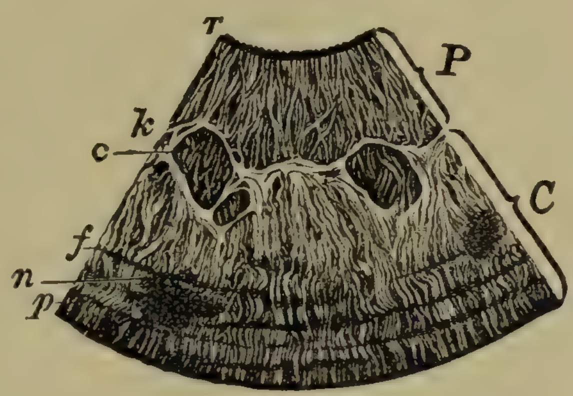

Fig. 19.—Anterior Surface of the Iris. Magnified 6×1.

P, pupillary zone; *C*, ciliary zone; *r*, fringe of retinal pigment; *k*, lesser circle; *c*, crypt; *f*, contraction groove; *n*, nævu;s *p* peripheral dark zone.

The examination of the iris in the living eye shows us, besides the details of relief mentioned above, a number of concentric curved lines near the ciliary margin of the iris (*f*, Fig. 19). They are particularly well seen in a dark iris with a contracted pupil, when owing to their light color they show off well upon the brown background. These are the *contraction furrows* of the iris; so called because, as the iris becomes narrower during the dilatation of the pupil, its anterior surface is disposed in folds, and depressions between the folds (*f*, *f*, Fig. 20) form the furrows in question, at the bottom of which the stroma of the iris generally contains less pigment. When the pupil contracts, these folds are smoothed down, and the furrows open out and are then easier to be seen.

With the varying dilatation and contraction of the pupil we also notice a change in the rim of pigment upon the pupillary margin: the more contracted the pupil is, the broader this becomes; on the other hand, when the pupil is greatly dilated, it disappears entirely. (Compare Fig. 20 with Fig. 219.)

When the pupil is very much contracted, we not infrequently observe even in normal eyes a faint tremulousness of the iris (iridodonesis), which otherwise occurs only in dislocation of the lens. This is due to the fact that with a contracted pupil the posterior chamber is deeper and at the same time the greatly dilated iris is considerably thinned—circumstances both of which favor wavering of the iris.

27. Microscopical Anatomy.—The iris consists essentially of the *vascular layer*. This is composed of (1) radially arranged blood-vessels; (2) a plexus of nerves; (3) the stroma which fills the interstices between the other constituents. The vessels have a thick adventitia, the nerves a neurilemma of like structure, and both are surrounded by branched pigmented cells (chromatophores), which with a very delicate fibrillated interstitial substance constitute the *stroma* (Fig. 301). Adjoining the pupil the vascular layer in its posterior layers lodges the sphincter pupillæ a flat band of smooth muscular fibres, 1 mm. broad (Fig. 20, *sp*).

On the anterior surface of the iris the cells are especially dense forming the *anterior limiting layer* (Fig. **301**, *v*). Upon this lies a layer of *endothelium*. This and the anterior limiting membrane are deficient only at those spots which correspond to the crypts, including both those at the pupillary (Fig. 20, *cr*) and those at the ciliary margin (*c*, *c*).

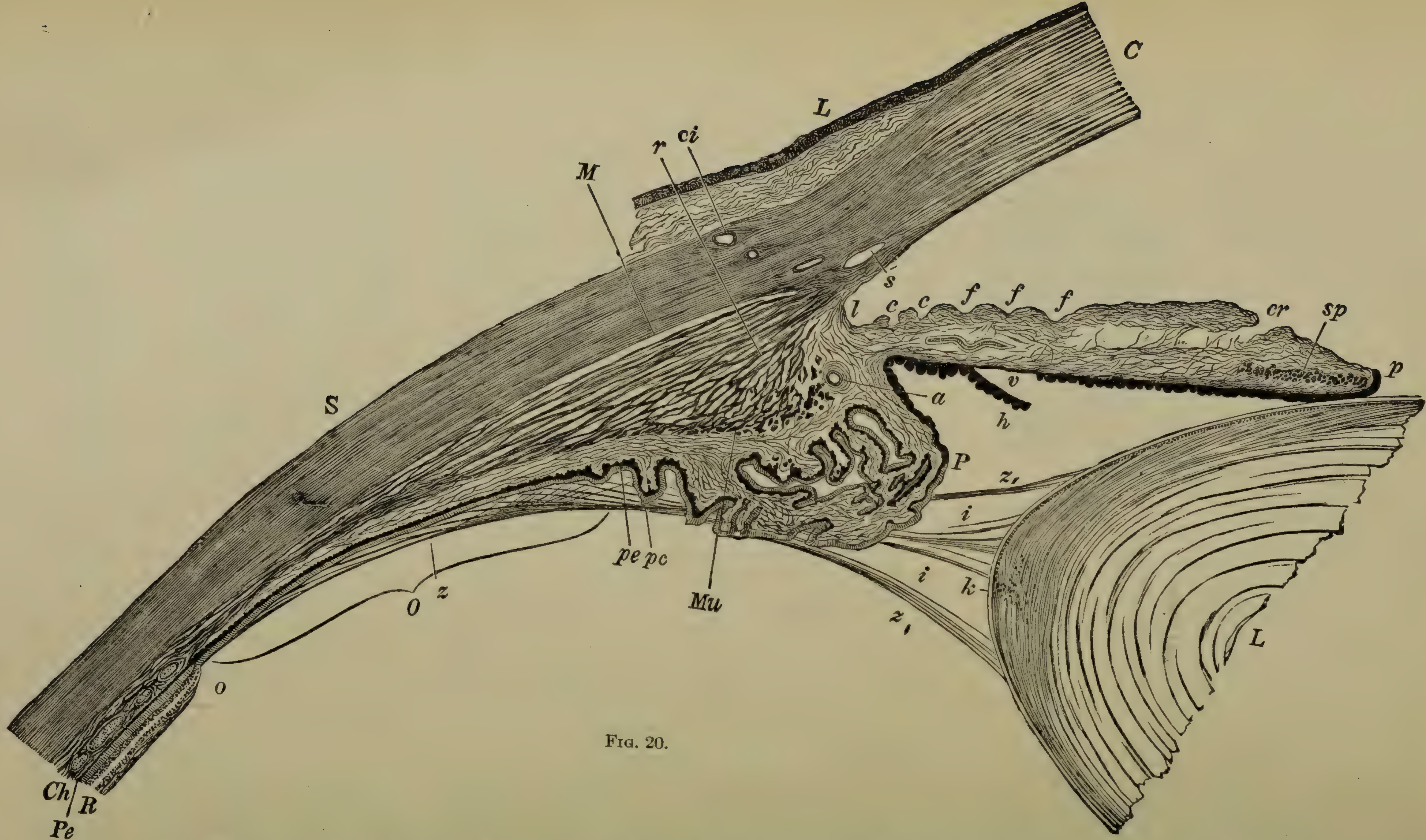

FIG. 20.

EXPLANATION OF FIG. 20.—MERIDIONAL SECTION THROUGH THE ANTERIOR PORTION OF THE EYE. Magnified 16×1.

The boundary between cornea, *C*, and sclera, *S*, is marked at its posterior surface by the cross section of Schlemm's canal, *s*. Anteriorly it is covered by the limbus conjunctivæ, *L*; farther back the cross section of an anterior ciliary vein, *ci*, is seen in the sclera. The iris is attached by the ligamentum pectinatum, *l*, to the inner posterior wall of Schlemm's canal. Since the pupil in this eye was contracted, the iris is long and thin, and its pupillary border is drawn out into a thin edge (cf. Fig. 219). On the anterior surface of the iris may be recognized the orifices of the crypts both in the circulus minor (*cr*) and in the periphery (*c*), also the contraction furrows, *f*, *f*. The posterior surface of the iris is covered with a sheet of retinal pigment which turns forward sharply like a spur at the pupillary margin, *p*. At one spot the posterior layer, *h*, of the pigment has separated so that the anterior layer, *v*, can be seen isolated. Close to the pupillary margin, the cross section, *sp*, of the sphincter pupillæ is visible. From the posterior wall of Schlemm's canal rises the ciliary muscle, consisting of longitudinal fibres, *M*, and circular fibres, *Mu*; the transition from one portion to another is effected by the radial fibres, *r*. At the anterior margin of the circular portion is seen the cross section of the circulus arteriosus iridis major (*a*). Upon the ciliary muscle are situated the ciliary processes, *P*, which are covered by the two layers of the pars ciliaris retinæ—namely, by the pigmented cellular layer, *pe*, which is the continuation of the pigment epithelium, *Pe*, and by the non-pigmented layer, *pc* the continuation of the retina proper, *R*. The flat part of the ciliary body, the orbiculus ciliaris, *O*, extends to the ora serrata, *o*, where the chorioid, *Ch*, and the retina, *R*, begin. Upon the orbiculus lie the fibres of the zonule of Zinn, *z*, which farther forward pass into the free portion, z_1, of the zonula and there inclose the cavity of the canal of Petit *i*. The lens, *L*, shows at its equator besides the attachments of the zonular fibres, the cross section, *k*, of the ring of nuclei.

These crypts, therefore, form apertures which lead into the interior of the tissue of the iris and place its tissue spaces in free communication with the cavity of the anterior chamber. This arrangement favors the rapid change in volume of the iris in the alternating movements of the pupil, since it enables fluid to pass quickly from the tissue of the iris into the anterior chamber and vice versa.

The posterior surface of the stroma of the iris is covered by the dilatator pupillæ and the pigment epithelium. The *dilatator pupillæ* consists of ectodermal muscle cells, i.e. of cells originating from the outer layer of the ocular cup (Vialleton, Grynfeltt, etc). Each of these cells in its inner portion (head) preserves the character of an epithelial cell and its original pigmentation while its outer portion, or base, which is directed toward the iris, develops into a radially directed smooth muscle fibre. In section (Fig. 21) therefore, the dilatator apparently consists of two layers, an anterior, finely striate and destitute of pigment and nuclei (Bruch's membrane, posterior limiting membrane, *m*), and a posterior, consisting of nucleated and pigmented spindle cells (anterior pigment layer, *v*). Behind this layer lies the posterior pigment layer or the *pigment epithelium* of the iris (*h*). This consists of large, densely pigmented cylindrical cells, which are arranged in a single layer but which jut out in round prominences. At

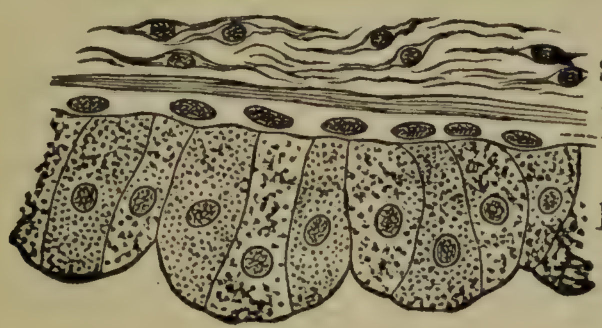

FIG. 21.—POSTERIOR LAYERS OF THE IRIS. MERIDIONAL SECTION THROUGH THE IRIS OF AN ADULT, DECOLORIZED BY FICK'S METHOD. Magnified 300 X 1.

To the posterior layers of the stroma of the iris, *s*, with its branched cells, succeeds the dilatator or the posterior limiting membrane, *m*, whose posterior surface is lined by the protoplasm and the oblong nuclei of the anterior stratum *v*, of the retinal pigment layer. In this stratum the boundaries of the individual cells cannot be recognized. The posterior stratum, *h*, of the retinal pigment layer consists of elevated cylindrical cells with round nuclei. The cells are of different heights and are so grouped that their posterior surface in section forms a series of rounded eminences such as also are to be found on a smaller scale in the iris shown in Fig. 20. These eminences represent the cross section of the dark brown annular projections, running concentrically with the pupil, which the iris presents on its posterior surface when examined with the naked eye. The posterior border of this layer of cells is characterized by its peculiarly sharp contour, which does not, however, in this spot assume the appearance of a true limiting membrane. The cells of the posterior layer still contain a few pigment granules which have escaped decolorization.

the margin of the pupil the pigment epithelium passes into the pigment layer, which here preserves its purely epithelial character. At this spot the pigment epithelium turns round the edge of the pupil so as to show somewhat on the front of the iris (Fig. 20, *p*) forming the black rim that we see there encircling the pupil (Fig. 19, *r*).

The pigment epithelium readily separates from the dilatator, both in microscopical specimens (Fig. 20, *h*) and in the living eye (in the case of rupture of synechiæ, § 678). The dilatator and the pigment epithelium originate from the anterior border of the fetal ocular cup, and are hence designated as the retinal layers of the iris (*pars retinalis iridis, pars iridica retinæ*). The remainder of the iris which springs from the mesoderm, is denoted by the name *pars uvealis iridis*.

28. Pigmentation of the Iris.—The *color* of the iris, which is either light (blue or gray) or dark (brown), is caused by the iridic pigment. There are two kinds of pigment in the iris: one lies in the branched cells of the stroma, and is hence called the stroma pigment; the other fills up the epithelial cells of the retinal pigment layer (retinal pigment). Upon the proportion between the amount of pigment deposited in these two the color of the iris depends. The retinal layer of the iris alway abounds in pigment, while the amount of stroma pigment that the iris contains varies greatly. When the

stroma contains little pigment, the retinal pigment shows through the thin iris, and appears blue. This is due to the same phenomenon that causes a dark background always to appear blue when looked at through a more or less opaque medium. Thus, for instance, through a delicate skin the veins look blue. If the stroma is deficient in pigment, but pretty thick and compact, the iris appears gray. And, finally, the greater the amount of brown stroma pigment that the iris contains, the more this pigment becomes visible and makes the iris appear brown, while the retinal pigment layer, which lies behind, is more and more concealed by the stroma pigment and withdrawn from view.

Not infrequently in an iris, that is but slightly pigmented as a whole, one or two isolated accumulations of pigment are found in the stroma. These then stand out as dark (rust-colored, brown, or black) spots in an otherwise gray or blue iris (*nævi iridis, n* in Fig. 19 and Fig. 219). The presence of a pretty large number of them gives the iris a mottled appearance. Exceptionally, cases occur in which the iris has no pigment either in its stroma or in its retinal layer. Such an iris is found in *albinos;* it is translucent, and, on account of its numerous vessels, has a delicate, grayish-red color.

As is universally known, the *color of the iris changes* in the first years of life. Most children are born with a deep-blue iris. The stroma contains but little pigment and is still very thin, so that the posterior pigment layer is seen through it, having a bluish look. With increasing age the stroma becomes thicker and thicker. If, while this is taking place, the pigmentation does not increase, the iris simply becomes of a light blue or gray; but if, simultaneously, there is an increase of the pigment of the stroma, the iris takes on a brown color. The transformation of a blue iris into a brown one is sometimes confined to a part of the membrane, so that a brown sector is seen in an otherwise light-colored iris. So too, in albinism the lack of pigment may be confined to a sector (Middleton). Moreover, the iris of one eye may be blue and that of the other brown. This is called *heterochromia iridis* and is often a pathological condition (see §708).

The color of the iris is always proportioned to the pigmentation of the rest of the body. The dark races always have a dark iris.

II. Ciliary Body

29. Gross Anatomy.—The ciliary body [4] (corpus ciliare) forms a zone, about 5 mm. broad nasally and above, 6-7 mm. broad temporally and below (Fig. 2). Its anterior border is attached to the sclera behind Schlemm's canal; posteriorly it merges with the sclera in such fashion that only on its inner surface is any clear separation between the two distinguishable. In meridional section it appears nearly triangular, the shortest side of the triangle looking forward and toward the axis of the eye and presenting in about its centre the root of the iris. Its outer surface, which is attached to the sclera, presents the *ciliary muscle* in the form of a band 3 mm. broad, which posteriorly is colored uniformly brown like the chorioid. The inner surface is turned toward the vitreous and can be brought into view by cutting the eye in two and removing from it vitreous, lens, and retina (Fig. 2). The spot where the retina is torn away anteriorly is marked by a jagged line—the ora serrata (*o, o,* Fig. 2). Corresponding to this there is a change in the coloration of the uvea, which behind this line is brown (chorioid), in front of it black (ciliary body). At the anterior margin of the black zone rise the

[4] From *cilia,* lashes, because of the fine, radiating folds. The ciliary body is also called *cyclon* (hence cyclitis) from κύκλος, a circle.

ciliary processes, about seventy in number. These are conspicuous not only by their prominence, but also because of their lighter color, their apices being less strongly pigmented than are the depressions between them. The anterior zone of the ciliary body, about 2.5 mm. broad, bearing the ciliary processes, is called the folded part of the ciliary body—*corona ciliaris* (*c*, Fig. 2); back of this is the posterior part of the ciliary body—pars plana or orbiculus ciliaris (*or*)—which is smooth and of a uniform black color.

30. Microscopical Anatomy.—The *ciliary muscle* consists of smooth muscular fibres, arranged in bundles. These bundles form a reticulum, whose interstices are filled with fibrillated connective tissue and chromatophores. At the outer side of the muscle these interspaces are directed meridionally, insomuch that all the muscle fibres run almost exactly in a meridional direction and parallel to the inner surface of the sclera (*meridional portion;* Fig. 20, *M*). Further inward, the trabecular structure of the muscle is more in evidence, the interspaces being broader, and in the main the muscular bundles run in a divergent radiating direction toward the inner surface of the ciliary body (*radial portion;* Fig. 20, *r*). Lastly, quite at the anterior and inner aspect, the inter-

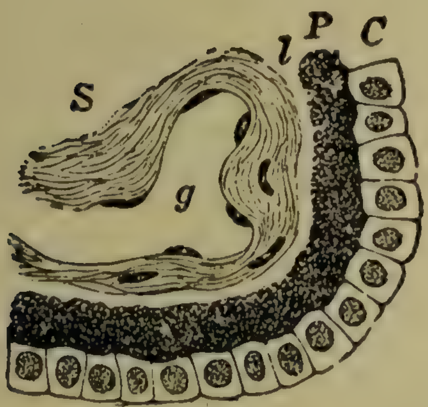

Fig. 22.

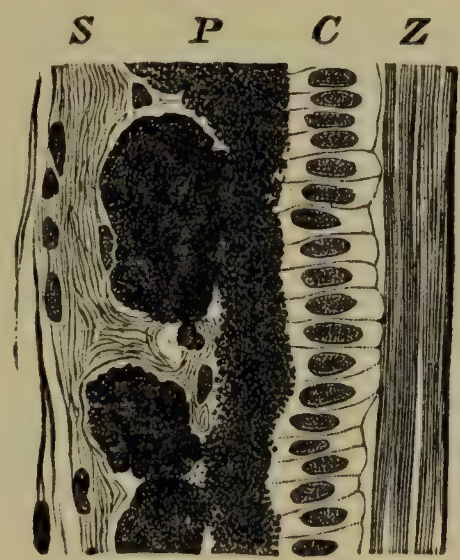

Fig. 23.

Fig. 22.—Portion of a Meridional Section through a Ciliary Process, somewhat Behind its Apex. Magnified 240×1. The stroma of the ciliary process, *S*, consists of delicate connective tissue in which lie the broad and very thin-walled blood-vessels, *g*. Succeeding these are the vitreous lamina, *l*, and next the two layers of the pars ciliaris retinæ. One is the pigment layer, *P*, representing the continuation of the pigment epithelium; its cells, on account of their deep pigmentation, disclose neither their nucleus nor their contour. The second layer, *C*, which is unpigmented, consists of a single stratum of cubical cells, representing the continuation of the retina proper.

Fig. 23.—Superficial Layers of the Flat Portion (Orbiculus Ciliaris) of the Ciliary body in Meridional Section. Magnified 270×1. Taken from the same section as Fig. 22. The cells of the pigmented layer, *P*, of the ciliary body dip into the stroma, *S*, of the latter in the form of processes which are club-shaped or gland-shaped (but contain no gland cavity). The light area surrounding them represents the vitreous lamina, which in this case is but indistinctly visible. The cells of the superficial non-pigmented layer *C* are here longer and cylindrical. On their free surface lie the fibres, *Z*, of the zonule of Zinn.

spaces take a markedly circular direction, so that these bundles in a meridional section appear cut straight across (*circular portion;* Fig. 20, *Mu*). The meridional and radial portions are known as Brücke's portion (tensor chorioideæ of Brücke). The circular portion, the amount of which varies greatly in individual cases, is called Müller's portion. (Wallace of New York described both portions before Brücke and Müller.)

On the inner side of the ciliary muscle is the vascular layer of the ciliary body. This consists of numerous blood-vessels which are imbedded in a delicate stroma of connective tissue, permeated with chromatophores (Figs. 22, 23, *S*). The *ciliary processes* are likewise composed of a vascular layer, so that the corona ciliaris is the most vascular portion of the whole eyeball. On the inner surface of this vascular layer lies a homogeneous membrane (the lamina vitrea of the ciliary body; Fig. 22, *l*); then follows a layer of richly pigmented cells (pigment epithelium; Fig's 22, 23, *P*); and lastly a layer

of non-pigmented cylindrical (Fig. 23, *C*) or cubical (Fig. 22, *C*) cells. The two layers last mentioned originate from the ocular cup, and together are denoted as the *pars ciliaris retinæ.*

III. Aqueous Chamber

31. Anterior Chamber and Ligamentum Pectinatum.—The anterior chamber is 2.5 to 3 mm. deep on an average, growing gradually narrower toward the periphery (Fig. 57). Its depth varies greatly according to the age, the size of cornea, the refraction, etc., and in the same eye varies as a result of the accommodation (see § 177). It is bounded in front by the cornea; behind by the iris and in the pupillary area by the lens. Its most peripheral portion is called the angle or sinus of the chamber.

The topographical and histological relations of this region are of great significance for the physiology and the pathology of the eye. Since the iris is not connected with the corneo-scleral margin directly, but merges with the inner layers of the ciliary body, the bottom of the sinus is formed by the anterior surface of the latter (Fig. 20, at *l*). Hence, at this spot inflammatory products may get from the ciliary body into the anterior chamber directly, without being obliged to make their way through the pupil. New growths, too, may enter the anterior chamber by this route (Fig. 325). In front and exteriorly the anterior chamber is lined by a peculiar tissue, which once universally and still pretty often is denoted by the rather unsuitable name of *ligamentum pectinatum*[5] but is also called the *trabecular* structure of the sinus (H. Virchow).

In meridional section (Fig. 24), this appears as a triangular mass of tissue, the apex of which is applied to the most posterior layers of the cornea. Its inner aspect is turned toward the sinus of the chamber; the outer surface in part lies on the cornea, in part forms the wall of an oblong, always patent canal, Schlemm's canal (Fig. 20, *s*). The base of the triangle in part abuts on the sclera, and in fact on a process of it which projects like a spur at the posterior wall of Schlemm's canal (*scleral spur* or process); partly passes directly into the ciliary muscle or by its innermost layers passes along the anterior surface of the ciliary body into the root of the iris. The scleral or sclero-corneal portion, which goes to the sclera and ciliary muscle, consists of a number of fenestrated lamellæ; (Fig. 25) the uveal (sclero-ciliary) portion, which goes to the iris forms a meshwork of very delicate fibres. All parts are covered with endothelium, and this is continuous on one side with the endothelium of the cornea and on the other with the endothelium of the anterior surface of the iris. All spaces within the trabecular structure therefore are in direct communication with the sinus of the chamber.

32. Schlemm's Canal.—This separates the cornea and sclera. Accordingly, the most anterior zone of the sclera belongs to the region of the anterior chamber—a fact which is of importance for the understanding of typical rupture of the sclera and for the performance of a number of operations. Schlemm's canal is an annular canal, which in places is divided into several separate passages. It is not in direct communication with the spaces in the ligamentum pectinatum but with the anterior ciliary veins. Leber hence calls it the sinus venosus scleræ; but under normal conditions, as ophthalmoscopic examination of the sinus of the chamber shows, it does not contain blood but only aqueous.

33. Posterior Chamber.—The posterior chamber is made by the stretching of the iris bridgewise over the ciliary processes and peripheral

[5] The name *ligamentum pectinatum* was given by Hueck to a structure which may be present in the horse but not in man. For the same reason the term Fontana's spaces for the gaps in the trabeculum is not a fitting one. On the other hand, the word "trabeculum" used as a translation for Virchow's term "Gerüistwerk" is not Latin.

portions of the lens (Figs. 20, 57). It is an annular space, triangular in cross section and growing deeper towards the periphery; banded in front by the iris, peripherally by the ciliary processes, behind by the fibres of the zonula and by the lens. In communication with it are the spaces in the free portion of the zonula (Fig. 20, z_1), which were formerly known as Petit's canal, and also the very narrow spaces which extend along the

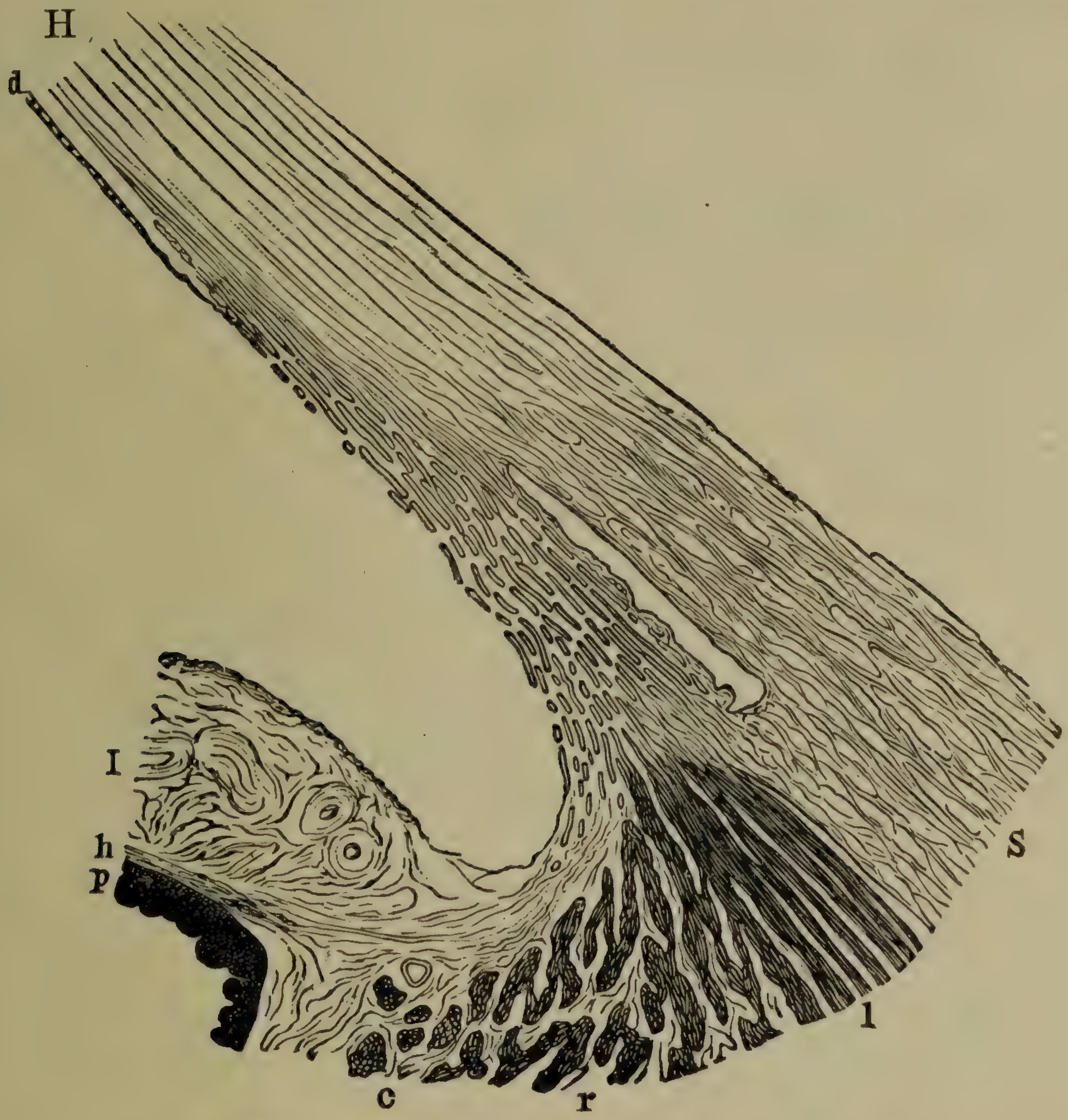

Fig. 24.—Sinus of the Anterior Chamber. Magnified 83×1.

H, the most posterior layers of the cornea; *S*, those of the sclera. The boundary between the two is marked by the elongated lumen of Schlemm's canal. *d*, Descemet's membrane, with its endothelium. It ends in a thinned-out edge and just before it comes to a stop the lamellæ of the cornea which lie directly in front of it can be seen to become fibrillated and to merge into the anterior lamellæ of the ligamentum pectinatum. The ligamentum pectinatum forms the posterior wall of Schlemm's canal and apparently consists solely of short fragments of fibres because the lamellæ which bound the fenestræ (see Fig. 25) have been cut transversely by the section. At the posterior end of Schlemm's canal is seen a bundle of circularly directed scleral fibres (represented of lighter tint in the drawing), which are divided transversely by the section, and jut out obliquely toward the sinus of the chamber (scleral spur). To the anterior surface of this spur are attached the anterior lamellæ of the ligamentum pectinatum, while its posterior surface serves as a point of attachment for the longitudinal bundles, *l*, of the ciliary muscle. The posterior lamellæ of the ligamentum pectinatum merge directly, without any interposition of scleral tissue, either into the longitudinal or into the radiating fibres (*r*) of the ciliary muscle; the most posterior (innermost) lamellæ of the ligamentum pectinatum curve backwards and are lost in the delicate tissue of the root of the iris. *c*, circular fibres of the ciliary muscle. *I*, uveal portion of the iris; *h*, dilatator pupillæ, which is detached from the pigment epithelium, *P*, and extends in a radial direction round the ciliary muscle.

zonula (Fig. 20, *z*) between the inner surface of the ciliary body and the vitreous as far as the orbiculus ciliaris (Fig. 20, *O*). The two chambers communicate only by means of the pupil (see § 75).

IV. Chorioid

34. Gross Anatomy.—The chorioid[5] is that part of the uvea which lines the posterior section of the eye from the ora serrata to the aperture for the optic nerve. If we observe it in situ, after opening the eyeball and removing the vitreous together with the retina, its inner surface appears smooth and uniformly brown. Then, if we try to strip if off from the sclera, we notice that at several spots it is attached more firmly than at others. The most intimate connection is at the margin of the aperture for the optic nerve; in addition, loose attachments exist in the places where vessels and nerves enter the chorioid from the sclera, and especially in the region of the posterior pole (region of the posterior ciliary arteries) and of the equator (venæ vorticosæ). When after tearing away these connections, we have

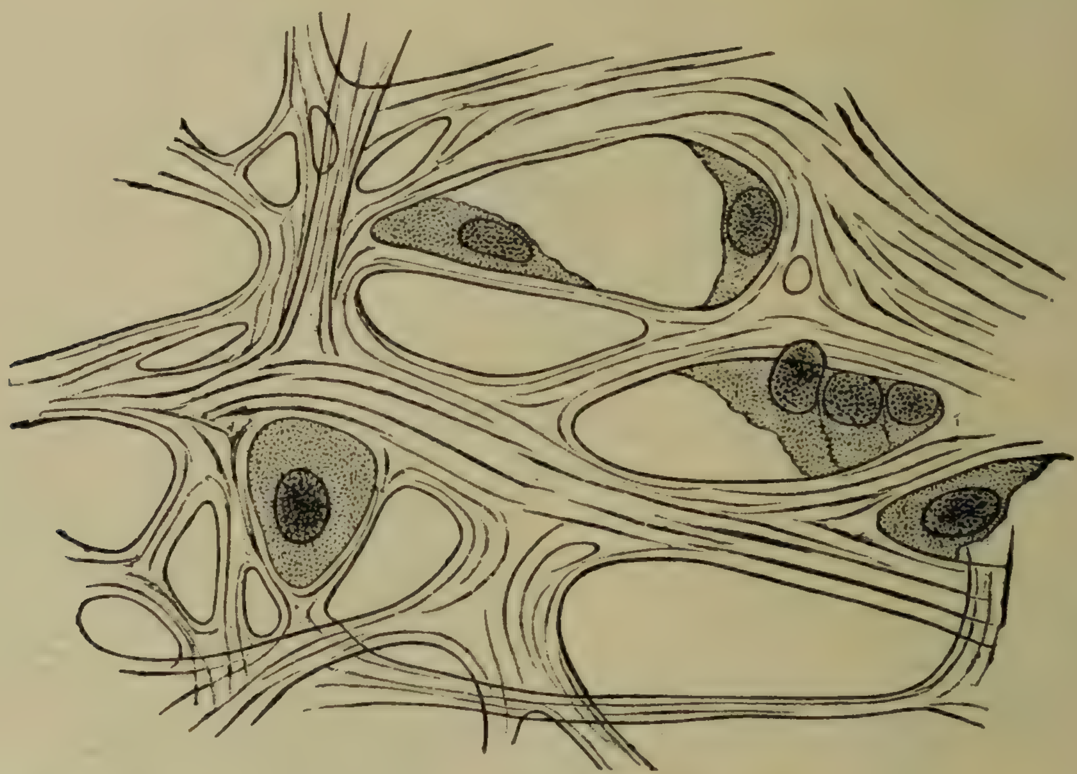

Fig. 25.—Scleral Portion of Ligamentum Pectinatum (Surface View). Magnified 700 × 1. Trabeculæ, which show a delicately fibrillar structure, inclose alveoli, the larger of which are elliptical, and directed so that their long axis lies parallel to the margin of the cornea. Upon the walls of these alveoli lie cells (endothelial cells) provided with nucleus and large protoplasmic cell body; small alveoli are sometimes entirely filled by such cells.

separated the chorioid from the sclera, we get a view of the outer surface of the chorioid, which has a shaggy appearance on account of the shreds of membrane adhering to it.

35. Microscopical Anatomy.—The chorioid consists of five layers which succeed one another in the following order, from without inward:

1. The *suprachorioid* (*s*, Fig. 26) consists of numerous minute non-vascular but richly pigmented lamellæ lying between the chorioid proper and the sclera (*sc*). Upon stripping these latter apart these lamellæ are torn in two, and are left hanging partly upon the inner surface of the sclera, partly upon the outer surface of the chorioid, which thus acquires the rough, shaggy aspect above mentioned.

2. The *layer of large vessels* (Haller) (*H*, Figs. 26 and 27). These are chiefly veins, which are placed very close to each other and anastomose repeatedly. The intervals

[5] From χοριοειδής—i. e., like the χόριον (=Lat., *corium*). This latter word signifies "skin," and not merely the epidermis, but also the envelope (chorion) of the embryo in utero; and, in fact, it is the latter that the chorioid resembles, from its abundant supply of vessels. This word is also erroneously written choroid or choroïd.

between the vessels (intervascular spaces) are richly supplied with pigment cells (*p*), and are hence of a brown color. This layer, accordingly, gives the same appearance upon a surface view as if we were looking at a plexus of bright lines (the vessels) upon a dark ground (Fig. 2). This is a picture which we often have the opportunity of seeing with the ophthalmoscope in the living eye (tessellated fundus, see Fig. 195).

3. The *layer of medium-sized vessels* (Sattler) (Fig. 26, *S*), which is very thin and but slightly pigmented.

4. The *layer of capillaries* (*chorio-capillaris*, —*R*, Figs. 26 and 27). This consists almost exclusively of capillaries which have a very wide bore, and at the same time are so closely packed together that the interspaces between the capillaries are often narrower than the capillaries themselves. This layer contains no pigment.

5. The *lamina vitrea* (or *lamina basalis* *G*, Figs. 26 and 27), a homogeneous membrane coating the inner surface of the chorioid.

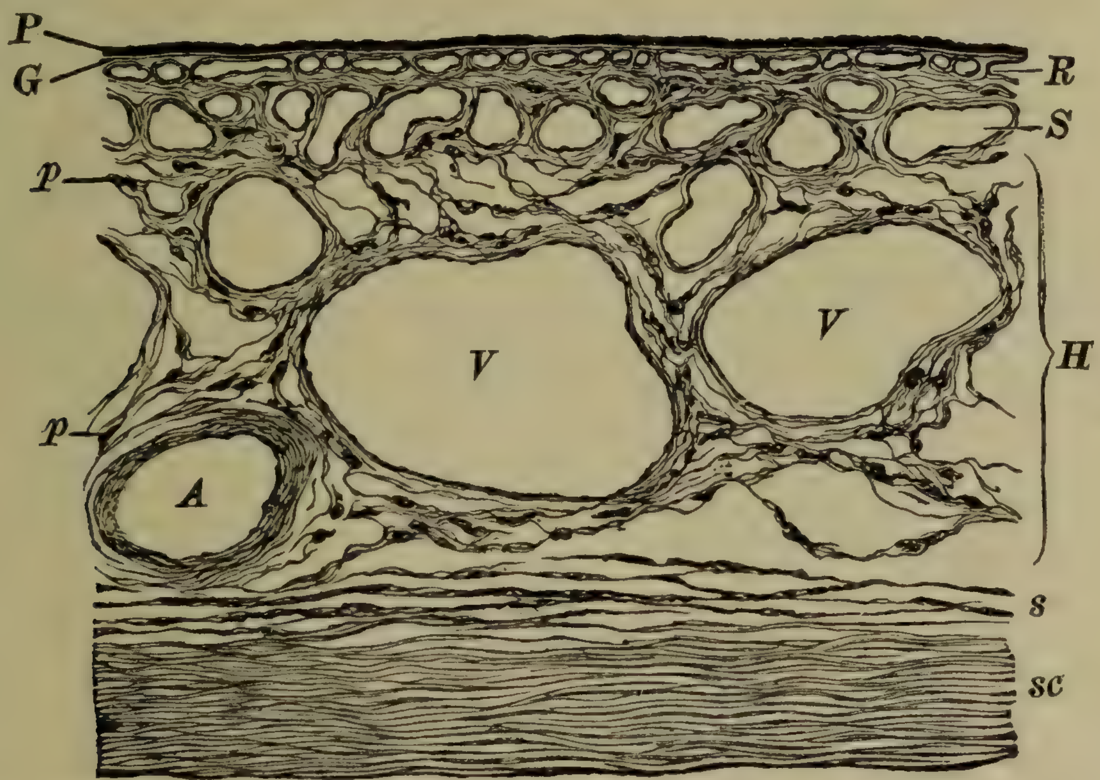

FIG. 26.—CROSS SECTION THROUGH THE CHORIOID. Magnified 175 × 1.

The chorioid consists of the suprachorioid, *s*, the layer of large vessels, *H*, the layer of medium-sized vessels, *S*, the chorio-capillaris, *R*, and the lamina vitrea, *G*. In the layer of large vessels are recognizable arteries, *A*, veins, *V*, and pigment cells, *p*. The inner surface of the chorioid is covered by the pigment epithelium, *P*, its outer surface by the sclera, *sc*.

36. Summary of Structure; Pigmentation.—We may briefly summarize the structure of chorioid as follows: The chorioid consists mainly of vessels which are arranged according to their calibre in three superimposed layers, in such a way that the largest vessels lie farthest to the outside, the smallest vessels farthest to the inside. The purpose of this arrangement is easily comprehended, since the chorioid is in great part designed for the nourishment of the retina which lies to the inner side of it. Hence the minutest vessels, the capillaries, from which the nutrient plasma of the blood exudes, must lie upon the inner surface of the chorioid. The vascular part of the chorioid is covered on either side by a non-vascular layer—i. e., on the outside by the suprachorioid, on the inside by the lamina vitrea.

All the layers of the chorioid, with the exception of the two innermost ones—the capillary layer and the lamina vitrea—contain *pigment* enclosed in branched pigment cells (chromatophores, Fig. 28). To its abundant supply of pigment the chorioid owes its dark-brown color. The inner surface of the chorioid is covered by the pigment epithelium (*P*, Fig. 26) which lies upon the lamina vitrea. This, too, was formerly reckoned in with the chorioid, because it remains attached to it after the retina has been

stripped off; embryological researches, however, have shown that it really belongs to the retina. It consists of regular hexagonal cells, each of which has an unpigmented nucleus, while the protoplasm contains an abundance of pigment granules (Fig. 29). From this the entire layer acquires a dark-brown color. The pigment of the chromatophores (stroma pigment) consists of small amorphous masses; the pigment granules in the cells of the pigment epithelium are short, rod-shaped structures, which should probably be regarded as small crystals, such as occur very distinctly in some of the lower vertebrates.

The pigmentiferous cells, including both of the chromatophores and the cells of the pigment epithelium, are similar in all eyes but the amount of pigment which they con-

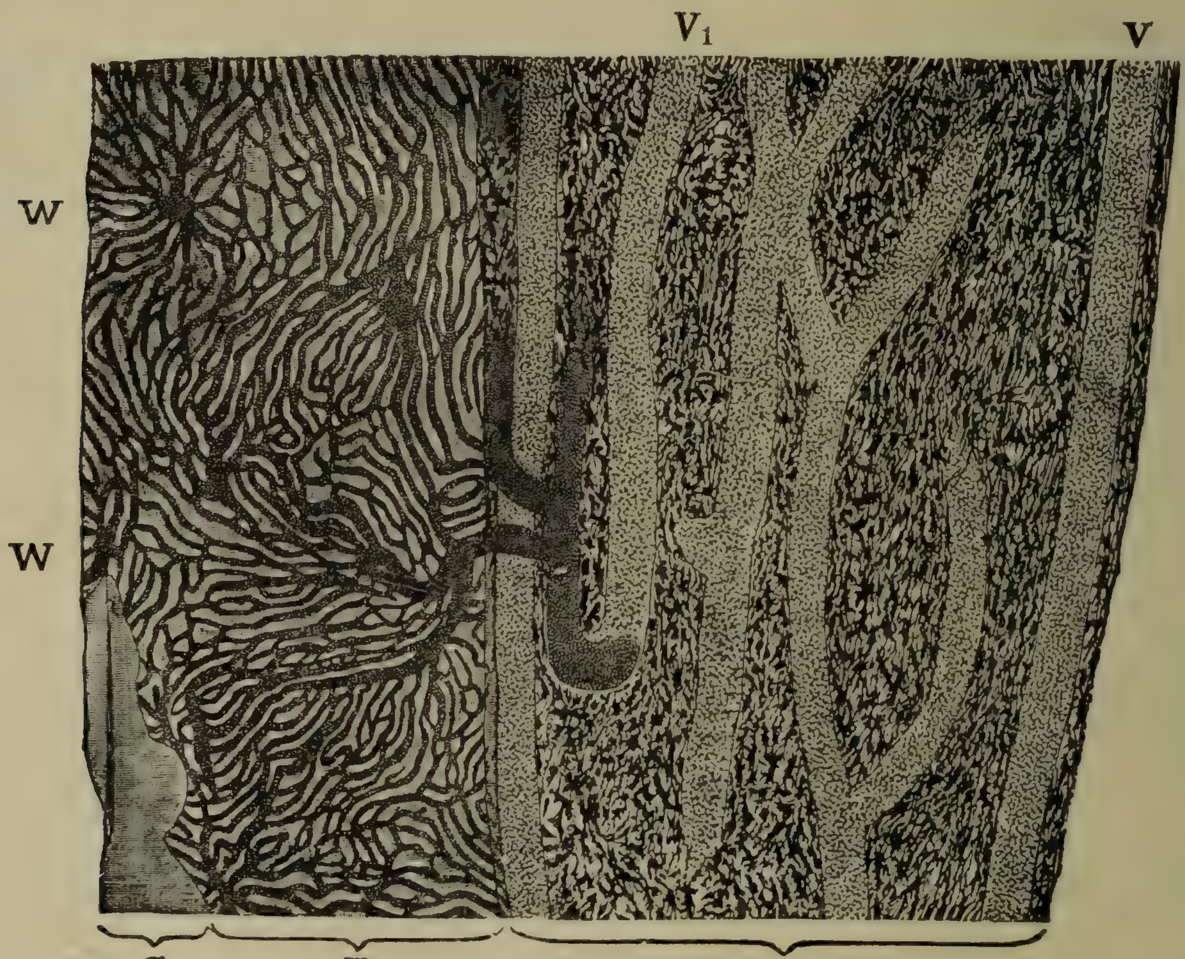

FIG. 27.—DISSECTION OF THE CHORIOID MADE PARALLEL WITH THE SURFACE. Magnified 27 × 1.

The suprachorioid has everywhere been stripped off and the pigment epithelium has been removed by penciling. Furthermore in the area marked *R*, the layer of large vessels was removed so that here the chorio-capillaris lies exposed. At *H* where the layer of large vessels is still present, the large veins, *V* V_1, which run approximately parallel to one another, appear like light-colored passageways, because the intervascular spaces are colored dark-brown by the presence of numerous stellate pigment cells. Some veins appear to have a blind ending, because at the point where they dip down into the opaque tissue they get out of view. The vein, V_1, bends on itself, enters the layer of medium-sized vessels, and here receives branches from the chorio-capillaris. This bent portion of the vein and also the capillaries are filled full of red blood corpuscles, while the large veins contain but few corpuscles. As a result of this natural injection the chorio-capillaris stands out with special distinctness. The distension with blood, however, is not everywhere alike, and for this reason the capillaries in the upper part of the drawing appear thinner and separated by wider intervals than in the lower part. So, too, the blind ending of some capillaries in the drawing is only apparent, being due to the fact that in this spot short segments of the capillaries are bloodless and hence invisible. The veins on their entrance into the chorio-capillaris at once divide into a number of capillaries and thus small vortices are produced. These are seen in the continuation of the vein, V_1 and also at *W W*; at the latter spot their continuation into a small vein cannot be seen because the latter was torn off when Haller's layer was removed. At *G* when Haller's layer was removed the chorio-capillaris also went with it, so that here the lamina vitrea alone is present; in making the preparation the latter was somewhat folded.

tain varies greatly. To this fact is due the great inequality in the pigmentation of eyes which is so evident with the ophthalmoscope (§ 410).

The chorioid is continuous with the flat part of the ciliary body, (pars plana or orbiculus ciliaris). The latter has essentially the same structure as the chorioid, and is distinguished fromit only by a somewhat different arrangement of the blood-vessels and by the absence of the chorio-capillaris, which ends at the ora serrata. The striking difference in color between the brown chorioid and the black orbiculus (Fig. 2) is attributable not to a difference in pigmentation of these parts of the uvea, but to a difference in the pigment epithelium which covers them.

V. Nerves of the Uvea

37. The uvea in all of its parts is very rich in *nerves*. The ciliary nerves get to the uvea by piercing the sclera near its posterior pole. They form in the chorioid, and particularly in the ciliary muscle, a dense plexus, in which numerous ganglion cells are intercalated. This iris is also very rich in nerves, but contains no ganglion cells. The iris and the ciliary body contain, in addition to the motor nerves designed for the ciliary muscle and the muscular apparatus of the iris, a very great number of sensory nerve fibres which arise from the trigeminus; hence, inflammation of these parts is frequently attended with great pain. The chorioid, on the contrary, seems to

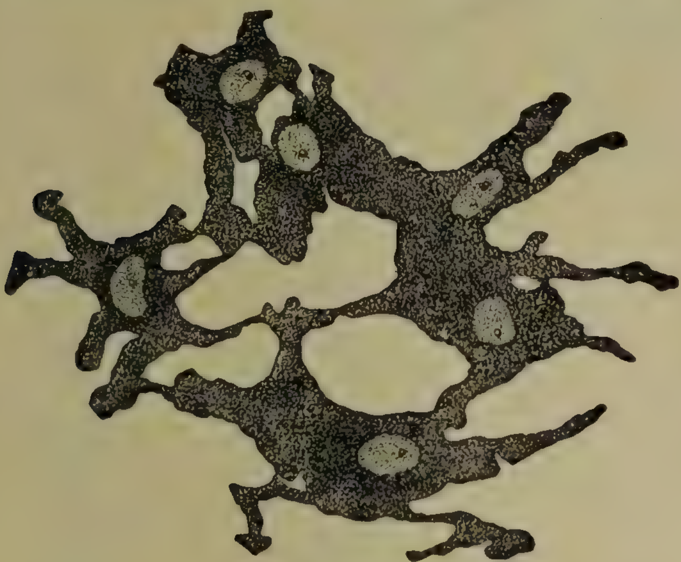

Fig. 28.

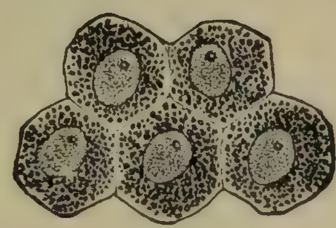

Fig. 29.

Fig. 28.—Chromatophores of the Chorioid. Magnified 400×1. They are branched, anastomosing, connective-tissue cells, with numerous pigment granules and an unpigmented nucleus.

Fig. 29.—Pigmented Epithelial Cells of the Retina. Magnified 500×1. They are hexagonal epithelial cells, with pigment granules and an unpigmented nucleus.

possess no sensory nerves, since inflammation of this membrane runs its course without producing any sensations of pain.

VI. Blood-vessels of the Uvea

38. Three systems of blood-vessels exist in the eye—that of the conjunctiva, that of the retina, and that of the uvea (ciliary system of vessels). The *arteries* of the ciliary system are: 1. The posterior ciliary arteries. These arise from the ophthalmic artery, and enter the interior of the eye through the sclera in the region of the posterior pole. The majority of them pass at once into the chorioid (short posterior ciliary arteries, *c*, *c*, Fig. 30 and *Ab*, Fig. 31). Two of them, however (the long posterior ciliary arteries, *d*, Fig. 30, *Al*, Fig. 31) run, one on the outer side, the other on the inner side, between chorioid and sclera as far forward as the ciliary muscle. Here each divides into two branches, which run in a direction concentric with the margin of the cornea, and unite with the branches of the artery of the opposite side to form an arterial circle, the circulus arteriosus iridis major (Fig. 30, *h*; Fig. 31, *Cima*; Fig. 20, *a*). This gives off the arteries for the iris, which extend radially from its ciliary to its pupillary margin (Fig. 30, *i*). Shortly before they reach the latter they form by anastomoses a second, smaller vascular circle, the circulus arteriosus iridis minor or the small circle of the iris (Fig. 30, *k*; Fig. 31, *Cimi*). 2. The anterior

ciliary arteries come from in front, arising from the arteries of the four recti muscles (Fig. 30, *e*; Fig. 31, *Aa*). They perforate the sclera near the margin of the cornea and help to form the circulus arteriosus iridis major. The short posterior ciliary arteries are therefore designed mainly for the chorioid,

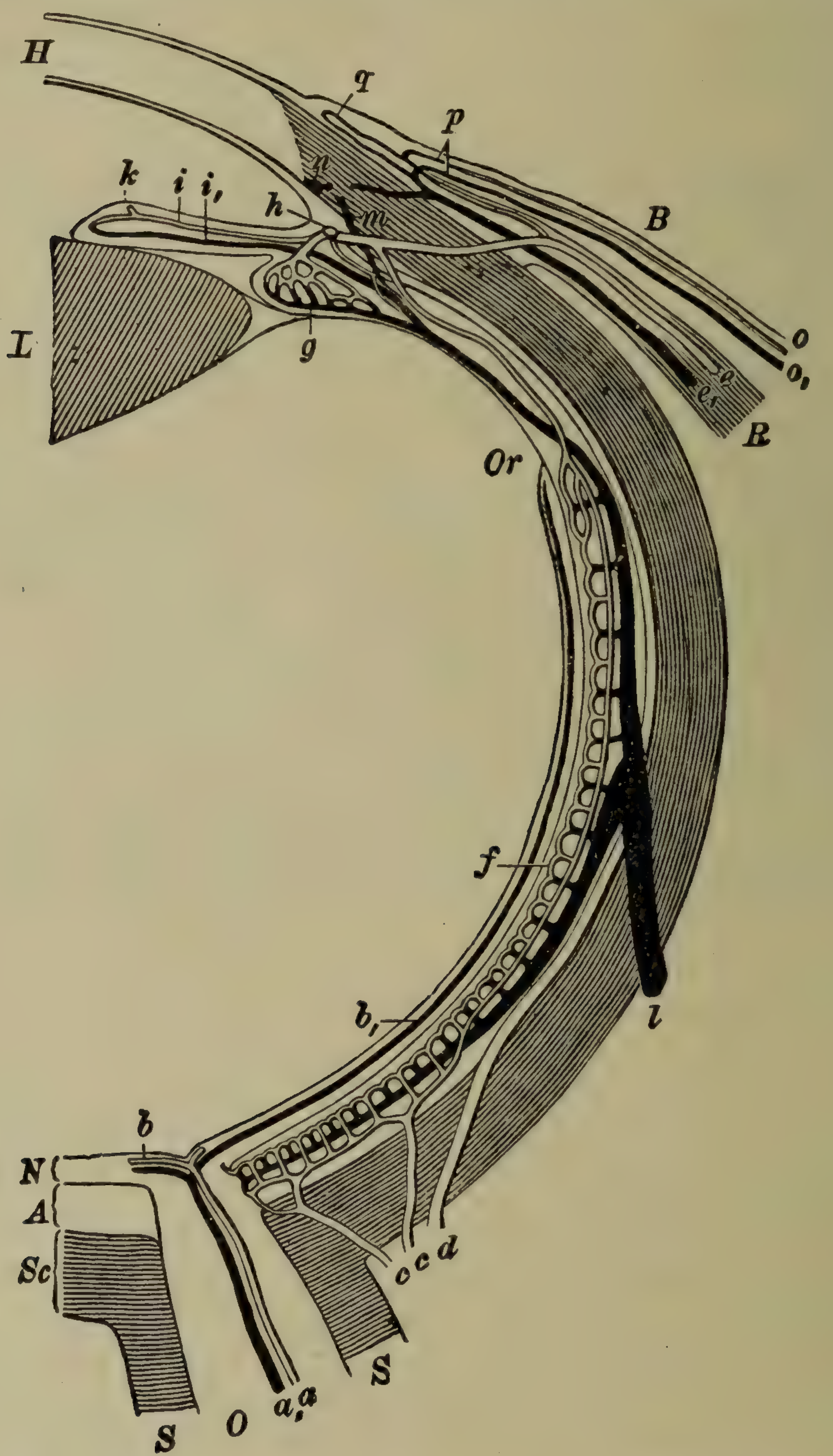

Fig. 30.—Blood-vessels of the Eye (Schematic). (After Leber.)

The *retinal system of vessels* is derived from the central artery, *a*, and the central vein, a_1, of the optic nerve, which give off the retinal arteries, *b*, and the retinal veins, b_1. These end at the ora serrata, *Or*.

The *system of ciliary vessels* is fed by the short posterior ciliary arteries, *c*, *c*, the long posterior ciliary arteries, *d*, and the anterior ciliary arteries, *e*. From these arise the vascular network of the chorioidal capillaries, *f*, and of the ciliary body, *g*, and the circulus arteriosus iridis major, *h*. From this last spring the arteries of the iris, *i*, which at the lesser (inner) circumference of the latter form the circulus arteriosus iridis minor, *k*. The veins of the iris, i_1, of the ciliary body, and of the chorioid are collected into the vasa vorticosa, *l*; some veins (*m*), however, that come from the ciliary muscle leave the eye as anterior ciliary veins, e_1. With the latter Schlemm's canal *n*, forms anastomoses.

The *system of conjunctival vessels* consists of the posterior conjunctival vessels, *o* and o_1. These communicate with those branches of the anterior ciliary vessels which run to meet them; that is, with the anterior conjunctival vessels, *p*, and form with these the marginal loops of the cornea, *q*. *O*, optic nerve; *S*, its sheath; *Sc*, sclera; *A*, chorioid; *N*, retina; *L*, lens; *H*, cornea; *R*, internal rectus; *B*, conjunctiva

the long posterior ciliary arteries and the anterior ciliary arteries for the ciliary body and the iris. Some recurrent branches of the latter, however, also supply a portion of the anterior segment of the chorioid.

The arrangement of the *veins* is essentially different from that of the arteries. In the chorioid the capillary network of the chorio-capillaris (Fig. 30, *f*.) is fed by the arteries directly. The blood from this flows off through a very great number of veins that keep uniting to form larger

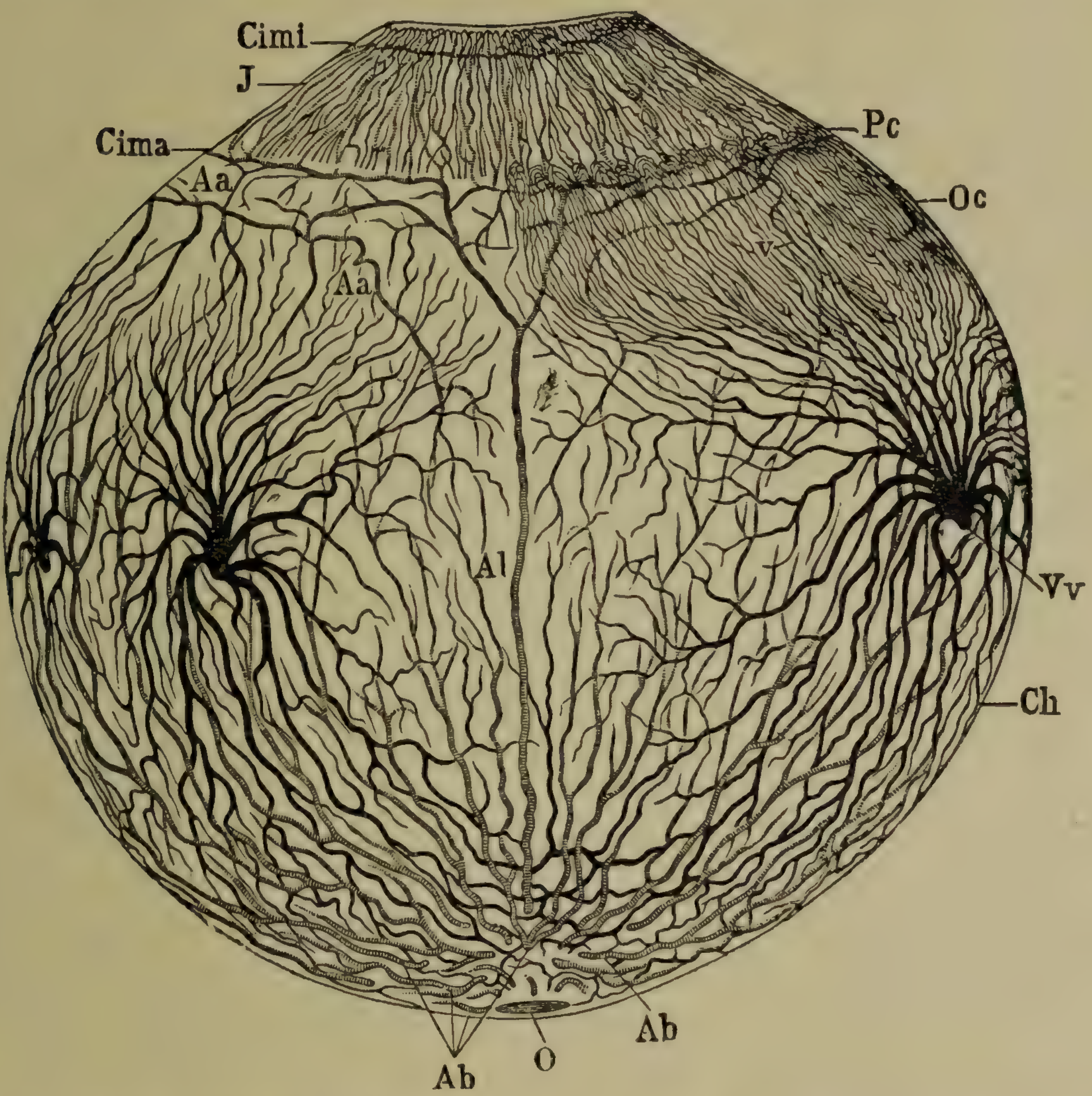

Fig. 31.—Blood-vessels of the Uvea (Ciliary System). (Schematic.) (After Leber.)

In the region surrounding the optic nerve, *o*, the short posterior ciliary arteries, *Ab*, enter the chorioid, *Ch*, and end in the anterior segment of the latter. In the horizontal meridian of the eye the long posterior ciliary artery, *Al*, runs to the orbiculus ciliaris, *Oc*, and here divides dichotomously to form the circulus arteriosus iridis, major, *Cima*. Branches of the anterior ciliary arteries, *Aa*, also take part in forming the circulus major, while other branches run backwards into the chorioid to meet the branches of the short posterior ciliary arteries. The circulus arteriosus iridis major gives off the radiating arteries of the iris, *J*, and these near the pupillary border of the iris form the circulus arteriosus iridis minor, *Cimi*. On the right side of the drawing the ciliary muscle is supposed to be detached, so that the abundant vascular network of the ciliary processes, *Pc*, comes into view. From the latter, numerous veins, *v*, run within the orbiculus ciliaris, at first parallel with each other, afterward converging, back to the venæ corticosæ, *Vv*, to which also pass the veins from the posterior segment of the chorioid.

trunks. A number of these trunks simultaneously converge to a common centre, where, consequently, a sort of whorl or vortex (Fig. 31, *Vv*) is produced by veins coming together from all sides. These vortices, the number of which amounts to four at least, usually more, lie somewhat behind the equator of the eye; from them are given off the venæ vorticosæ which, perforating the sclera in a very oblique direction, carry off the blood to the outside (Fig. 30, *l*).

In the ciliary processes the arteries break up into a great number of twigs, which pass over into thin-walled veins (*g*, Fig. 30). These constitute the greater part of the ciliary processes, which, accordingly, consist mainly of vessels. The larger veins (Fig. 31, *Vv*) which are formed by the union of these vessels, and also most of the veins of the ciliary muscle, pass backward to the venæ vorticosæ. The veins that run back from the iris (i_1, Fig. 30) likewise pass to the venæ vorticosæ. Hence almost all the venous blood of the uvea empties into the latter. A portion of the veins coming from the ciliary muscle (*m*, Fig. 30), however, take another course, as they pass out directly through the sclera and thus come into view beneath the conjunctiva, near the margin of the cornea (anterior ciliary veins, Fig. 30, e_1). In their course these correspond to the anterior ciliary arteries, but have a much smaller area of ramification; it is they that principally constitute the violet vessels which we see running backward beneath the conjunctiva (see Fig. 228) in ciliary injection or in stasis within the eyeball (glaucoma). The anterior ciliary veins anastomose with the conjunctival veins and also with Schlemm's canal.

All the arteries and nerves distributed to the uvea and the veins coming from it pass through special canals in the sclera. They are connected with the walls of these canals only by lax connective tissue, which may serve as a medium of exit for the intraocular fluid (§ 78). These canals, which are called *emissaries* and represent anastomoses between Tenon's space and the perichorioidal space, are most evident along the long posterior ciliary arteries.

CHAPTER V

SENSORY APPARATUS

39. The sensory apparatus of the eye consists of the retina, the optic nerve, and the central connections of the latter.

I. Retina

40. The retina is a thin membrane which in the living eye is perfectly transparent and of a purplish-red color. This latter depends upon the visual purple contained in the rods (Boll). After death the retina becomes very rapidly opaque, and, as at the same time the visual purple is bleached out under the influence of light, the retina in the eye of a cadaver appears under the form of a very frail white membrane.

There are two points that are particularly prominent in the retina when in situ. One is a small white disc, which lies to the inner side of the posterior pole of the eye, and from which the vessels of the retina emanate; this is the point of entrance or head of the optic nerve, the *papilla nervi optici*. The second spot occupies precisely the posterior pole of the eye, and is distinguished by its faint yellow color. It is hence called the yellow spot, the *macula lutea*. In its centre is found a small depression, the fovea of the retina, or *fovea centralis* (*f*, Fig. 2).

41. Structure.—If we try to lift the retina from the subjacent chorioid, we see that it is connected with its bed only at two places. One of these is the head of the optic nerve, the other is the anterior border of the retina. The latter is formed by a zigzag line, the *ora serrata* (Fig. 2, *o o*). This line, which also represents the boundary between the chorioid and ciliary body, extends farther forward nasally and above than it does temporally and below.

The retina consists of a series of *layers*. The innermost or fibre layer (1, Fig. 36; Fig. 32 A, IX; Fig. 32 B, X) arises from the optic nerve, the fibres of which spread out in all directions (see § 47). The outermost layer, that of the rods and cones (8, Fig. 36; Fig. 32 A and B, II) is the light-perceiving stratum of the retina. The retina is composed of two kinds of tissue, the nervous tissue (Fig. 32 B, I to X) and the sustentacular tissue (*neuroglia;* Fig. 32 B, 5, 6). The latter holds the extremely delicate nervous tissue in proper position and also insulates the nervous elements from one another. Internally it forms a sheet-like expansion (*membrana limitans interna,* Fig. 32 A, X), which separates the retina from the vitreous.

With regard to nutrition the layers of the retina are comprised in two groups, the *cerebral layer* (Fig. 32 B. IV to X), which is supplied by the central artery (§ 45) and the non-vascular visual epithelium (*neuro-epithelium,* Fig. 32 B, II and III), which is supplied by the chorio-capillaris.

42. Fovea Centralis.—The depression at the site of the fovea centralis arises from a thinning of the retina, the inner layers of the latter being here entirely absent. Furthermore, the retinal fovea is also distinguished by the fact that the most external layer here consists only of cones. The rods do not begin until at the border of the macula lutea, and as we pass toward

the ora serrata, grow more and more numerous, while the number of cones diminishes in like proportion.

43. Pigment Epithelium.—The membrane here described—the retina, in the narrower sense of the word—develops from the inner layer of the ocular cup (§ 57 and Fig. 54, *r*). From the outer layer of the vesicle (*p*, Fig. 54) is developed the pigment epithelium, which therefore must, on

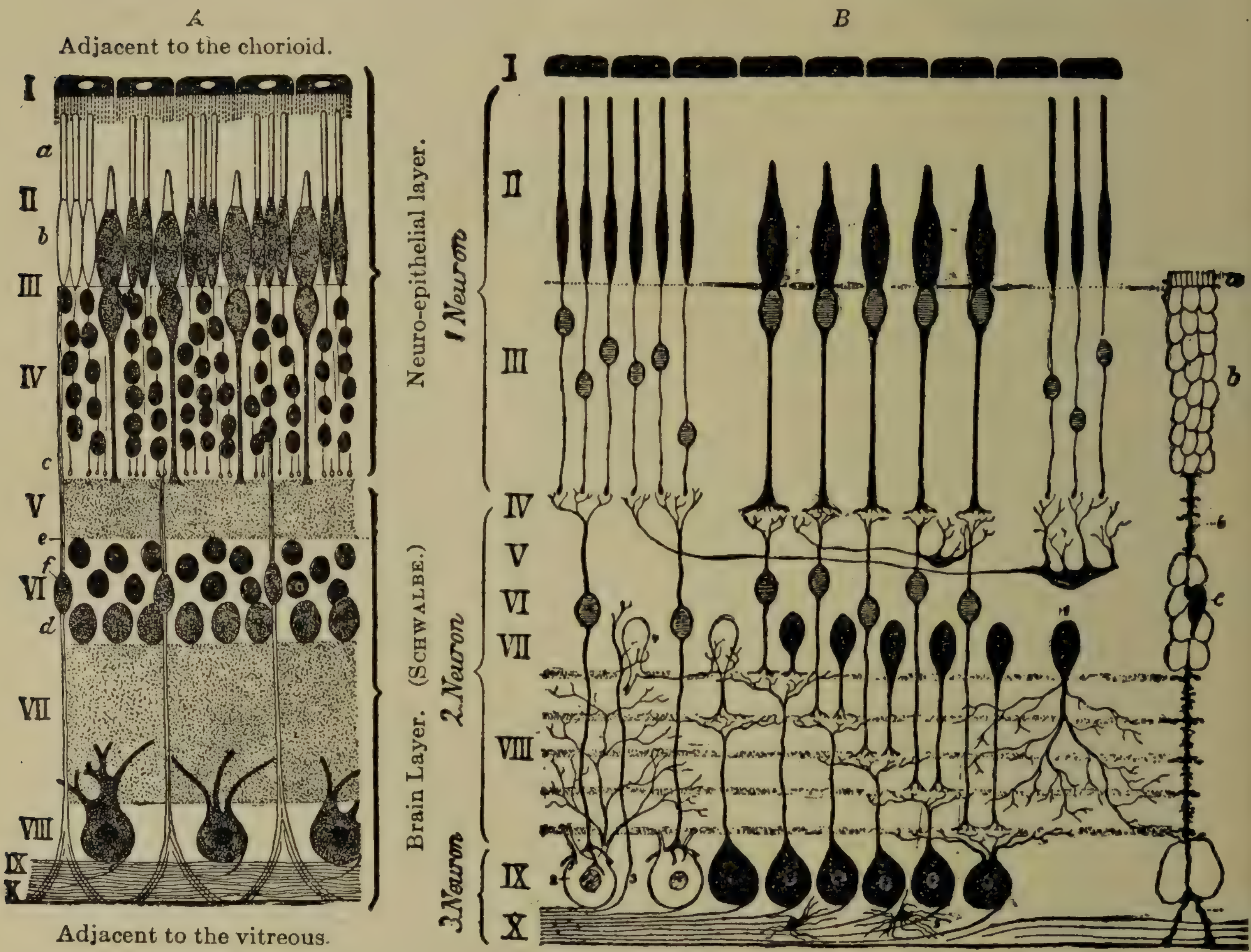

FIG. 32.—SCHEME OF THE STRUCTURE OF THE HUMAN RETINA. (After Greef.)

A. Transverse section (Hæmatoxylin stain);—Schultze's nomenclature. I. Layer of pigment epithelium (stratum pigmenti, tapetum nigrum). II. Layer of rods and cones; *a*, external; *b*, internal elements. III. External limiting membrane. IV. Layer of outer granules (external granular or external nuclear layer); *c*, fibre layers. V. External plexiform (external molecular layer; intergranular layer) VI. Internal nuclear (internal granular) layer; *d*, spongioblasts; *e*, sustentacular fibre of Müller with its nucleus (*f*). VII. Internal plexiform (internal molecular) layer. VIII. Layer of ganglion cells. IX. Layer of nerve fibres (stratum opticum). X. Internal limiting membrane. The above layers are divided into three sets of neurones, the first comprising layers I to IV; the second, layers V to VII; the third, layers VIII to X.

B. Demonstration *by* Golgi's method (Cajal's nomenclature).—I. Layer of pigment epithelium. II. Layer of rods and cones. III. Cone granules and rod granules. IV. External plexiform layer. V. Layer of horizontal cells. VI. Layer of bipolar cells. VII. Layer of amacrine[1] cells (spongioblasts) which ramify either in a single stratum of the inner plexiform layer (stratified amacrine cells) or in several strata (diffuse amacrine cells—1). 4, association amacrines, connecting widely separated amacrine cells. VIII. Inner plexiform layer (in five strata). IX. Layer of ganglion cells; the dendrites of these cells either are confined to single strata or in the case of the diffuse ganglion cells (2) ramify in several strata. X. Layer of nerve fibres. 3, centrifugal fibre. The above layers constitute the retina proper, layers II and III forming the neuro-epithelium, IV to X the cerebral layer. In addition the retina contains a supporting framework of neuroglia consisting of the neuroglia cells or spider cells (5) lying in the ganglion cell and nerve fibre layers and sending ramifications into the plexiform layer and among the nerve fibres; and of sustentacular fibres (6) whose broad upper extremities (at *a*) blend to form the external limiting membrane and whose lower ends similarly form the internal limiting membrane. *c*, nucleus of fibre.

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[1]Form ἀ privative, μακρός long, and ἴς, tendon, because of their short processes.

embryological grounds, be counted in with the retina (in the wider sense of the term). The connection between the retina and pigment epithelium consists in the fact that the cells of the latter send minute cilia-like proc-

FIG. 33.—SECTION OF HUMAN RETINA AT THE ORA SERRATA. (After Piersol in Norris and Oliver.) Shows the abrupt termination of the usual retinal layers and the continuation of the retinal sheet as the pars ciliaris.—*a*, pigment layer; *b*, rods and cones; *c*, outer nuclear layer; *d*, outer plexiform; *e*, inner nuclear; *f*, inner plexiform; *g*, ganglion cells; *h*, point of transition into inner stratum (*k*) of pars ciliaris; *i*, section of cyst. Magnified 165 diameters.

esses in between the rods and cones; in these processes lie the minute crystals of the brown retinal pigment.

44. Continuation on Ciliary Body and Iris.—The cessation of the retina at the ora serrata is apparent only; the microscope shows that under

FIG. 34.—BLOOD-VESSELS OF THE YELLOW SPOT INJECTED. After Böhm and Von Davidoff. (Norris and Oliver.)

a simpler form it extends still farther, even up to the edge of the pupil. It therefore lines the inner surface of the ciliary body, forming the *pars ciliaris retinæ* (§ 30) and the posterior surface of the iris, forming the *pars iridica retinæ* (§ 27). (Cf. also Fig. 33.)

45. Retinal Vessels.—The retina has its own system of blood-vessels, which is confined to its inner layers and which is almost entirely separate from the adjoining system of ciliary vessels. It is formed by an expansion of the arteria and vena centralis nervi optici, which break up into branches in the optic disc. These branches subdivide in the retina as far as the ora serrata without anastomosing (see Fig. 30, a, a_1, and b, b^1; Fig. 190 represents the branching of vessels in the retina as seen with the ophthalmoscope). At the disc alone, minute communications exist between the retinal and the ciliary vessels.

Not infrequently it happens that a rather large branch of Zinn's scleral circle of vessels (§ 48) passes through the optic nerve into the retina. Such a *cilio-retinal artery* is not an anastomotic vessel but a ciliary artery which supplies a part of the retina—particularly a district of greater or less size on the temporal side of the disc. With the ophthalmoscope a vessel like this can be recognized by the way in which it emerges at the edge of the disc and curls over it (Fig. 35). In the veins a similar anomaly is found, though not nearly so often, in the form of the *optico-ciliary veins* (Elschnig), which carry blood from the chorioid to the central vein.

Fig. 35.—Cilio-retinal Artery.
From the outer and lower margin of the papilla rises a cilio-retinal artery (a), making a hook-like bend. In this case it is larger than usual because it replaces the lacking inferior temporal branch of the central artery.

II. Optic Nerve

46. Divisions.—The optic nerve (nervus opticus) collects its fibres from the retina, and passes from the eye through orbit and optic foramen into the cavity of the skull. Hence, three divisions are distinguished in the optic nerve: (a) The intra-ocular termination, which is found within the sclera; (b) the orbital portion from the eyeball to the optic foramen; and (c) the intra-cranial portion from the optic foramen to the chiasm.

Explanation of Fig. 36.—Longitudinal Section Through the Head of the Optic Nerve. Magnified 60×1.

In its passage through the sclerotico-chorioidal canal the optic nerve shows an irregular conical contraction. The fibres of the nerve are collected into bundles, n, separated by septa, s. Under the form of rows of nuclei, which belong to the neuroglia cells, the continuation of the septa can be followed to the head of the optic nerve. The axis of the nerve is occupied by the central vein, v, and the central artery, a, which is situated to the nasal side of the vein. The optic nerve is traversed transversely by the lamina cribrosa, which separates the trunk from the head of the nerve. The fibres of the lamina cribrosa arise from the wall of the scleral canal, traverse the nerve in a slightly concave arch (the concavity being directed to the front), and are inserted into the connective tissue that accompanies the central vessels. About at the level of the inner layers of the chorioid, the nerve fibres diverge like a sheaf, so as to form a funnel-shaped depression—the vascular funnel, G. More fibres pass to the nasal than to the temporal side of the papilla, for which reason the former side is the higher. The fibres of the optic nerve pass over into the fibre layer (*1*), of the retina. Succeeding this are the other layers of the retina, namely the layer of ganglion cells (*2*), the inner plexiform layer (*3*), the layer of inner granules or bipolar cells (*4*), the outer plexiform layer (*5*), the layer of the outer granules or of the bodies of the visual cells (*6*), the limitans externa (*7*), and the layer of rods and cones (*8*). The layers of the retina stop short at the head of the optic nerve, the outermost layer, *8*, extending the furthest in. The innermost fibres of the sclera which form the wall of the scleral canal, accompany the optic nerve backward and form its pial sheath, P, which is in intimate relation with it. At a point further back from the nerve-head the outer layers of the sclera are reflected backwards and form the dural sheath, D, which envelops the nerve loosely. Between these two sheaths lies a third, the arachnoid sheath, A which divides the intervaginal space of the optic nerve into the subdural space sd, and the subarachnoid space, sa. Anteriorly both end by a cul-de-sac in the substance of the sclera. b is the cross section of one of the numerous subarachnoid trabeculæ which connect the arachnoid to the pial sheath. In the wall of the scleral canal is seen the cross section of some blood-vessels, belonging to Zinn's scleral circle. Between the sclera, S, and the retina, R, lies the chorioid, Ch. The innermost layer of the latter, the lamina vitrea, *10*, is the one that extends the furthest in toward the nerve-head, and the fibres of the nerve are

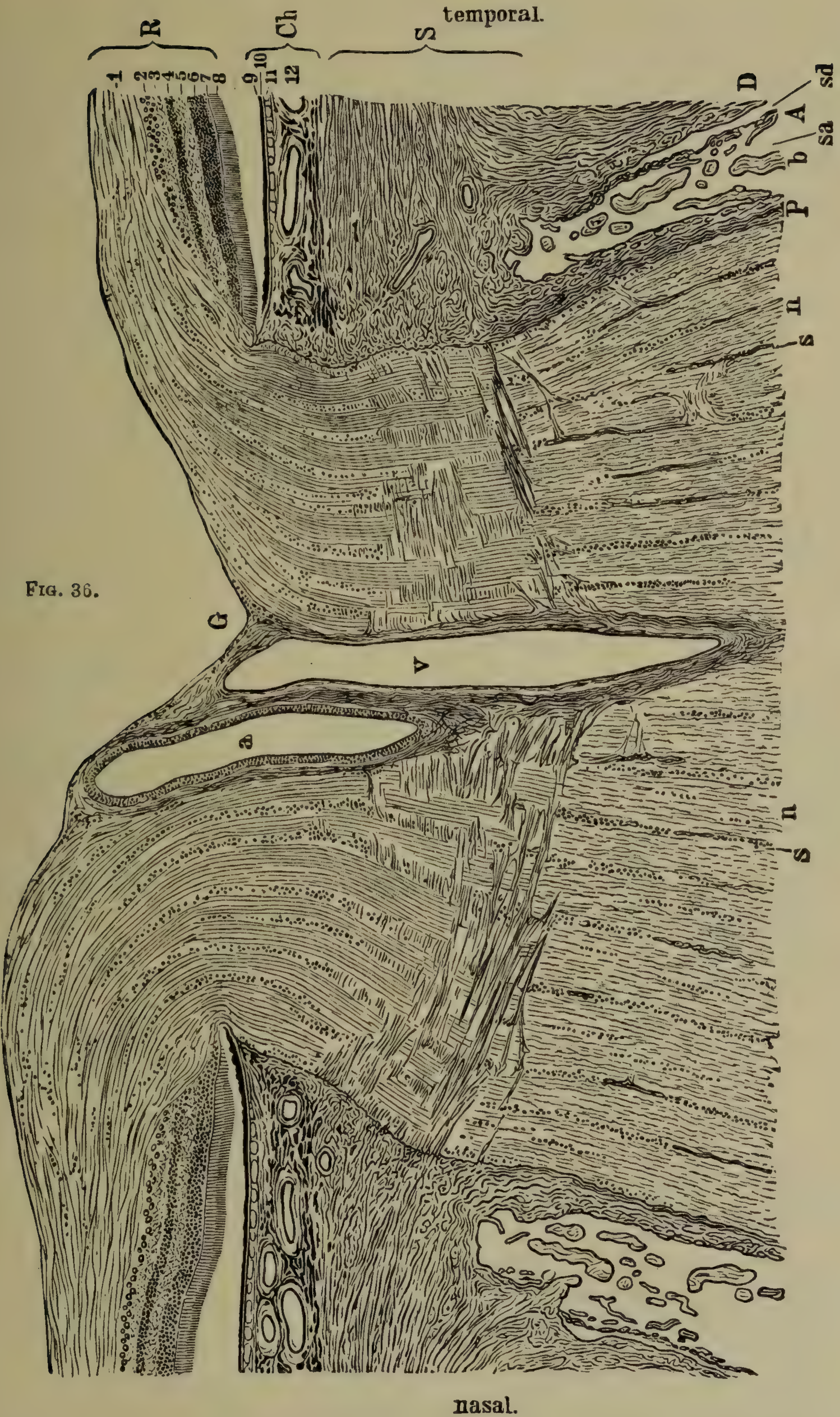


Fig. 36.

constricted by the edge of the lamina. Upon the lamina vitrea lies the pigment epithelium, *9*, which belongs to the retina and which on the nasal side extends as far as the lamina vitrea, but on the temporal side stops somewhat short of it. On both sides the pigment epithelium gets to be thicker and more pigmented toward its edge—a state of things which answers to the chorioidal ring that can be seen with the ophthalmoscope. The succeeding layers of the chorioid, the chorio-capillaris, *11*, and the layer of medium and large sized vessels, *12*, do not extend quite up to the optic nerve on the temporal side, because a layer of connective tissue representing a continuation of the sclera juts in between the two.

47. Intra-ocular Portion.—To get from the retina to the exterior of the eye, the optic nerve must pierce the chorioid and sclera. The spot where this takes place lies a little to the inner side of the posterior pole of the eye (Fig. 2). The opening in the sclera through which the optic nerve leaves the eye is called the *foramen scleræ,* and really consists of a short canal (sclerotico-chorioidal canal); the segment of the optic nerve lodged in this is its intra-scleral portion. Accurately speaking, a complete aperture for the optic nerve exists neither in the sclera nor in the chorioid, but the two membranes conduct themselves as follows: The external lamellæ of the sclera, which occupy about two-thirds of its thickness, are not perforated by the optic nerve at all, but are reflected backward upon it to form its exterior sheath (Fig. 36, *D*). The innermost lamellæ of the sclera, on the contrary, stretch over the foramen scleræ, forming a diaphragm which is perforated by numerous openings designed for the passage of the separate funiculi of the optic nerve and is hence called the *lamina cribrosa.* (Seen in longitudinal section in Fig. 36, in cross section in Fig. 39). In a similar fashion, a few fibrous bands from the chorioid are also continued over the foramen scleræ.

The lamina cribrosa plays an important part in *pathological* processes. In the first place, it is the weakest spot of all the tunics of the eye, being here constituted only by the innermost layers of the sclera (together with a few fibres of the chorioid), which, moreover, are perforated by the foramina for the bundles of fibres of the optic nerve. Hence, in case of increase of tension, this spot is the first to give way. In the normal eye the lamina cribrosa runs straight, or with but a slight backward curvature, across the optic nerve. With increase of tension, it recedes more and more, and thus forms the glaucomatous excavation. A second reason for the production of pathological changes lies in the fact that within the foramen scleræ, and particularly within the limits of the lamina cribrosa, the optic nerve is tightly inclosed between firm, fibrous walls, a thing that occurs at no other spot in its course. Here, therefore, when swelling of the optic nerve takes place, constriction and strangulation of it may readily occur. The foramen scleræ, accordingly, in this case play a part like that which the fibrous ring of the hernial orifice does for the viscera lying in front if it.

That portion of the optic nerve situated in front of the lamina cribrosa, in the interior of the eye itself, is the *head of the optic nerve* (papilla nervi optici, optic disc). It is the part of the optic nerve which in life can be seen by means of the ophthalmoscope, (see § 403).

From the papilla the optic-nerve fibres spread out in a sheaf to form the most anterior (most internal) layer of the retina (Fig. 36, *1*; Fig. 32 B, X).

The fibres are grouped in four sets of bundles, nasal, supero-temporal, infero-temporal, and papillo-macular (Fig. 40). The *nasal* fibres pass in slightly diverging fan-like curves to supply the nasal half of the retina. The *papillo-macular bundle* comprises one third of all the nerve fibres. These occupy a section on the temporal side of the disc (cf. *pm.* Fig. 41) and proceed in elliptical curves to the macular region (*E*). The *supero-temporal* and *infero-temporal bundles* (Elliot) comprise two sets, peripheral and intermediate. The peripheral fibres like the nasal pursue a curving fan-like course to the periphery. The intermediate fibres curve round so that those of the supero-temporal meet those of the infero-temporal bundle in the horizontal meridian in a line (rhaphe) extending 10° to 60° from the fovea. This line neither set of fibres passes.

The fibres of the supero-temporal and infero-temporal bundles, which supply such a large area of the retina must as Fig. 40 shows, be very closely crowded together as

they pass over the edge of the disc. (For the pathological significance of this see § 840). On the other hand, the papillo-macular bundle is very large in comparison with the small size of the area of retina which it supplies. This relation, which is in harmony with the predominant physiological importance of the macular region, is explainable by supposing that each one of the terminal elements of the macula is connected with the brain by a nerve fibre of its own, so that excitations of this element are conveyed to the brain, iso-

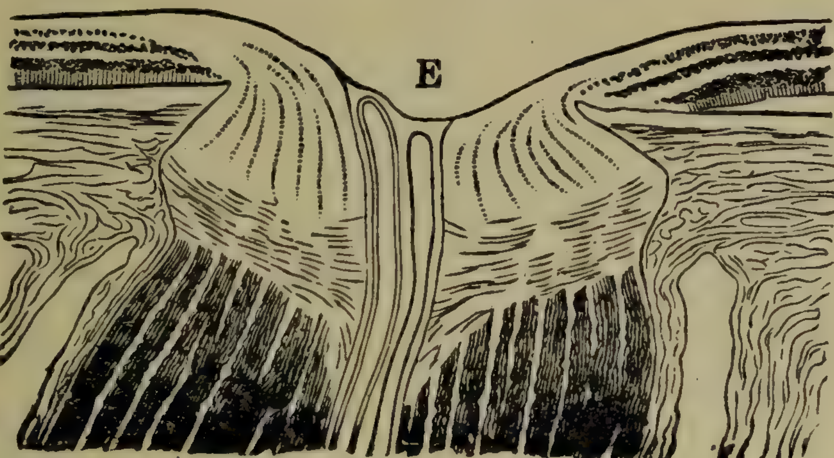


Fig. 37.—Longitudinal Section through the Optic Nerve Head. Magnified 20×3.
The nerve fibres of the trunk of the nerve, whenever they are medullated, are stained black by Weigert's hæmatoxylin stain The medullary coating extends as far as the lamina. Proceeding from behind forward, the sclerotico-chorioidal canal shows first a dilatation, then a contraction. *E*, physiological excavation. (Cf. Fig. 192.)

Fig. 38.

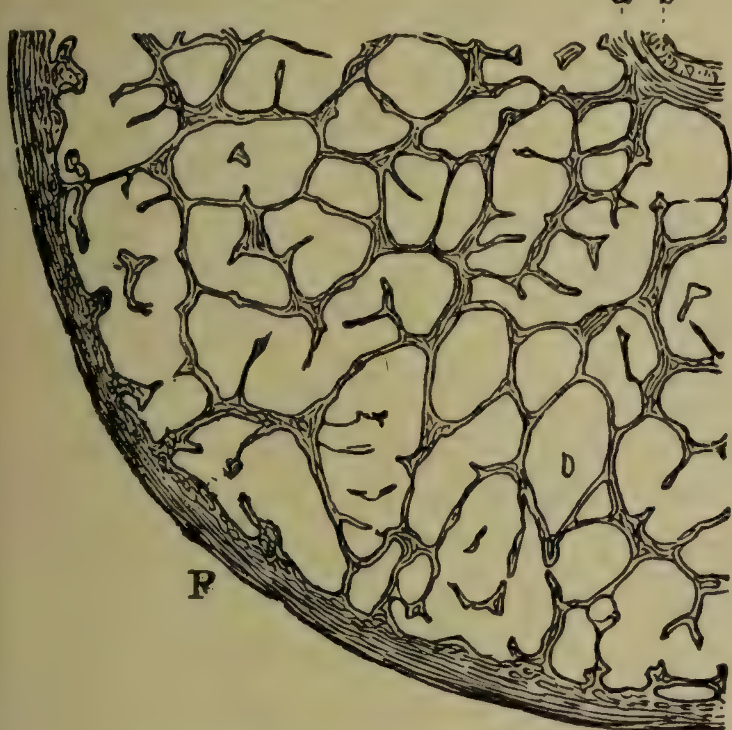


Fig. 39.

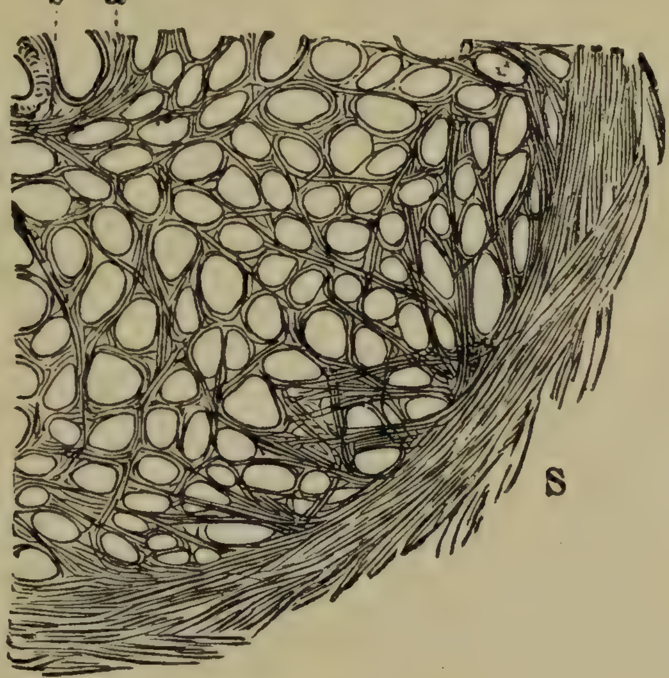


Fig. 38.—Frontal Section through the Trunk of the Optic Nerve behind the Lamina Cribrosa. Magnified 50×1. The septa start from the pial sheath, *P*. They traverse the optic nerve branching repeatedly and forming numerous interconnections, and join with the connective tissue, *a*, which surrounds the central vessel, *b*.

Fig. 39.—Frontal Section through the same Nerve at the Level of the Lamina Cribrosa. Magnified 50×1. The septa, *S*, starting from the wall of the scleral canal are compact and broad and run, forming a dense plexus, through the entire optic nerve as far as the connective-tissue investment, *a*, of the central vessels. In the vicinity of the latter the gaps between the septa are larger than at the periphery of the nerve, because in the former spot the section falls somewhat in front of the densest portion of the lamina. For, since the lamina as a whole curves slightly backwards, it can never completely coincide with the plane of the section.

lated from those of other elements, while in the peripheral portions of the retina probably a number of terminal elements are continuous with one fibre.

It is probable that the fibres which are derived from the peripheral portions of the retina pass to the centre, and those derived from the central portions of the retina pass to the margin of the optic disc. The contrary view expressed by Collins and Mayou seems unlikely (Elliot).

The fibres in the retina and also in the nerve head when traversing the lamina cribrosa are non-medullated and therefore transparent. Behind the lamina they acquire a medullary sheath, so that they become not only white and opaque but also thicker. The orbital portion of the optic nerve

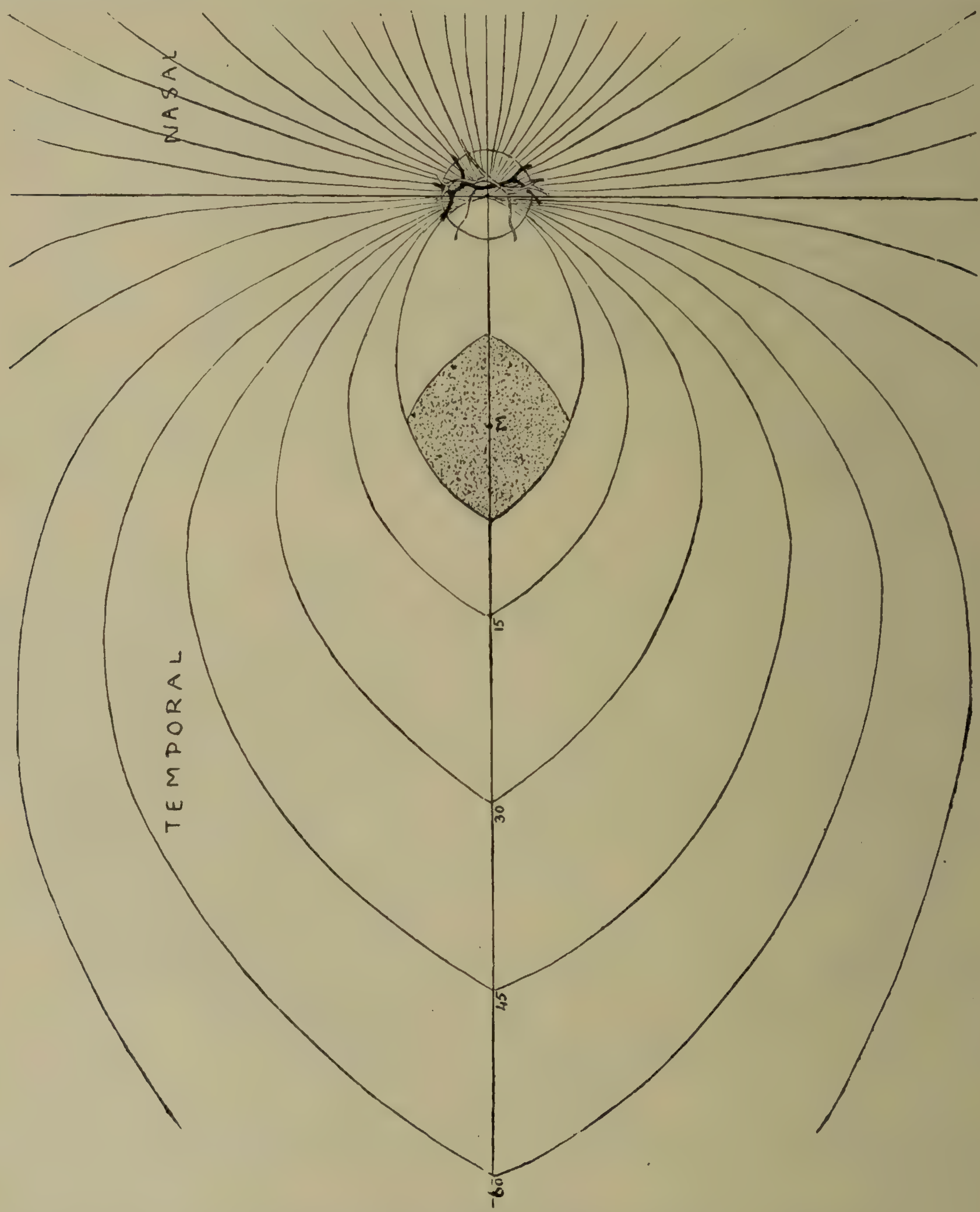


Fig. 40.—Distribution of Optic Nerve Fibres in Retina. (After Elliot).

consequently is decidedly broader than its intra-scleral portion (cf. Figs. 37 and 192).

48. Orbital Portion.—The optic nerve on its way from the eye to the foramen opticum makes a bend like an italic *f* (*O*, Fig. 2). Owing to this, the eyeball can move freely within wide limits. For the movements of the eye take place about a centre of rotation which lies nearly in the centre of the eyeball. Hence, when the cornea is turned toward one side, the

posterior pole of the eye goes about as far to the opposite side. If now the optic nerve were stretched in a straight line between the eyeball and the optic foramen, it would keep the posterior segment of the eyeball fast in its place, and hinder the movements of the whole eye. This, in fact, occurs when the optic nerve is put on the stretch by protrusion of the eyeball from the orbit (exophthalmus).

The orbital portion of the optic nerve consists of the trunk of the nerve and the sheaths enveloping it.

(*a*) The *trunk* of the optic nerve is composed of nerve fibres and connective tissue. The nerve fibres vary greatly in calibre and are extremely numerous, some estimating their amount at half a million or more. Most of them are centripetal, but there are also some centrifugal fibres in the optic nerve. Lying between the fibres as a supporting and insulating substance is the neuroglia tissue. The nerve fibres are combined into bundles (*n*, Fig. 36) which run parallel to one another, and anastomose by a mutual interchange of fibres. Between the bundles lies the connective tissue which furnishes the supporting framework for the entire optic nerve. It forms thick or thin septa which are everywhere connected and traverse the entire optic nerve (Fig. 36, *s*; Fig. 38). Between the outer surface of a nerve bundle and the inner surface of the septa is found a space which acts as a lymph cavity.

In the trunk of the nerve, the fibres of the *papillo-macular bundle* (§ 47) have peculiar arrangement. Immediately behind the eye they are aggregated to form a sector whose apex is directed toward the centre of the nerve, while its base corresponds to the outer margin of the latter (the paler-looking bundle, *pm*, in Fig. 41). Farther back the arrangement changes, so that these fibres get to lie in the axis of the nerve.

(*b*) The *sheaths* of the optic nerve are three—an interior, a middle, and an exterior one. As these originate from the three enveloping membranes of the brain, they are designated by the names of the pial, arachnoid, and dural sheaths (Axel Key and Retzius). The inner or pial sheath (*P*, Figs. 36 and 41) closely embraces the trunk of the optic nerve. From it bands of connective tissue, which form the septa, pass into the interior of the nerve; and with them pass the blood-vessels. The exterior or dural sheath (*D*, Fig. 36 and *du*, Fig. 41) is much thicker than the interior sheath, and surrounds the nerve loosely. By reason of this, a pretty broad space—the intervaginal space—is left between it and the internal sheath. The middle or arachnoid sheath (*A*, Fig. 36 and *ar*, Fig. 41) is a very delicate pellicle which for the most part is intimately attached to the dural sheath. It is united by manifold trabeculæ of connective tissue to the external and internal sheaths. It divides the intervaginal space into two portions, the subdural (*sd*) and subarachnoid (*sa*) spaces, which communicate with the cerebral spaces of the same names. These appear particularly prominent in Fig. 366, where they are pathologically dilated by an accumulation of fluid. The surfaces of the sheaths that are turned toward these spaces are provided with an endothelial coating, so that these spaces are lined completely with endothelium, and must be regarded as lymph channels (Schwalbe).

Upon the eyeball the three sheaths become united to the sclera. The exterior and middle sheaths pass into the outer two-thirds of the sclera; the inner sheath goes to the innermost lamellæ of the sclera which form the lamina cribrosa, and it is also connected with the chorioid. The intervaginal space ends by a cul-de-sac situated within the sclera. Posteriorly the three sheaths are continuous with the corresponding membranes of the brain.

The *blood-vessels* pass from the pial sheath into the optic nerve. In addition, in the anterior portion of the orbital division there are found the central vessels of the optic nerve. The central artery is a branch of the ophthalmic artery; the central vein empties into the superior ophthalmic vein

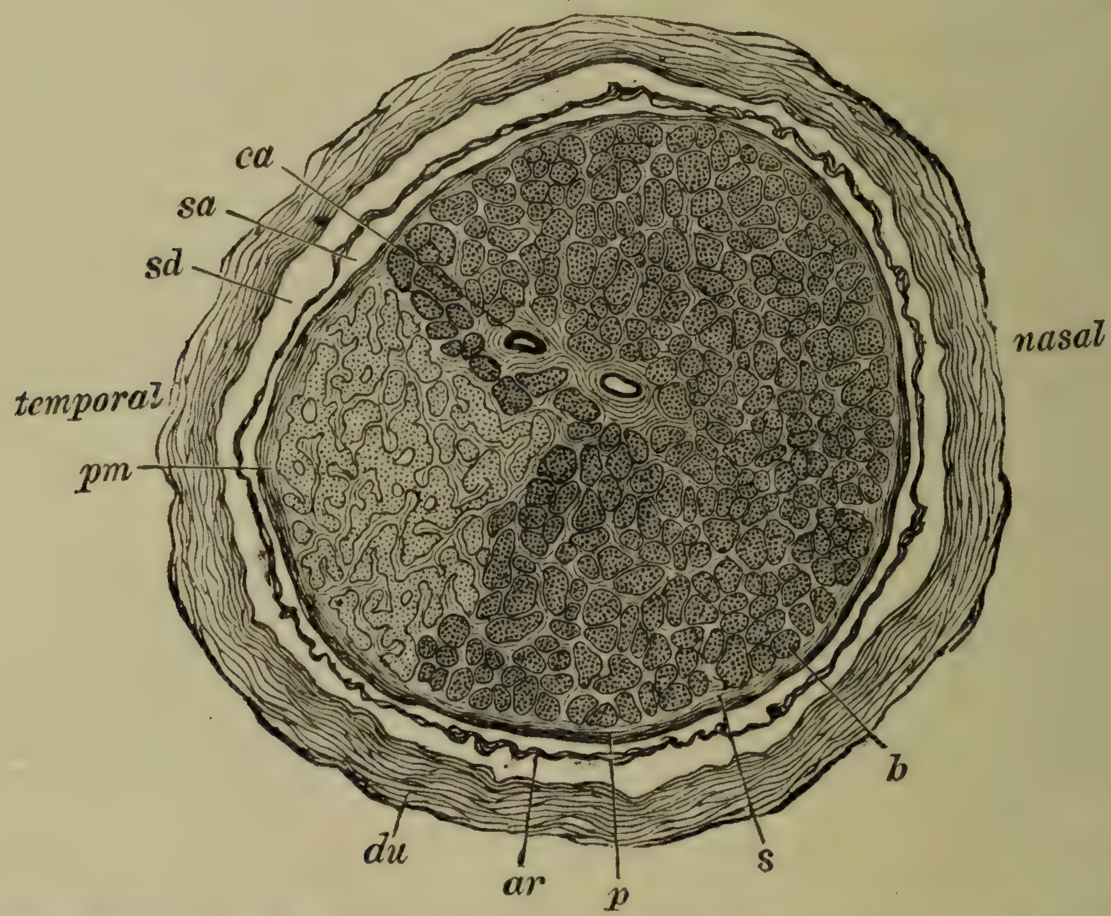


FIG. 41.—CROSS SECTION OF THE OPTIC NERVE, WITH ATROPHY OF THE PAPILLO-MACULAR BUNDLE (SECTION MADE 4 MM. BEHIND THE EYEBALL). Magnified 15×1.

The optic nerve is enveloped in the dural sheath, *du*, the arachnoid sheath, *ar*, and the pial sheath, *p*. Between the first and second is found the subdural space, *sd*; between the second and third, the subarachnoid space, *sa*. On the outer and upper side of the centre of the section is seen the central artery, *ca*; and more centrally is seen the central vein. These are surrounded by the cross sections of the nerve bundles, *b*, which are separated from each other by the septa, *s*, of connective tissue. At the temporal side, a wedge-shaped segment, *pm*, is distinguished from the rest of the cross section of the nerve by its paler color. This represents the atrophic papillo-macular bundle. Within the confines of it the cross sections of the nerve bundles are narrower, and the septa of connective tissue are correspondingly broader.

or directly into the cavernous sinus. Both vessels enter the optic nerve on its lower and inner side at a distance of 7 to 12 mm. behind the eyeball (Fig. 2, *e*) and run in the axis of the nerve as far as the papilla, where they divide into the retinal vessels.

The region of the lamina cribrosa derives its blood-vessels from two sources, the central vessels of the optic nerve and Zinn's or Haller's *circle of vessels* (circulus arteriosus nervi optici). This circle of anastomotic vessels is supplied by the short posterior ciliary arteries, and lies in the sclera in front of the blind end of the intervaginal space (Fig. 36). The capillary network of the lamina cribrosa represents the ultimate connection between the retinal and ciliary systems of vessels. To the distal side of the lamina cribrosa the retinal system is completely isolated. (cf. § 45).

49. Intra-cranial Portion.—The optic nerve leaves the orbit through the optic foramen. The latter really forms a short bony canal (canalis opticus), which contains besides the optic nerve only the ophthalmic artery (lying on the inner side of the optic nerve).

The intra-cranial portion of the optic nerve extends from the optic foramen to the chiasm; is therefore short (scarcely one centimetre). It is flattened and is enveloped only by the pial sheath, since the other two sheaths after passing through the optic foramen become united with the two outer membranes of the brain.

50. Chiasm and Optic Tract.—The two optic nerves join together in the chiasm, where they decussate (see Fig. 110) and then reappear on the posterior side of the chiasm as the optic tracts, T, T_1. The chiasm lies in the optic groove of the body of the sphenoid bone, directly in front of the infundibulum and above the hypophysis. Starting from the chiasm the optic tracts pass backward, diverging as they go, and, winding about the crus cerebri, arrive at the primary subcortical optic centres. The most important of these are the external geniculate body, the anterior corpus quadrigeminum, and the thalamus opticus. Thence fibres pass to the oculo-motor nucleus and mainly to the cortex (see §§ 233, 234, 366).

CHAPTER VI

THE ORBIT AND ITS CONTENTS

I. Orbit

51. The bony orbit forms a quadrilateral pyramid, whose base corresponds to the anterior aperture of the orbit, and whose apex to the optic foramen. The nasal walls of the two orbits are about parallel to each other; but the temporal walls diverge from each other a good deal from behind forward. The nasal wall is the thinnest, as it is formed by the lacrimal bone, which is as thin as paper, and by the delicate lamina papyracea of the ethmoid (Fig. 2, *T* and *L*, Fig. 42, *L* and *l*). At its anterior extremity it bears the fossa lacrimalis for the reception of the lacrimal sac (Fig. 48, *fl*). In the posterior portions of the orbit are found three apertures which connect the orbit with the parts adjacent. These are: 1. The optic foramen (Fig. 42 , *o*) which passes between the two roots of the lesser wing of the sphenoid (*k*) into the middle fossa of the skull. Through it the optic nerve and beneath the latter the ophthalmic artery pass into the orbit. 2. The superior orbital fissure (*s*) which lies at the junction of the upper and outer wall, and is bounded by the lesser and greater wings of the sphenoid (*k* and *g*). It also opens into the middle fossa of the skull, and transmits the nerves for the ocular muscles and the first branch of the trigeminus. 3. The inferior orbital fissure (*i*) which is longer than the superior orbital fissure, and lies at the junction of the outer and the lower wall of the orbit, between the great wing of the sphenoid (*g*) and the superior maxilla (*M*). It connects the orbit with the temporal fossa (fossa spheno-maxillaris). Through it the twigs of the second branch of the trigeminus, the largest of which is the infra-orbital nerve, pass into the orbit.

The inferior orbital fissure is closed by a fascia with which numerous smooth muscular fibres are interwoven (musculus orbitalis of Müller). These are innervated by the sympathetic.

In the growth of the body the orbit expands in proportion as the eyeball enlarges. If the eyeball is backward in its growth, and more especially if it is entirely destroyed in childhood, the dimensions of the orbit also remain smaller. If, therefore, in such cases it is desired later on in life to wear an artificial eye, we must content ourselves with one which is too small when compared with the other eye.

The walls of the orbit at their anterior margin become thickened into a strong bony ring, the *margin* of the orbit. This constitutes the most important defense of the eye against external force, especially above and below where it juts farthest out. On the inner side there is no sharply defined margin to the orbit, but here the eye is protected by the bridge of the nose. On the outer side the orbital margin recedes the farthest (Fig. 47, *A*), so that here the eye is most exposed to injuries. At the upper margin of the orbit is found the supra-orbital notch designed for the artery

and nerve of the same name (Fig. 48, *i*; Fig. 42, *is*). At the lower margin of the orbit there is a canal (the infra-orbital canal; Fig. 42, *ci*) for the infra-orbital artery and nerve, and this opens upon the cheek—by means of the infra-orbital foramen (*fi*, Fig. 42)—about 4 mm. beneath the orbital margin. This point and the supra-orbital notch are of practical importance, as constituting the points of exit of the aforesaid nerves. Sensitiveness to pressure at these spots is a frequent symptom in neuralgiæ of the trigeminus and also in essential blepharospasm.

The orbit is surrounded by several other cavities in disease of which it can itself be implicated. These cavities are the nasal fossæ and the

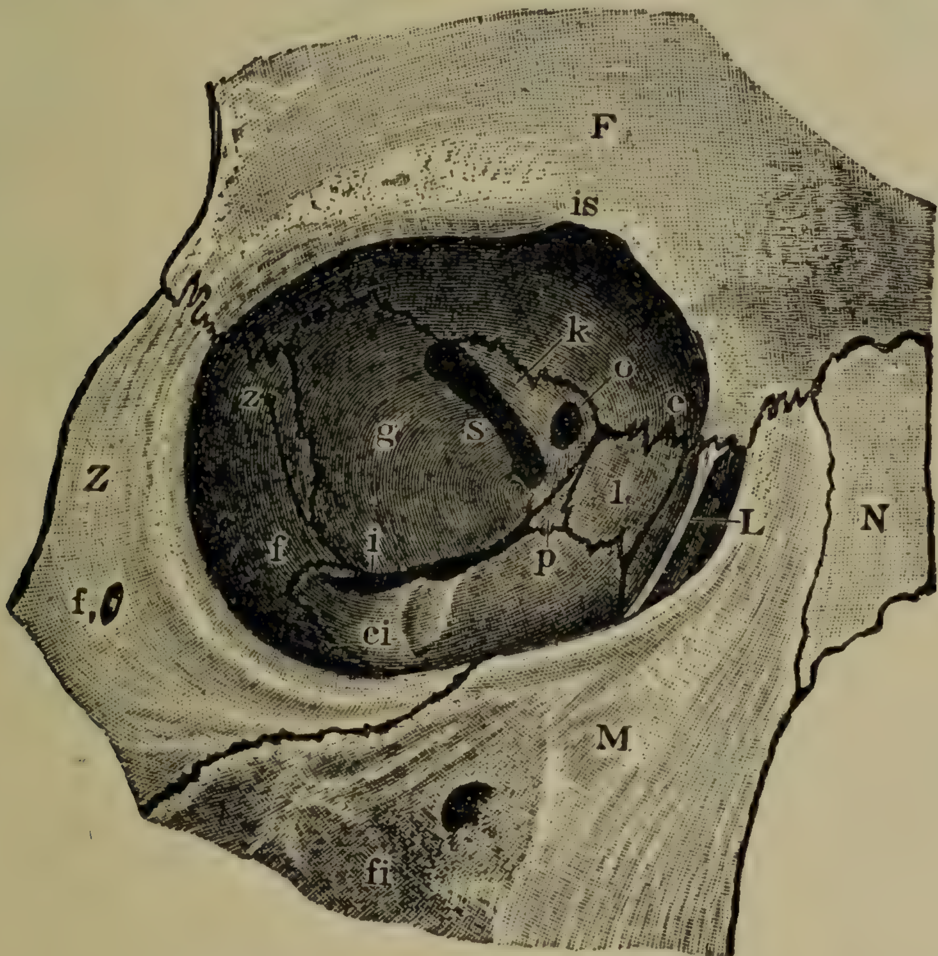


Fig. 42.—Wall of the Right Bony Orbit. (After Zuckerkandl.)

The wall of the orbit is formed on the outside by the malar bone, and the greater wing of the sphenoid, *g;* above by the frontal bone, *F*, and the small wing of the sphenoid, *k*, which incloses the foramen opticum, *o*, and, together with the greater wing of the sphenoid, bounds the superior orbital fissure, *s;* on the inner side by the frontal process of the superior maxilla, *M*, the lacrimal bone, *L*, and the lamina papyracea of the ethmoid bone, *l;* below by the superior maxilla, *M*, and the orbital process of the palatal bone, *p*. Between the greater wing of the sphenoid and the superior macilla lies the inferior orbital fissure, *i*, from which runs the sulcus infra-orbitalis, *ci*, which has its exit as the infra-orbital foramen, *fi*, on the anterior surface of the superior maxilla; *is*, supra-orbital notch; *f*, inner and, f_1, outer orifice of the zygomatico-facial canal; *z*, inner orifice of the zygomatico-temporal canal; *e*, anterior ethmoid foramen; *N*, nasal bone.

cavities accessory to them—namely, the frontal sinus, the antrum of Highmore, the sphenoidal sinus, and the ethmoid cells.

52. Contents.—The contents of the orbit consist of the eyeball with the optic nerve and the muscles, the lacrimal gland, the vessels, and the nerves. The interstices between these structures are filled with orbital fat, and the whole is maintained in a state of firm connection by a system of *fasciæ*. The latter display a greater strength and a more intimate union with one another in three places, viz.: 1. Along the walls of the orbit. They cover the latter under the form of a periosteum (here called periorbita), and likewise make a sort of anterior wall for the orbit. This

anterior wall is formed by the fascia (fascia tarso-orbitalis) that starts from the margin of the orbit and is attached to both tarsi and also to the ligamentum canthi internum and externum. These structures combined represent the septum orbitale, which, when the lids are closed, shuts off the orbit anteriorly and keeps its contents in (Fig. 3). 2. The ocular muscles are surrounded by fasciæ which send out processes connecting the muscles with each other, with the lids, and with the margins of the orbit (see § 54). 3. Surrounding the eyeball the fasciæ are condensed into a fibrous capsule, the *fascia bulbi* (also called *Tenon's* or *Bonnet's capsule*). This extends forward as far as the conjunctiva of the eyeball and backward nearly to the optic nerve. It is thus open in front and behind, and may be said to represent a broad ring placed about the eyeball. It forms the articular socket for the eyeball, which can move in it freely in all directions. The contiguous surfaces of Tenon's capsule and of the eyeball are smooth, and are provided with an endothelial covering (Schwalbe). The intervening space, (*t*, Fig. 57), is called Tenon's space. According to some it is a lymph space (see § 78). At the points where the tendons of the ocular muscles pierce Tenon's capsule, the latter is reflected upon the muscles and becomes continuous with the fasciæ covering them (lateral invaginations of the muscles, *e* and e_1, Fig. 57).

The eyeball is suspended in the orbit and held in place fore and aft by the oblique muscles in front, the recti muscles behind, and strong fascial attachments that bind the eye itself and the muscles to each other and to the orbital walls and the lids. These fascial attachments form a *framework*, feeble behind, very stout in front, which, assisted by the tension of the muscles, holds the eye quite firmly in its place.

The *blood-vessels* of the orbit arise from the ophthalmic artery which springs from the internal carotid and enters the orbit through the optic foramen. The venous blood leaves the orbit through the superior and inferior ophthalmic veins, both of which make their way through the superior orbital fissure to the cavernous sinus, into which they empty. The ophthalmic veins form numerous anastomoses with the veins of the forehead.

Lymph vessels and lymphatic glands are wanting in the orbit.

The *nerves* of the orbit are motor—these being the nerves destined for the ocular muscles—,sensory—these belonging to the first and second branch of the trigeminus—,and sympathetic. To the outer side of the trunk of the optic nerve lies the ciliary ganglion. This contains motor fibres derived from the oculo-motor nerve (short root), and sensory fibres from the trigeminus (long root). The short ciliary nerves pass from the ciliary ganglion to the eye, through the posterior division of which they enter the interior of the organ. The long ciliary nerves, which likewise enter the eyeball, do not arise from the ciliary ganglion, but come directly from the trigeminus (from the branch of it called the naso-ciliary nerve), and convey sympathetic fibres which originate ultimately from the cilio-spinal centre in the cord (Fig. 168).

53. Dimensions of Orbit; Position of Eye in Orbit.—A knowledge of the dimensions of the orbit and of the situation of the eye with relation to it

is important from a surgical point of view. Particularly important is it to know whether the eyes are normally prominent or not. In this regard the researches of Jackson have given the best information. He makes his measurements from a base line connecting the most receding point of the external angle of the right orbit with a like point of the left. In males the apex of the cornea lies on an average 17 mm., and in females 16 mm. in front of this line.

Individual variations are considerable, the degree of prominence being as low as 10 mm. in some cases and as high as 24 mm. in others. (In the latter the whole eye is really outside the orbit). Usually, however, the degree of prominence is within 2 mm. of the average stated. In 21 per cent. of the cases the eyes are unequally prominent, although the difference rarely exceeds 1 mm. When a more marked difference than this occurs it may be combined with an asymmetrical formation of the face, which is frequently associated with a difference in the refraction of the eyes. In this case the difference in position may be only apparent, since the myopic eye is longer and hence is more prominent, so that an exophthalmus is simulated. In children of 5 the average prominence is 10 mm. It increases to 15 mm. at the age of 20, and thereafter much more slowly, especially in males in whom it does not reach its maximum till the age of 25.

The average length of the base line is 101 mm. in males and 97 mm. in females, the measurements ranging between 88 and 110 mm., but being usually close to the average. The apex of the orbit is 28 to 30 mm. behind the base line. The distance between the centres of the corneæ (interpupillary distance) averages 63 mm. in males and 61 or 62 mm. in females, the extreme ranges being from 54 to 70 mm. It increases during the period of growth, being 52 at the age of 5, 58 mm. at the age of 10, 61 at the age of 15, and reaching its maximum between the ages of 20 and 25. (This should be carefully distinguished from the true intercentral distance of the eyes. See § 387).

It is frequently stated that the prominence of the eye increases with the amount of fat in the orbit and diminishes in conditions of emaciation. As Jackson shows, the variation here is only apparent, the eye being held in place not by the fat in the orbit but by the muscles and framework of connective tissue supporting it.

The same author states that when the eye is wide open it advances somewhat. The protrusion so produced rarely amounts to a millimeter, and the appearance of considerable prominence afforded by a marked retraction of the lids and of recession afforded by their semi-closure is for the most part illusory.

II. Ocular Muscles

54. The ocular muscles are distinguished into extrinsic and intrinsic. The latter, also called the interior muscles of the eyes, are the sphincter pupillæ and the ciliary muscle. For their descriptions see § 27. The extrinsic muscles are six in number, four straight and two oblique.

The four straight muscles are the rectus medialis sive internus, lateralis sive externus, superior, and inferior. All four take their origin from the apex of the orbit along the bony circumference of the foramen opticum (*F*, Fig. 47), and from this point run forward, diverging as they go. They thus bound a funnel-shaped space, the muscular funnel (*t t*) (see also Figs. 43–46), the apex of which lies at the foramen opticum, while the eyeball forms its base and the optic nerve (*o*) runs along its axis. The external and internal recti muscles (*i* and *e*) are inserted into the sclera to the outer and inner

side of the cornea; the superior rectus (*su*) has its insertion (s_1) above, the inferior rectus below the cornea. The attachment is effected by means of short tendons, which spread out in the form of a fan and become fused with the sclera, which in this way is thickened in its most anterior portion.

The two oblique ocular muscles are the obliquus superior and inferior. Their course is more complicated than that of the straight muscles. The *superior oblique* (*os*, Fig. 47) also arises from the margin of the optic foramen and runs forward upon the upper and inner wall of the orbit as far as the

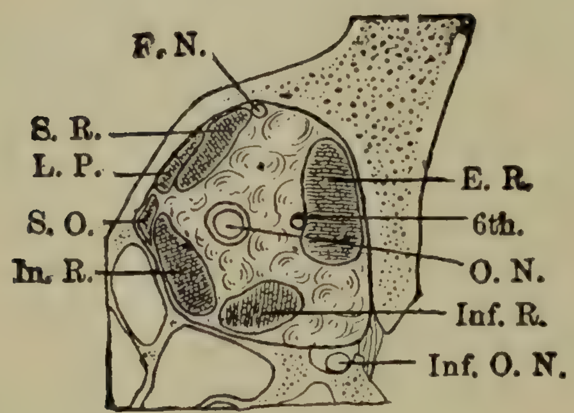


Fig. 43.

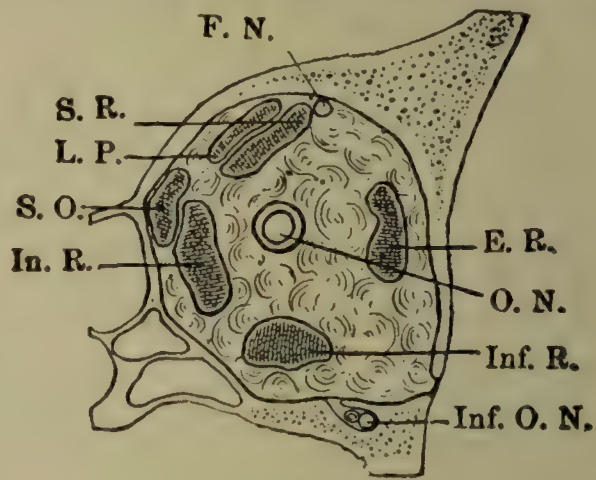


Fig 44

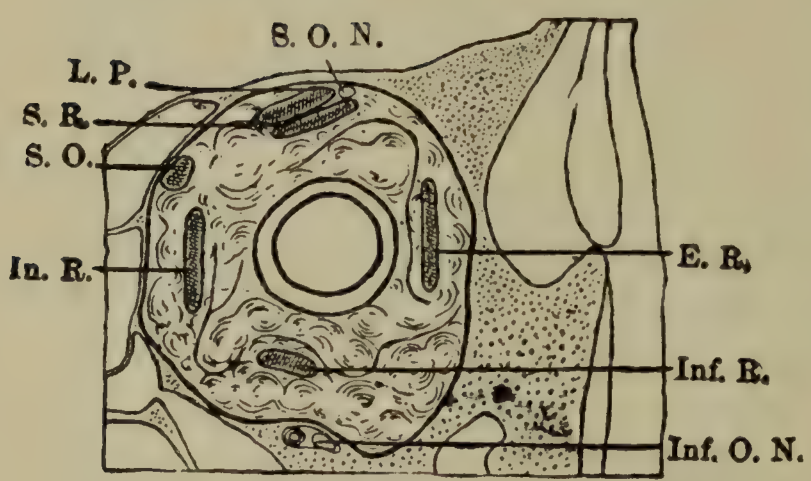


Fig. 45.

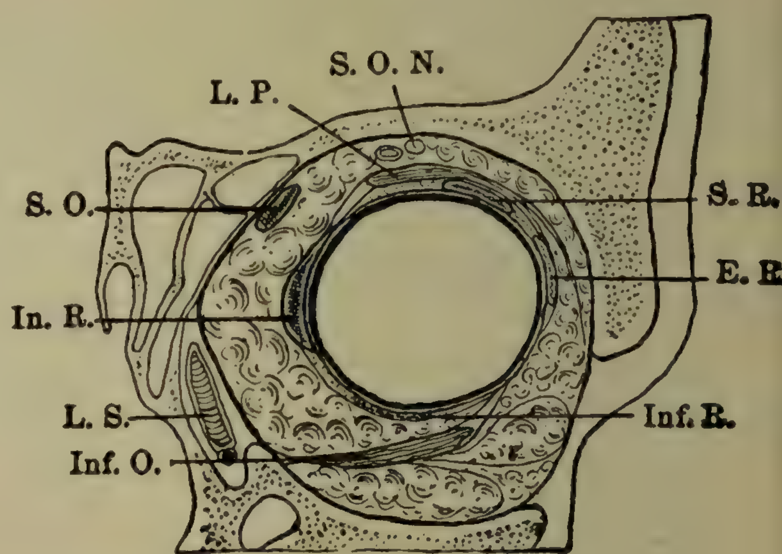


Fig. 46.

Figs. 43-46.—Frozen Frontal Sections of Left Orbit seen from before. (After Dwight from Norris and Oliver.)

Fig. 43.—Section about 12mm. behind globe. *S. R.*, superior rectus, *L. P.*, levator; *S. O.*, superior oblique; *In. R.*, internal rectus; *Inf. R.*, inferior rectus; *E. R.*, external rectus; *O. N.*, optic nerve; *F. N.*, frontal nerve; *Inf. O. N.*, infra-orbital nerve; *6th*, sixth nerve.
Fig. 44.—Section about 5 mm. behind globe. Letters as in preceding figure.
Fig. 45.—Section about 3 mm. in front of back of globe. Letters as before.—*S. O. N.*, supra-orbitral nerve.
Fig. 46.—Section near equator of globe. Letters as before.—*Inf. O.*, inferior oblique. *L. S.*, lacrimal sac.

trochlea, before reaching which it passes into its tendon. The trochlea itself (*T*, Figs. 47 and 48) lies a little behind the upper and inner margin of the orbit. It consists of a firm fibrous loop through which the tendon of the muscle is carried in such a way as to be able to glide back and forth in it. After traversing the trochlea, the tendon bends backward at an acute angle, and passes beneath the superior rectus to the eyeball. Here it spreads out in the form of a fan, and is inserted in the upper half of the eyeball about in the vertical meridian and behind the equator (os_1, Fig. 47).

The *inferior oblique* arises from the lower margin of the orbit near its inner extremity (*oi*, Fig. 48). From here it runs upward and outward

beneath the inferior rectus and arrives at the outer side of the eyeball, into which it is inserted about in the horizontal meridian and also behind the equator (*oi*, Fig. 47).

The tendons of the ocular muscles before reaching the sclera must pass through the fascia of Tenon (fascia bulbi) which surrounds the eyeball. In the spot where a tendon of the muscle perforates the fascia, the latter does not present a corresponding aperture, but is reflected backward upon the tendon (e and e_1, Fig. 57). It ensheathes the tendon and further

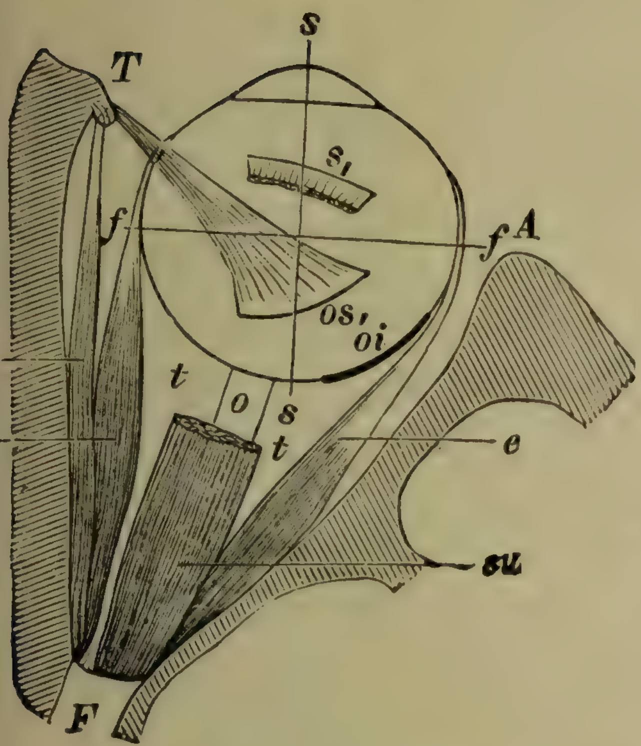


Fig. 47.

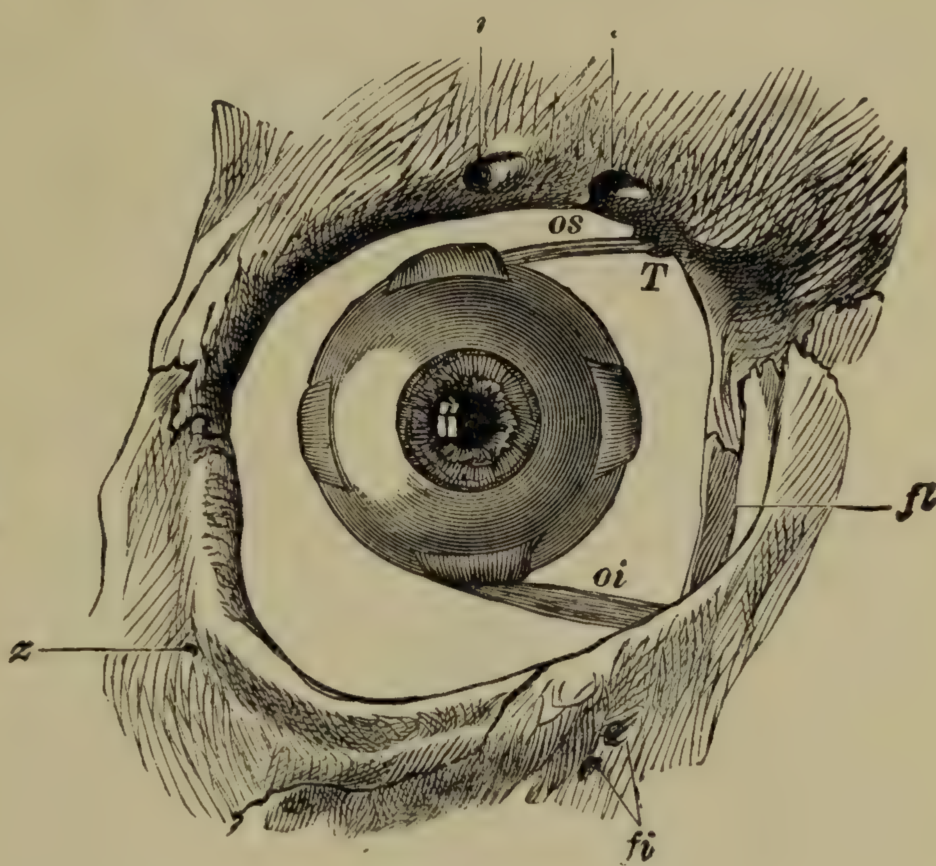


Fig. 48.

Fig. 47.—Horizontal Section through the Orbit (Schematic). Natural size. The apex of the orbit is formed by the foramen opticum, *F*. The external wall of the orbit does not reach as far forward as the internal, so that its anterior margin, *A*, lies in the same plane as the equator of the eyeball. At the anterior extremity of the internal wall of the orbit is shown the trochlea, *T*, which in reality lies in the upper and inner angle of the orbit, and hence would not be met with in the cross section of the orbit that is here represented. The external rectus, *e*, and the internal rectus, *i*, bound the muscular funnel, *t t*. A portion of the superior rectus (*su*) is cut away to show the optic nerve, *o*. s_1 is the insertion of the superior rectus upon the eyeball, lying oblique to the margin of the cornea. os_1 is the fan-shaped insertion of the tendon of the superior oblique, *os*; *oi*, the line of insertion of the inferior oblique. *f f* is the frontal, *s s* the sagittal axis of the eyeball.

Fig. 48.—Anterior Orifice of the Orbit with the Eyeball. Natural size. The tendons of the four recti muscles are cut off near their insertion upon the eyeball, but the inferior oblique, *oi*, and the tendon, *os*, of the superior oblique are left entire. The latter comes out from the loop of the trochlea, *T*. To the temporal side of the trochlea lies the supra-orbital notch, *i*, and somewhat to the outside of this there is a foramen, i_1, which is not regularly present, for a branch of the supra-orbital nerve. In this case the infra-orbital foramen, *fi*, is also abnormally divided into two distinct foramina. *z* is the orifice of the zygomatico-facial canal, *fl* is the lacrimal fossa.

back is continuous with the fascia which envelopes the muscle itself. By these "*lateral invaginations*," therefore, the tendons are connected with Tenon's capsule—a fact which is of importance with regard to the operation for squint.

The *lines of insertion* of the four recti muscles are situated at unequal distances from the margin of the cornea, and usually, too, are not quite concentric with it. Moreover, they are not perfectly symmetrical in their relation to the horizontal and vertical meridians. The mean relation of the lines of insertion are shown as accurately as possible in Fig. 49, which represents the anterior half of the eyeball projected upon a

plane. In it are marked the distances of the lines of insertion from the cornea in millimetres, as Fuchs found them from the mean of a great number of measurements.

The muscles are surrounded by *fasciæ* which are continuous anteriorly with Tenon's capsule at the spot where the latter is reflected upon the tendons of the muscles. Lateral prolongations of the fasciæ unite the muscles together, and also pass from them to the bony wall of the orbit. By means of this system of fasciæ pervading the orbit, the contents of the latter are fixed in place. It is owing to them that the eye does not leave its place when performing its movements, but turns about a fixed centre. The continuations of the fasciæ, passing from the muscles to the walls of the orbit, act as a sort of

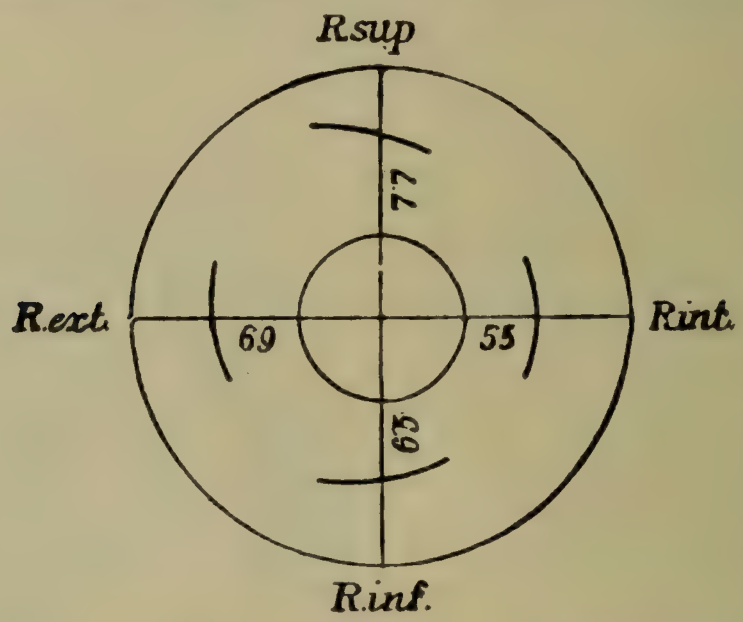


FIG. 49.—LINES OF INSERTION OF THE FOUR RECTI MUSCLES PROJECTED UPON A PLANE. Natural size.

restraining apparatus (*check ligaments*) which prevent extreme excursions of the eyeball (Merkel, Motais). They are most strongly developed upon the internal and external recti (*fi* and *fe*, Fig. 2). The superior oblique has two check ligaments, one attached to the direct, the other to the reflected portion of the tendon. The inferior oblique has no check ligament, its place being taken by the ligament attached to the reflected tendon of the superior oblique. The levator palpebræ superioris, too, which is associated in action with the superior rectus, is united to it directly by bands of fascia (see § 66). The inferior rectus is similarly connected with the inferior oblique. Furthermore, bands of fascia pass from the levator to the skin of the upper lid and also to the upper retrotarsal fold, so that these structures follow the movements of the eyeball and the upper lid when these are elevated. An analogous apparatus comes into play when the eyeball is depressed, bands of fascia running from the inferior rectus into the lower lid and to the lower retrotarsal fold (see § 66).

CHAPTER VII

DEVELOPMENT OF THE EYE

I. Embryology

55. Primordial Eye; Ocular Vesicle; Rudimentary Lens.[1]—The eye develops partly from the ectoderm, partly from the mesoderm. Two different portions of the ectoderm take part in the process, i. e., the medullary plate and the ectoderm on the surface of the body.

At the end of the second week in oblong furrow (*ocular furrow*) is formed in the most anterior part of the medullary plate on one side of the median line.[2] While the medullary plate in this anterior portion, which ultimately develops into the brain, is closing in to form the medullary tube, the laterally placed ocular furrow, constituting the rudimentary eye, deepens and enlarges to form a rounded vesicle (*ocular vesicle*; Fig. 50). At first this vesicle is in free communication with the lumen of the medullary tube, and its apex extends right up to, the ectoderm, from which it is separated only by embryonic (ectodermal) supporting tissue.

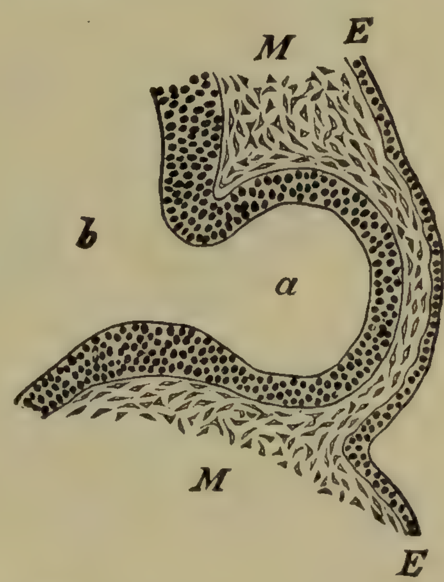


Fig. 50.—Section through the Embryonic Eye of a Human Embryo of Twenty-one Days. Magnified 100×1.

The primitive ocular vesicle, *a*, is a protrusion from the medullary tube (first cerebral vesicle), *b*, from which it is separated by a shallow constriction. It is surrounded by the cells of the mesoderm, over which passes the ectoderm of the external surface of the body.

At the apex of the ocular vesicle, which is directed somewhat ventrally (downward), a thickening takes place in the ectoderm, which represents the first rudiment of the lens and is hence called the *lens plate*. In the course of the third week this rudimentary lens sinks further and further in, so as to form, first, the *lens furrow* (Fig. 51, *L*) and then the *lens saccule* (Fig. 52, *L*), and finally is cut off altogether from the ectoderm to form the *lens vesicle* (Fig. 54, *L*). In the meantime the ocular vesicle grows round the rudimentary lens in front, dorsally (above), and behind, while its apex, which originally was convex, now since it remains in immediate contact with the lens vesicle, becomes quite concave (Fig. 51, *r*; Fig. 54, *r*), so that the lumen of the ocular vesicle becomes contracted to a narrow slit. The rounded ocular vesicle has thus become converted into the *ocular cup* (Fig. 53).

56. Fetal Ocular Cleft.—Owing to the fact that the growth of the ocular vesicle fails to take place on the lower (ventral) side, the ocular cup

[1] With some minor additions this chapter except § 63 is a free translation of the chapter on the same subject in the 13th German edition, edited by Salzmann.

[2] Facts observed with regard to the development of the monstrosity called cyclopia indicate that the primordial eye is single, developing from the median part of the medullary plate and later extending to either side (Hill). Thus the two eyes would be genetically one organ as they are physiologically (§ 234) (Translator).

presents a fissure (*fetal ocular cleft,* Fig. 53). The ocular cup is now some distance from the cerebral vesicle and connected with it only by a stalk (*ocular pedicle,* Figs. 51, 52, 53, 54). On the distal portion of this stalk the ocular cleft is continued as a groove, which grows shallower as it proceeds.

The ocular cup thus is produced by a sort of invagination of the apex of the ocular vesicle, and consequently has a wall composed of two laminæ, the thinner (*p*, Fig. 54) being on the outside, the thicker, *r*, on the inside. At the border of the cup and at the ocular cleft the two laminæ come together. The cavity of the ocular cup is filled by the lens vesicle (*L*).

By the invagination that produces the ocular cup the original cavity of the ocular vesicle is converted into a virtual cavity only, since the outer and inner layers of the cup are everywhere in contact though never in connection. This remains so through life, and hence arise consequences that are pathologically important. The retina separates from the pigment epithelium even more readily than the chorioid separates from the sclera (as it does, for example, after operation); and the dreaded detachment of the retina

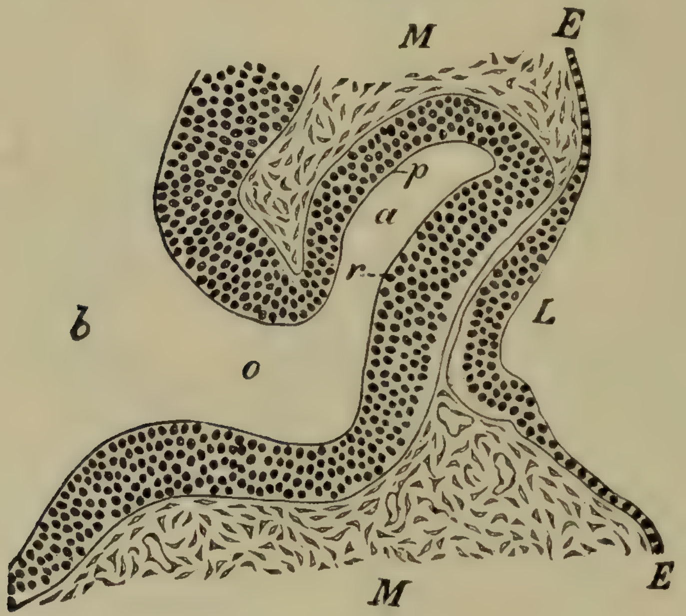


Fig. 51.

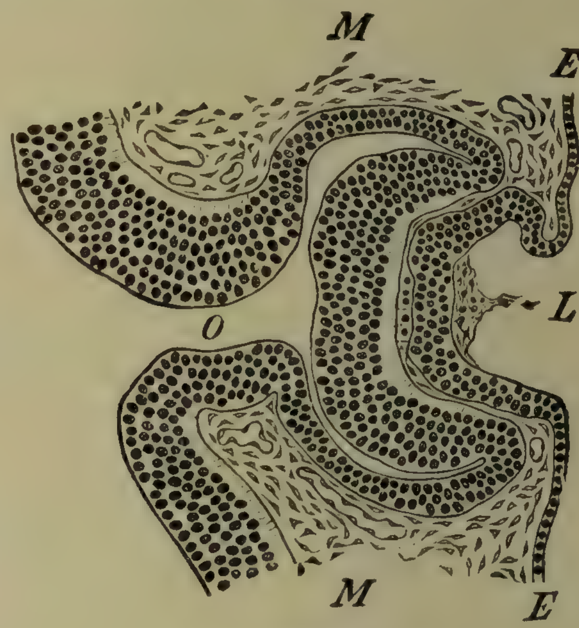


Fig. 52.

Fig. 51.—Section through the Embryonic Eye of a Human Embryo of Twenty-two to Twenty-three Days. Magnified 100×1. The vertically made section passes through the fetal ocular fissure. The ectoderm, E, dips in somewhat at the lens furrow, *L*, marking the site of the rudimentary lens, and besides is thickened, being made up here of several layers of cells. Corresponding to this in-dipping, the ocular vesicle, *a*, presents an indentation in its cavity, and is consequently converted into the ocular cup, the inner wall, *r*, of which subsequently becomes the retina, the outer wall, *p*, becoming the pigment epithelium. The interior of the ocular cup communicates with the medullary tube (first cerebral vesicle), *b*, through the ocular pedicle or rudimentary optic nerve, *o*. The projecting portion of the wall of the ocular cup is wanting below, because the fetal ocular cleft is situated here. *M*, mesoderm, in which, near the lower edge of the rudimentary lens, can be seen the cross section of a capillary vessel.

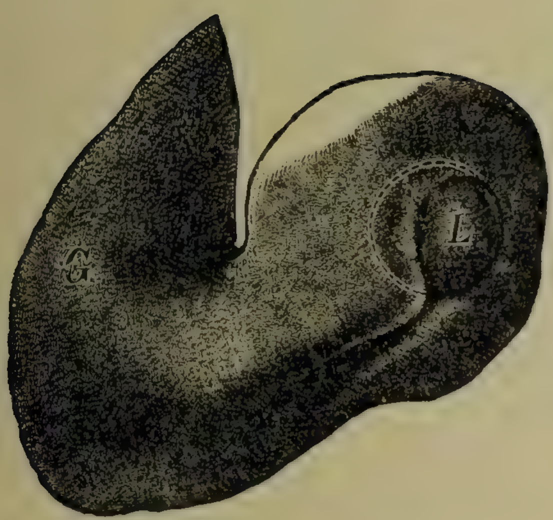
Fig. 52.—Section through the Embryonic Eye of a Human Embryo of Twenty-four to Twenty-five Days. Magnified 100×1. The section here depicted does not pass through the fetal ocular cleft, so that the secondary ocular vesicle appears as a complete cup; the more so since the portion of ectoderm, *E*, representing the rudimentary lens, *L*, has become invaginated further than in Fig. 51. In the bottom of the depression occupied by the lens there lies some cell detritus, and between the lens and the inner wall of the ocular cup are seen a few cells derived from the mesoderm *M*. In one or two spots in the mesoderm are visible the cross sections of capillaries. *O*, rudimentary optic nerve.

is nothing but the re-transformation of the virtual cavity of the ocular vesicle into a real one. Similarly in the iris the posterior epithelial layer (inner stratum of the ocular vesicle) separates readily from the anterior layer (outer stratum); this, for example, taking place in the rupture of posterior synechiæ, when the posterior layer remains on the lens, the anterior on the iris. Indeed, a spontaneous separation of the posterior from the anterior epithelial layer occurs not infrequently as a senile change.

The next, and for the understanding of the malformations of the eye a particularly important step in the development of the eye, is the *closure of the ocular cleft.* The lateral walls of the cleft grow toward each other, and finally come into contact and fuse. This fusion begins in the centre of the

cleft and in the course of the fifth week extends from this point forward and back. In front the fusion is complete, so that the anterior orifice of the cup —the primitive pupil—takes on a circular shape. At the posterior end of the cleft, however, a small gap remains.

When the ocular cleft closes, a portion of the mesoderm which surrounds the rudimentary eye is pinched off and thus gains access to the cavity of the ocular cup. At the border of the ocular cup and also at the posterior end of the ocular cleft this band of mesoderm retains connection with the mesoderm surrounding the rudimentary eye. It forms the starting point of the interior system of vessels. Meantime the mesoderm has also grown in between the lens vesicle and the ectoderm, forming the rudimentary cornea.



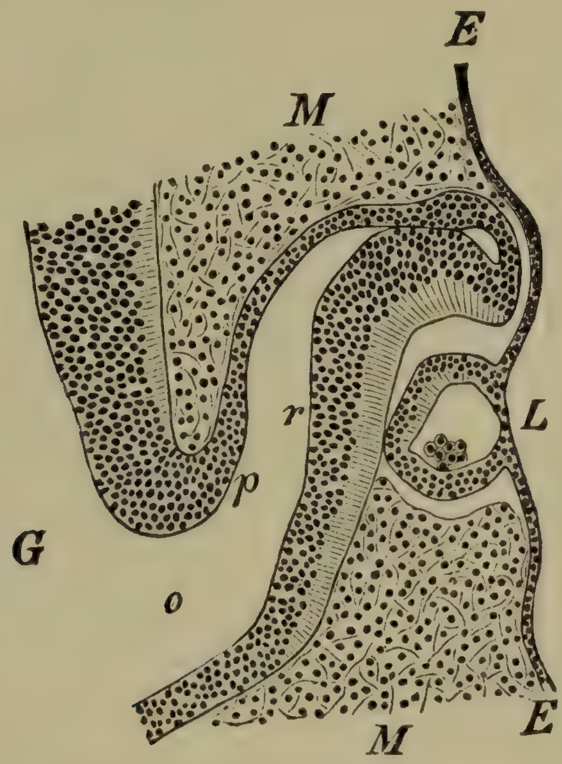


Fig. 53. Fig. 54.

Fig. 53.—Ocular Cup with Ocular Cleft Seen from in Front and a little Below. The drawing is taken from a model of Prof. Hochstetter's. It represents the rudimentary brain with its processes, but omitting the ectoderm and mesoderm. The eye rises by a thick hollow pedicle from the lateral wall of the first cerebral vesicle, *G*. At its distal end it presents an indentation, in which lies the embryonic lens, *L;* the posterior limits of the latter are indicated by a dotted curved line. From the lower margin of the indentation runs the fetal ocular cleft. This, at first, very narrow, afterward widens somewhat, and extends down upon the lower side of the ocular pedicle.

Fig. 54.—Ocular Cup with Ocular Cleft. Left Eye of a Human Fetus Twenty-seven Days Old, Seen in Vertical Section. Magnified 88×1. Out of the sections combined to form Fig. 53, the one selected for representation is that passing precisely through the ocular cleft; hence the lower wall of the ocular fissure is wanting here, just as it is in Fig. 51. The walls of the first cerebral vesicle, *G*, approach each other and form the ocular pedicle *o*, (subsequently the optic nerve), and further along form the reduplicated wall of the ocular cup itself. The external lamina, *p*, of this reduplicated wall which later is transformed into the pigment epithelium, composed of a single layer of cells, is at this time still destitute of pigment and composed of several layers of cells. At the anterior border of the ocular vesicle it is reflected to form the thick inner lamina, *r*. This latter, from which the retina proper is developed, shows a non-nucleated layer, constituting what is called the marginal mantle or rudimentary form of the supporting tissue of the retina. The anterior border of the ocular vesicle is covered by the ectoderm, *E*, *E*. The rudimentary lens, *L*, has already become closed so as to form a vesicle, but is not yet completely detached from the ectoderm. Above, the mesoderm, *M*, fills the space between the cerebral vesicle, the ocular vesicle, and ectoderm; but below, the mesoderm, wherever the ocular cleft extends, penetrates into the interior of the ocular cup till it reaches the lens vesicle.

57. Formation of Optic Nerve, Retina and Vitreous.—After this, i. e., from the sixth week on, the development of the separate portions of the eye proceeds as follows.

The optic pedicle, *O*, becomes the *optic nerve*. It grows considerably longer and loses its central cavity. From its cells develops the glial framework, while the nerve fibres grow out from the retina. Both septa and sheaths are derived from the surrounding mesoderm.

The outer lamina, *p*, of the ocular cup very soon becomes pigmented

Subsequently it becomes reduced to a single layer and develops into the *pigment epithelium*. The inner layer, *r*, becomes the *retina* in the narrower sense of the word. First to be differentiated is the rudimentary supporting framework, (Fig. 54), next the layer of ganglion cells. At first the retina is thickest in the region of the posterior pole (area centralis), and it is not until the end of the sixth month that there begins in the middle of the area that reduction of the cerebral layer, which results in the formation of the fovea centralis.

In the formation of the *vitreous* both lens and retina take part at the outset. Later the retina alone is concerned in this, and finally only the posterior portion of the pars ciliaris, to which even in after life the vitreous remains adherent.

58. Blood-Vessels of Fetal Eye.—The primitive interior system of vessels is differentiated in the third month, when there is formed a trunk, the *hyaloid artery* (arteria centralis corporis vitrei, Fig. 55, *C*). This starts from the posterior end of the ocular cleft in the ocular pedicle (at a point where later the central vessels enter the optic nerve) and runs forward in the axis of the eye as far as the lens. This trunk sends lateral branches to the primitive vitreous (vasa hyaloidea propria), and ultimately breaks up into a capillary network (*tunica vasculosa lentis*) which envelops the lens and also lies in the cavity of the ocular cup.

It is only in the posterior segment of this system, which is enclosed in the ocular pedicle, that a vein (vena centralis retinæ) is present. Anteriorly there are no veins, the tunica vasculosa lentis emptying round the edge of the cup directly into the surrounding mesoderm.

From the hyaloid artery blood-vessels bud out into the retina, and where the artery passes out into the vitreous it is enveloped in a mantle of glia tissue which projects like a plug into the vitreous and when fully developed is 2 mm. long.

In the fifth month this system begins to *disappear*, the vitreous vessels being the first to go. Between the seventh and the ninth month the hyaloid artery and the tunica vasculosa lentis also disappear. The only part that remains is that situated in the optic nerve, which supplies the retinal system of vessels and now bears the name of the arteria centralis retinæ.

59. Formation of Lens, Chorioid, and Sclera.—The cavity of the lens vesicle is filled up by the outgrowth of the cells of the posterior wall, developing in a sagittal direction so as to form fibres (Fig. 55). The anterior half of the vesicle remains in its original state, and later forms the epithelium of the lens. At first the lens, which at this time is almost spherical, grows in such a way that only sagittal fibres are formed. These fibres all contain nuclei, and the arc formed by them, therefore, traverses the entire lens. It is not till much later that the new formed fibres assume a direction parallel to the surface and thus give rise to the laminated structure of the lens. When this occurs the nuclei in the centre of the lens, where the fibres still have a sagittal direction, disappear, and the arc of nuclei terminates in the equatorial zone on either side. From this time on growth in the lens takes place in the same way as after birth.

The mesoderm which surrounds the ocular cup very early differen-

tiates into a capillary layer adjoining the cup and an exterior, tougher layer, poor in vessels. The former is the rudimentary *chorioid*; the latter the *sclera* (Fig. 55).

60. Formation of Cornea, Iris, and Anterior Chamber.—The primitive cornea differentiates into an anterior, non-vascular layer and a posterior layer containing an abundance of vessels and cells. The former is the origin

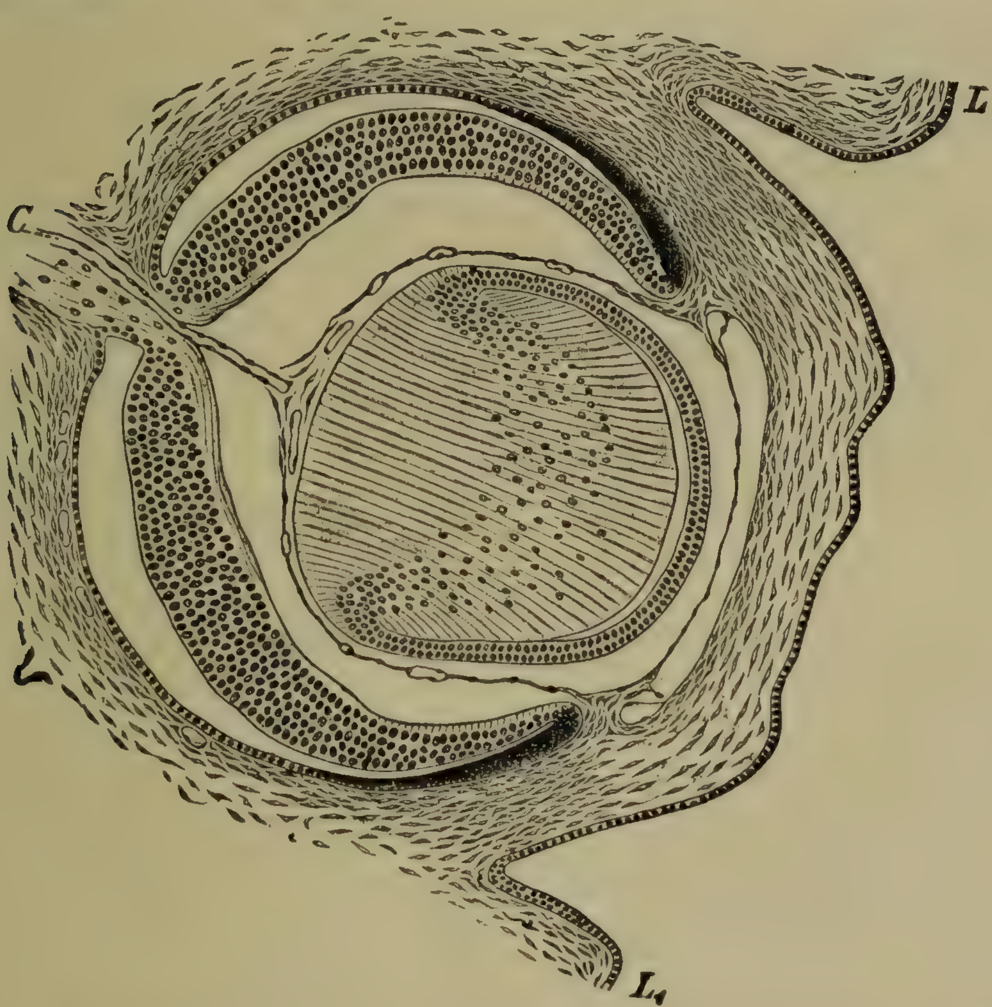


FIG. 55.—SECTION THROUGH AN EYE OF A HUMAN EMBRYO IN THE MIDDLE OF THE THIRD MONTH. Magnified 73×1.

The envelope of the ocular vesicle is formed of mesoderm, and in its anterior segment consists of the cornea, which contains an abundance of nuclei throughout and is separated by a particularly marked accumulation of nuclei from the posterior segment. In this posterior segment no delimitation between sclera and uvea has as yet taken place. The uvea will develop from the inner layers which are distinguished by containing more nuclei—a characteristic which is continued over into the hindmost, or uveal, layers of the cornea. At a point corresponding to the anterior margin of the ocular vesicle the mesoderm projects into the interior of the eye, and from the free border of the ring-shaped process thus formed (constituting what is later the iris) rise two delicate vascular membranes which surround the lens constituting a sort of vascular lenticular capsule. The one lying in front of the lens is the lamina iridopupillaris. Into the hinder of these two membranes the hyaloid artery enters at a point corresponding to the posterior pole of the lens. This artery rises from the central artery, *C*, of the optic nerve. Of the two laminæ composing the secondary ocular vesicle, the external, or pigment epithelium, has been reduced in its posterior part to a single layer of cells, while in its anterior portion there are still several cell layers present which have already taken up pigment. The inner lamina, or retina, consists of numerous layers of cells. In the immediate vicinity of the optic-nerve entrance can be seen the way in which one lamina is reflected into the other. The anterior point of reflection corresponds to what is later the pupillary margin of the iris. The lens is of an almost spherical shape; its antero-posterior diameter, in fact, is actually somewhat larger than its equatorial. Upon the anterior surface of the lens lies the epithelium, which still consists of several layers of cells; and no distinct lens capsule has yet been differentiated. In the region of what is later the equator of the lens the epithelial cells are growing out into lens fibres, which are still nucleated throughout, and take a sagittal direction. The posterior surface of the lens is destitute of epithelium, and is covered by an extremely delicate capsule. The vitreous cavity is very small. *L*, L_1, the eyelids growing out.

of what is later the *cornea;* the latter is the *lamina iridopupillaris* (Fig. 55; *P*, Fig. 56), which at the border of the ocular cup is in communication with the tunica vasculosa lentis and serves to prolong the latter anteriorly.

Toward the end of the third month the border of the ocular cup together with the adjoining mesoderm begins to grow out. The border of the

cup is thus converted into the *pars cæca retinæ* (Fig. 56). This consequently consist of two layers. The exterior is pigmented throughout, and at the point where the inner layer is reflected this pigmentation passes over onto the latter also. At this point of reflection of the inner layer there develops in the fourth month a ring-shaped accumulation of cells, which subsequently grows out into the mesodermal tissue and becomes the *sphincter pupillæ*. In this way the development of the iris commences. A fold in the pars cæca further back represents the beginning of the *ciliary body*.

As the sphincter develops the lamina iridopupillaris divides into a thicker, peripheral portion (*primitive iris*) and into the *pupillary membrane* proper.

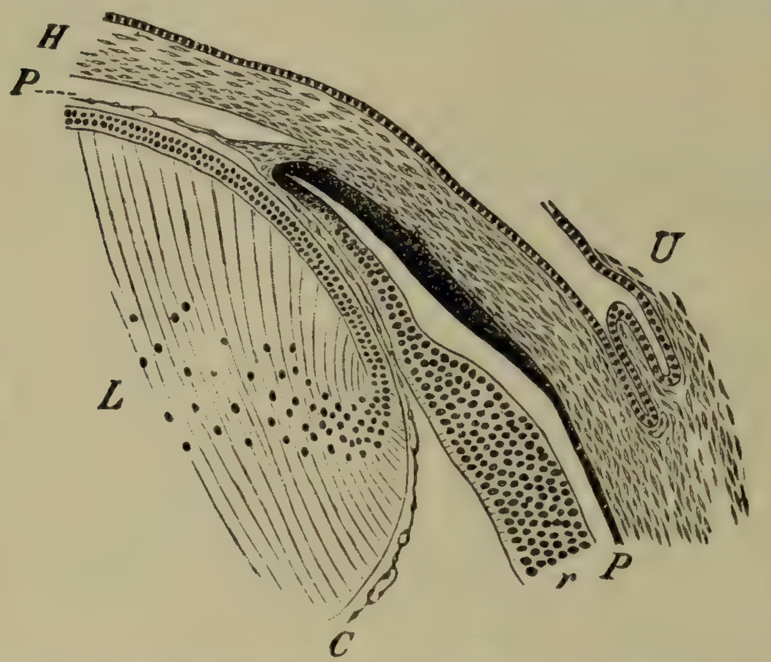


FIG. 56.—ANTERIOR SEGMENT OF THE EYE OF A HUMAN EMBYRO AT THE END OF THE THIRD MONTH. Magnified 80×1.

The epithelium of the cornea, *H*, is continued over upon the conjunctiva as far as the retrotarsal fold, *U*. Behind the cornea is seen the front wall of the ocular vesicle. The two laminæ forming this wall are reflected so as to unite with each other at a point corresponding to the margin of the pupil. The external lamina, *p*, is pigmented throughout, the internal lamina being pigmented only in its anterior portion, where it later is converted into the posterior stratum of the retinal pigment layer of the iris. This portion of *r* is the beginning of the pars cæca retinæ. Farther back, where the inner lamina is destitute of pigment, it is afterward converted into the inner layer of cells of the pars ciliaris retinæ, which lines the ciliary body. Still farther back a sudden swelling out of the inner lamina denotes the beginning of the retina proper, *r*, at a spot corresponding to what is afterward the ora serrata. The two laminæ of the ocular cup, so far as they constitute the coating of the ciliary body, lie closely applied to the mesodermal envelope. Farther, forward, at a point corresponding to the rudimentary iris they separate from the cornea, from which they receive a covering of mesodermal tissue, afterward converted into the stroma of the iris. From the free border of this tissue are given off two membranes—the membrana pupillaris, *P*, which passes to the opposite pupillary border, and the membrana capsularis (tunica vasculosa lentis), *C*, which runs backward, between ciliary body and lens, to the posterior surface of the latter. In the lens, *L*, the circle of nuclei is carried farther forward than in Fig. 55 and the shape of the lens in cross section has already become more elliptical.

According to S. Cirincione, the *anterior chamber* develops first at the periphery at a point corresponding to the situation of the rudimentary iris, and remains in this state as a ring-shaped slit until the beginning of the eighth month. The rudimentary iris, especially the part that contains the sphincter, gets constantly broader. More and more evident also becomes the division of the pars cæca retinæ into the *pars ciliaris* and *pars iridica retinæ* (see §§ 27, 30, 44). From the outer layer of the latter there develops in the sixth month the *dilatator pupillæ*. In the eighth month the pupillary membrane becomes completely detached from the cornea. Then it begins to atrophy, the process starting in the centre first and then extending to the periphery. The lesser circle of the iris represents the only remains left in this process of retrogression.

61. Genetic Relations Summarized.—From the preceding description it will be seen that the outer and middle coats of the eye (corneo-sclera and uvea) are derived from the mesoderm, the lens is derived from the ectoderm of the external surface of the body, and the retina, including the pigment epithelium and its anterior prolongations (pars ciliaris retinæ and pars iridica retinæ together with the sphincter pupillæ and dilatator pupillæ) is derived from the interior ectoderm, i. e., from the medullary plate (primordial brain). The vitreous is an ectodermal structure, being derived partly from the lens, partly from the retina.

It is to be noted that the structural interrelations of these parts of the eye are determined by function, rather than by their embryonic source. Thus the uvea and the corneo-sclera, which have the same genetic origin, being both derived from the homogeneous mesoderm, not only become anatomically distinct, but ultimately get to be separated by a spatial interval (the anterior chamber in front, the perichorioidal space behind).

Thus the membranes which originally formed one single tissue are finally connected at two places only, i. e., at the spot where the ciliary body is attached to the sclera and at the margin of the optic-nerve entrance.

Quite the reverse are the spatial relations of the middle coat of the eye to the inner coat, namely, the retina and its anterior prolongation. The inner coat of the eye originates from the secondary ocular vesicle. With this the uvea has nothing at all in common as a tissue, but simply lies in contact with the outer layer of the vesicle. From this layer develops later the retinal pigment. This latter simply lies on the inner surface of the chorioid and ciliary body without being connected with them, yet in the living eye it very rarely separates from the chorioid and never from the ciliary body. Still more intimate is the connection of the retinal pigment with the iris, in whose tissue the muscle fibres of the sphincter and dilatator pupillæ, which are derived from the retinal pigment, lie imbedded. That is, the genetically related membranes, the outer and the middle coats of the eye, lose their anatomical connection almost completely, because functionally they have little to do with each other, while, on the other hand, a functional relationship leads to an intimate connection between the middle and inner coats, which genetically are perfectly distinct.

62. Lids, Lacrimal Passages.—The lids originate as folds, which keep growing out above and below the eye from the skin surrounding it, until their edges come into contact. They then become united, but only by their epithelial lining; and shortly before birth this union is dissolved. The lacrimal gland originates from a bud-like intrusion of the epithelium of the conjunctiva into the orbital tissue. The lacrimal canal starts from a channel which exists even at an early period between the superior maxillary and external nasal processes.

II. Postnatal Development; the Adult Eye

63. Eye at Birth; Postnatal Changes.—In the newborn infant the antero-posterior diameter of the eye varies from 15.8 to 17.5 mm. and the vertical diameter from 14.5 to 17.0 mm. To offset the comparative shortness of the eye, which would make it exceedingly hypermetropic, the media are more highly refractive than in the adult, the seat of the excess of refractivity being in the lens (§ 190). The uvea is much less pigmented. (see § 28).

The eye grows rapidly during the first few years of life, the vertical diameter growing faster so that the eye becomes more nearly spherical (Weiss). According to Occhi it reaches the adult size at about the age of 8 or 9.

As the eye elongates the lens becomes less refractive (see § 191). More pigment is laid down so that the uvea as a whole and especially the iris becomes darker, (see § 28). The eyes get farther apart, and the orbits enlarge, especially anteriorly, so that their temporal borders are more widely separated. As the eyes separate they also tend to diverge, since the separation makes the external rectus act to greater, and the internal rectus to less advantage than before (see § 331).

These pigmentary, refractive, and positional changes produced by the process of growth have a considerable bearing on the functioning of the eyes.

The lens keeps growing all through life (see §§ 22, 841).

In the adult the dimensions of the eye in mm. are as follows (Merkel and Kallius).

Antero-posterior diameter	24.3
Transverse diameter	23.6
Vertical diameter	23.3
Thickness of cornea at apex (Gullstrand)	0.5
Thickness of cornea at base	1.1
Diameter of cornea (diameter of anterior chamber)	11.9
Depth of anterior chamber	2.3
Thickness of lens (eye not accommodating)	3.7
Thickness of lens (eye accommodating)	4.3

The antero-posterior diameter of the eye is usually greater in myopes and less in hyperopes than that above given (see §§ 193 and 198).

The centre of rotation of the eye is 13.5 mm. back of the apex of the cornea (Helmholtz).

For the position of the cardinal points see § 161.

PART II

GENERAL PHYSIOLOGY AND PATHOLOGY OF THE EYE

GENERAL PHYSIOLOGY AND PATHOLOGY OF THE EYE

64. The eye, or more properly both eyes together constitute an organ in which two mechanisms are more or less intimately fused. The first mechanism (corneo-sclera, uvea, lens, and vitreous) has two functions. First, it protects the other mechanism and nourishes it; second, it conveys to it its specific stimulus, light, in a manner suited to set up the proper excitation. The other mechanism (retina, optic nerve, muscles, and their central connections) is a sensorio-motor apparatus which receives the light stimuli, transmits them to the brain, and receives from the latter motor impulses which assist in the sensory act.

In Part II of this book we shall consider the general physiology and pathology of the first of these mechanisms, in so far as it subserves protection and metabolism; and in Part III the actions and derangements of the same mechanism when serving as a conductive (refracting) medium. In Parts IV and V the neurology of the eye, i. e., the physiology and pathology of the second, or sensorio-motor mechanism will be considered. The three parts just named necessarily take up in detail the functional examination of the eyes in all its details, and, with this is given a description of the methods used in objective examination, so that the whole routine of examining patients shall be brought together in orderly sequence. Thus the reader is better prepared to consider the remaining Parts VI and VII, which take up the symptomatology and treatment of diseases of the separate parts of the eye.

CHAPTER VIII

GENERAL PHYSIOLOGY OF THE EYE

I. Protective Mechanism

65. Protective Agencies.—Exteriorly the eye is protected from injury by bony walls and the fat of the orbit behind and by the lids and conjunctiva in front. The lids act in two ways. First by their tight closure they shut out foreign bodies and excess of light. Second, by winking movements they remove foreign bodies including bacteria from the surface of the eye. They cannot do this unless at the same time the surface of the eye is moist and the moisture can be drained off from the eye. Both objects are effected by the lacrimal organs which both secrete the tears and carry them off into the nose. Finally the conjunctiva, especially that coating the surface of the cornea protects the eye from the entrance of germs and, in the case of the cornea from an imbibition of liquid which would impair the transparency of the latter.

In its own structure the eye finds protection in the rigidity of the corneo-sclera and in the sensitiveness of the cornea, which causes a reflex winking as soon as any foreign body lights on the cornea.

The way in which these mechanisms act is as follows.

66. Movements of the Lids.—These are performed in the following way: In *opening the eye* the upper lid is raised by the levator palpebræ superioris, while the lower lid sinks by its own weight, although it does so but very little. Owing to the fact that fibres from the tendon of the levator run to it, the skin of the lids above the convex border of the tarsus is drawn quite far in between the eyeball and the upper margin of the orbit, at the same time that the upper lid is raised. In this way there is formed a furrow, over which the lax skin of the lid hangs down under the form of a fold (covering fold, *d*, Fig. 1). In many cases this is so large as to reach the free border of the lid, and cause disfigurement (ptosis adiposa; § 425).

The upper lid is raised not only by the levator, but also by the musculus tarsalis superior (see § 3) and by the superior rectus. For the tendon of the latter is so solidly united with that of the levator by fascial bands that it may be said to have a common insertion with the levator in the tarsus of the upper lid. Hence, when the superior rectus contracts and raises the eye, it assists in lifting the upper lid and the retrotarsal fold at the same time. The same thing happens if the superior rectus retracts from any cause, e. g., as the result of a complete tenotomy producing a traumatic paralysis. The upper lid is then pulled up, and the eye consequently is wider open than its fellow. On the contrary, in an advancement, in which the tendon of the superior rectus and with it the fascial bands are brought forward, the upper lid is carried forward too, and the eye is 1 or 2 mm. less open than its fellow. The same thing happens in a paralysis of the superior rectus, not due to division of its tendon; the relaxed muscle allows the lid to droop somewhat, especially when the eyes are carried up.

The lower lid is carried down not only by its weight, but also by the action of the musculus tarsalis inferior (supplied by the sympathetic) and by a slip which runs from

the tendon of the inferior rectus directly to the tarsus. Hence, when the inferior rectus pulls the eye down it depresses the lower lid at the same time, and hence also, as in the case of the superior rectus, a complete tenotomy of the inferior rectus makes the palpebral fissure wider and an advancement of this muscle makes it narrower.

By expansions from their tendons the external and internal recti also act upon the lids; so that when the eye is abducted the outer canthus is pulled outward, and when the eye is adducted the inner canthus is pulled back and in (Dwight).

With regard to the shutting of the eyes, we must distinguish between winking and tight closure of the lids. *Winking* consists in a quick contraction of the palpebral fissure, in which the lids do not come into perfect contact. It can be performed voluntarily but usually results through reflex action, which is excited by the sense of dryness in the eye or by the presence of foreign bodies—dust, smoke, etc. It is effected by means of the trigeminus, which is the sensory nerve of the eye and its vicinity, and is hence rightly called the sentinel of the eye. The purpose of winking is to cover the surface of the eyeball with a uniform layer of the lacrimal fluid and thus both prevent the drying of the eye and also wash the dust off of it.

In *firm closure* of the lids, which usually is done voluntarily, the edges of the lid are brought into complete contact. This may be done gently as in sleep or forcibly as in the act of squeezing the lids together. In the latter case the skin about the lids is drawn toward the palpebral fissure and at the same time is thrown into numerous wrinkles. When the lids close as, for example, in sleep, the eyeball also performs a movement, rolling upward (Bell's phenomenon). Any one, when he is fighting against sleep and his lids are shutting together, can feel that the eyes are being drawn up as by an invisible force. In persons with thin lids (women and children) we can recognize the convex cornea through the upper lid, and determine that it is directed upward beneath the closed lids. It is still easier to do this in cases of staphyloma of the cornea. This behavior on the part of the eyeball is important, inasmuch as the protection of the cornea by the upper lid is thus provided for, even when the palpebral fissure is not completely closed in sleep. It is not till lagophthalmus reaches quite a high degree that a portion of the cornea remains constantly visible in the palpebral fissure; and this portion is, in fact, always the lowest part of the cornea, which consequently is most exposed to the danger of desiccation (keratitis e lagophthalmo; see § 558).

Winking is effected by the peripheral portion of the orbicularis, while in firm closure of the lids, the orbital portion of this muscle also comes into play.

67. Secretion and Discharge of Tears.—*Secretion of tears* results from psychic excitation ("weeping") or in a reflex way from irritation of the trigeminus or optic nerve ("watering of the eyes"). Psychic weeping occurs only in man, and even in man is absent in the case of infants. The tears contain only a few solid constituents, the main part of which is sodium chloride (hence "salty" tears). In the normal state the lacrimal gland secretes scarcely any more liquid than is lost by evaporation from the surface of the eyeball, so that but very small quantities of fluid are discharged into the nose. It is only when the secretion is much increased that

any considerable quantity of tears is discharged into the nose, where its presence is manifested by repeated blowing of the nose.

The moistening of the eyeball is not due to the lacrimal glands alone. The secretion of the conjunctiva itself, and also of its glands, helps to effect this. Hence it follows that even after removal or degeneration of the lacrimal gland the eye does not become dry.

In the *conduction of tears* into the nose there are two factors to be considered: The entrance of the tears into the lacrimal sac, and their transmission from the latter to the nose.

The conveyance of tears through the puncta into the lacrimal sac is effected by the act of winking. The tears accumulate in the horseshoe-shaped notch in the inner angle of the eye, which forms the lacus lacrimalis into which the puncta dip. Then a winking movement takes place, the fibres of the palpebral portion of the orbicularis contracting. These fibres (see § 3), spring from the internal palpebral ligament and when they contract draw the ligament away from the lacrimal bone. The wall of the lacrimal sac, being connected with the palpebral ligament, is drawn along with it, so that the sac is dilated, and the contents of the canaliculi are sucked into it. The subsequent conveyance of the tears from the sac into the nose is effected by the elasticity of the sac. In virtue of this elasticity, the sac when distended by the tears tends to contract again and thus expel the tears. Hence, in those pathological cases in which the lacrimal sac has lost its elasticity (atony of the sac) we observe that the conduction of tears downward is arrested, even though the nasal duct is completely pervious. The reason why the tears are not driven back again into the canaliculi by the contractions of the lacrimal sac, but pass on into the nasal duct, is that the latter is much wider than the narrow canaliculi.

68. Protection of the Middle and Inner Coats.—Exteriorly the uvea and retina are protected by the corneo-sclera, which form a rigid capsule closely applied to the uvea; interiorly they are protected by the aqueous and vitreous which fill the interior of the eye and prevent collapse of its coats.

The *rigidity* of the cornea and sclera is due to the fact that they are composed of densely laminated and interlaced, inflexible connective-tissue fibres, which are traversed by a moderate number of elastic fibres, so that with great rigidity there is afforded a slight, but only a slight, degree of elasticity.

The rigidity of the corneo-sclera is lessened at two points—first and most at the spot where the optic nerve enters the eye and where the sclera is reduced to a set of delicate fibrous bands called the lamina cribrosa; next at the corneo-scleral margin where the scleral gutter is hollowed out to form Schlemm's canal. Hence the spot where the optic nerve enters the eye is the first place to yield to intra-ocular pressure (pressure-excavation); and the corneo-scleral margin is the typical location for a rupture of the fibrous coats of the eye due to external violence. The slight degree of elasticity of the corneo-sclera is the reason why a uniform expansion of the latter does not take place when the intra-ocular pressure is increased, except in youth, when the elasticity is still fairly great (hydrophthalmus). As age increases, the corneo-sclera becomes less and less elastic, and admits of only partial protrusions, and in these, moreover, it is no longer a question of simple stretching but of rupture also (in the cornea, ruptures of Descemet's membrane in the case of hydrophthalmus and keratoconus; in the sclera, ruptures of its innermost lamellæ in the case of scleral staphylomata). The slightness of the elasticity

makes itself apparent also when there is reduction of the intra-ocular tension. In a more elastic eyeball such reduction would lead to a uniform contraction of the organ, whilst in the human eye in this case folds are formed both in the cornea and sclera (e. g., in atrophic and phthisical eyes).

The character of the *uvea* with regard to rigidity varies. The iris is extremely elastic. The elasticity of the iris tissue is necessary if the play of the sphincter and the dilatator of the pupil is to take place readily. The elastic character of the iris is shown clinically in the frequent cases of peripheral incarceration of the iris, e.g., after phlyctenular ulcers, when we often see the iris of the opposite side drawn over to the very margin of the cornea (Fig. 239). If we wish to detach an anterior synechia by introducing a narrow knife into the anterior chamber and trying with this to divide the tag of iris that runs forward to the cornea, the attempt is usually unsuccessful, because the iris is so elastic that it is more readily stretched than cut even by the sharp knife. On account of the great elasticity of the iris, we practically never find transverse rupture of the iris after injuries, but simply separation of it from the ciliary body, or radial fissures, the latter because, owing to the radial course of its blood-vessels, the iris is very readily indeed split in a radial direction. Compared with the iris, the ciliary body and chorioid are inelastic. They rupture easily upon contusion of the eyeball, splits in the chorioid being then often observed. (Splits in the ciliary body, although also not rare, cannot be seen clinically.) Stress of a more gradual kind may also lead to rupture of the chorioid. Corresponding to the atrophic spots which occur in very myopic eyes in the region of the macula lutea, we often find ruptures, especially of the lamina vitrea, with gaping of the torn edges, caused by stretching of the chorioid (Salzmann).

Rigidity alone would not afford the eye sufficient protection. "Pain is man's guardian." The sclera, which in large part lies imbedded in the orbital tissues, does not need to have any great sensitiveness, and hence has but few nerves; but the cornea, which is directed toward the outer world, has the greatest *sensitiveness* of any part of the surface of our body and has consequently the greatest profusion of nerves. A dense plexus of nerve fibres, in fact, lies in the epithelium itself, a thing which is not the case elsewhere on the surface of the body. Hence it is that in most men even the slightest contact of anything with the cornea starts up a sensation not of pressure but of actual pain; and thus it is guaranteed that the most inconsiderable lesions, the smallest foreign bodies, excite out attention and are not neglected.

II. Maintenance of Optical Efficiency

69. Factors Producing Transparency.—The refracting media of the eye (i. e., the cornea, aqueous, lens, and vitreous) as well as the anterior layers of the retina which lie in front of the percipient elements (rods and cones) must be transparent. Transparency in the case of these organs is due to two factors—first, the homogeneous character of the elements composing them; second, the absence of opaque structures, especially blood-vessels.

As regards the first factor, the cornea is built up of lamellæ which are very homogeneous, refract the light equally, and have comparatively few cells between them. Very much the same thing is true of the lens which is composed of a series of homogeneous lamellæ arranged in layers, which though they differ from each other in refractivity, yet differ in a regular fashion. The aqueous is a limpid liquid; the vitreous a homogeneous jelly with very few structural elements.

As regards the second factor, the cornea, aqueous, lens, and vitreous are destitute of vessels and contain practically no other opaque tissue elements. In the retina there is no opaque connective tissue and the vessels are comparatively scanty and in the part most essential to clear vision—the fovea—are absent altogether. Moreover, in the retina the nerves lose their myelin, so as to be transparent (see § 47).

Conditions unfavorably affecting either of these factors cause *opacity* of the media.

This is produced, for example, by anything that alters the homogeneous character of the cornea or lens. Thus a temporary opacity of the cornea may be produced by strong pressure which while it lasts renders the corneal lamellæ unequally refractive; or it may be produced by wrinkling of the corneal lamellæ, even though these themselves are transparent. Such a wrinkling accounts for the striate opacity following operations. Again, the lens may become more or less opaque from irregular changes in the refractivity of its layers, from the formation of clefts, or from displacement and distortion of its fibres.

Most opacities, however, are produced by tissue changes. Even mere soaking with liquid suffices to make the substance of the cornea or lens swell and become opaque. Thus when a breach is made in the anterior epithelium of the cornea, e.g., by an ulcer or a flap incision, the edges of the defect often become swollen and cloudy from imbibition of the lacrimal fluid. In glaucoma the cloudiness of the cornea is also caused by fluid which is thrown out in minute droplets, particularly between the epithelial cells. Again, wherever the endothelium of the cornea or the capsular epithelium of lens is injured so that the aqueous can obtain access to these tissues, the latter swell and become opaque. In the case of the lens every lesion of the capsular epithelium leads sooner or later to cloudiness of the lens. Thus are explained not only traumatic cataracts with rupture of the capsule, but also many other cases of cataract in which the epithelium is injured without rupture of the capsule. This behavior on the part of the lens finds practical application in the procedure of making a partially clouded lens completely opaque (artificial ripening of cataract). This procedure consists in massaging the anterior surface of the lens, so that the capsular epithelium is injured by compression. Another cause of clouding of the lens is afforded if the composition of the aqueous or vitreous is essentially altered, or poisonous substances are present in these liquids, so that now as a result of diosmosis through the intact lens capsule the liquid that permeates the lens undergoes a change of composition. In this way either the capsular epithelium may be injured and thus the lens fibres be rendered cloudy in an indirect way, or the lens fibres may be affected directly. Thus are explained the opacities of the lens dependent on poisoning (e. g., naphthalinic cataract) and also most cases of complicated cataract, in which by disease of the inner coats of the eye an essential change is set up in the aqueous or vitreous. (Cf. also §§ 656, 658.)

Tissue changes leading to opacity are produced not only by the entrance of liquid, but even more often by the deposition of cells and exudates. This occurs, e. g., in the cornea in all cases of recent keratitis, in the aqueous as a result of inflammation or injury of neighboring structures, and in the vitreous and retina as a result of inflammation in these organs or in the parts adjacent. After the subsidence of inflammation, especially in the cornea, new-formed connective tissue and vessels may be left, and thus produce a permanent opacity, while in true scars of the cornea the corneal tissue is actually replaced by opaque connective tissue.

70. Maintenance of Other Optical Requirements.—To be a satisfactory optical instrument the eye must retain the proper *curvature and smoothness* of its refractive surfaces. These latter are the anterior and posterior surfaces of the cornea and of the lens. In the case of the cornea its curvature is maintained by its rigidity and it is kept smooth by being constantly moistened by the tears (see §§ 65, 67). In the lens the shape is the result of two counteracting forces—the elasticity of the lens which tends to make it bulge and the tension of the zonula which tends to flatten it. The shape may be altered by the contraction of the ciliary muscle, which as it relaxes the zonula allows the lens to bulge.

The eye has been justly likened to a camera. A good camera must have absolutely *opaque walls* so that light cannot enter except through the camera lens, it should have a *stop* over the lens to cut off the peripheral rays which cause aberration and to cut down the illumination when too great, and it must have an *adjusting mechanism* by which it can be focused

In the case of the eye extraneous light is cut out by the profuse pigment of the uveal tract; the stop (in this case an automatically adjustable one) is the iris (see § 365), and the focusing is done by the ciliary muscle (see § 177).

Defects in any of these regards impair the optical efficiency of the eye. Thus deformations of curvature of the cornea or lens cause errors of refraction and consequent poor vision; dryness of the cornea produces blurring of the sight; absence of uveal pigment or undue dilatation of the pupil causes dazzling; paralysis of the ciliary muscle prevents the adjustment of the eye for near points.

III. Nutrition of the Eye

71. Vascularity of Different Parts of the Eye.—The conjunctiva and uvea are very vascular, the uvea, indeed, being mainly composed of vessels. On the other hand, the sclera contains very few vessels, which, however, suffice for its nutrition, and the cornea, lens, and vitreous contain none at all and hence must derive their nourishment entirely from adjacent structures.

The *cornea* gets its nutritive material from the vessels of the corneo-scleral margin—i. e., mainly from the pretty dense marginal network of the limbus and from the scanty twigs which the anterior ciliary arteries that pass through the sclera give off deep down. Because the tissue metamorphosis of the cornea is so meagre, the deep vessels, few as they are, suffice to maintain it, for we can remove or cauterize the marginal network very extensively or even all the way round the limbus without the cornea's becoming necrotic. The *lens* and *vitreous* are dependent ultimately on the uvea.

The absence or poverty of vessels in the corneo-sclera and in the lens and vitreous is possible because the nutritional demands made by these structures is small. Their functions being merely rigidity or transparency, no consumption of material of any amount takes place in them, and their tissue metamorphosis is, therefore, very slight. And, in the case of the refractive media it is important that vessels should be absent in order that the media themselves shall be transparent (§ 67).

To satisfy optical conditions the *retina* also must be transparent and hence its vessels are comparatively few. But the nutritional demands here are so great that this limited blood supply would not suffice and the retina, therefore, both for nutrition and function is also dependent on the chorioid. The vessels of the retina proper lie in the inner layers, the vessels of the chorioid (chorio-capillaris) adjoin the outer layers of the retina, and consequently the inner layers are nourished by the retinal, the outer by the chorioidal, vessels. Neither one of the two vascular systems suffices by itself to maintain the function of the retina, and the latter, therefore, is dependent on both. Diseases of either one system or the other cause injury to the retina. And there is the additional fact that the relations of the circulation are very unfavorable for the compensation of circulatory disturbances. The retinal vessels have no anastomoses with each other. The retinal arteries are end-arteries, and closure of any of them at once puts the section of retina that is supplied by it out of the circulation. Furthermore, there are no anastomoses worth mentioning between the retinal and ciliary systems of vessels, so that the latter, whose circulatory relations are far more favorable, cannot substitute for the former. These circulatory relations together with the extremely delicate and complicated structure of the retina cause it to be readily diseased in spite of its protected situation.

Owing to the provisions for compensation afforded by its abundant blood supply and the very numerous anastomoses between its vessels, the uvea is much better able than the retina to withstand disturbances of circulation. The evidences of vascular occlusion—embolism and thrombosis—which in the retina afford such characteristic clinical pictures, in the uvea can at most be demonstrated anatomically, and produce no symptoms that can be clinically recognized. A further evidence of compensation is

seen in glaucoma, in which when the outflow of venous blood through the venæ vorticosæ is impeded, the anterior ciliary veins take their place and carry off more blood then usual. The only unfavorable factor in the circulatory conditions of the uvea is caused by the arrangement of the veins as they leave the eye. The vortex veins have to carry off out of the eye almost all the blood of the uvea, and owing to the impossibility of speedy compensation, occlusion of these veins leads to serious disturbances of circulation and to increase of tension. The vortex veins are not numerous in comparison with the abundant network of veins in the uvea, and in the region of the equator are unfavorably placed, in that the blood from the posterior half of the uvea must enter them by a retrograde course (i. e., from behind forward). Furthermore, the very oblique course of the vortex veins through the sclera in comparatively narrow and indistensible canals may lead to interference with the discharge of blood from the eye

72. Consequences of Non-vascularity.—The absence of vessels in the refractive media is necessary for optical reasons, and yet entails consequences which may sometimes be disastrous.

Under normal conditions the nutrition of the cornea is effected satisfactorily even without vessels, but under pathological conditions greater demands are made upon tissue metamorphosis. When, after a lesion of the surface epithelium bacteria invade the tissues of our body, the latter defends itself against the invaders by means of bactericidal and antitoxic substances, which to a certain extent are already present in the blood, and by means of leucocytes which, under the form of phagocytes, take up the bacteria and digest them. When the invasion of bacteria occurs in vascularized tissue (e. g., the cutis), capillary loops lie everywhere very close at hand, from which the blood can act upon the germs at once in the manner above described. Certainly it is in this way that the vast majority of infections in our body are nipped in the bud. It is otherwise in the non-vascular cornea. This normally contains *no protective substances* in its tissue fluid. Invading germs at first multiply unhindered until they have formed so much toxin that the latter gets to the marginal vessels of the cornea and causes their dilatation. As a result of this, the protective substances of the blood and also the leucocytes pass over into the cornea, but even then they have to get back to the place where the germs are accumulated. Hence the distance from the site of the lesion to the corneal margin has to be traversed twice before any onslaught is made upon the bacteria, and in the meantime a considerable multiplication of the latter may have taken place. The distance, of course, is the longest in the case of lesions which occupy the centre of the cornea, and this accounts for the fact that the most dangerous lesions—those which lead to ulcus serpens—lie almost always in the centre of the cornea. The superficial lesions of the cornea, such as produce an ulcus serpens, certainly affect the marginal portions of the cornea more often than its centre, since the area of the former is greater; but in this case the marginal loops of vessels are so near the lesion that the germs entering the latter are speedily rendered harmless.

To the non-vascularity of the cornea is probably also attributable the fact that the latter in spite of its more solid structure is so much more often the site of *ulcers* than is the conjunctiva and that wounds of it heal slowly and are readily infected.

The non-vascularity of the cornea is in part the cause of its low *temperature*. Owing to the evaporation of the lacrimal fluid on the surface of the cornea, the latter gives off more heat exteriorly than does, for example, the dry skin. To the latter, moreover, more heat is continually supplied by the blood circulating in the capillaries, while for the non-vascular cornea this direct supply of heat is wanting. Hence when the eye is open the temperature of the cornea is scarcely 30° C, and because of the thinness of the cornea (less than 1 mm. in the centre), the low temperature continues to the deeper parts as well, amounting in the anterior chamber to but 32°, and finally in the vitreous to 36°. Closure of the lids, which stop the evaporation at the surface of the cornea and supply warm blood by means of their vessels, obviously causes the temperature to rise at once, so that when the lids are shut it is between 35° and 36° in the conjunctival sac. The low temperature of the cornea makes it possible for mold fungi to grow in it, while they

cannot flourish elsewhere in the living human body because the temperature is too high. There is a mold-fungus keratitis, although, to be sure, it is rare (see § 556).

The fact that the *vitreous* so readily succumbs to infection is doubtless also in large part attributable to its lack of vessels.

73. Nutrition of Cornea and Lens.—The nutritive fluid passing from the surrounding blood-vessels into the cornea is distributed in the latter by diffusion.

Moreover salts and, to a slight extent, albuminous substances also, may pass by diosmosis from the aqueous through the endothelium and Descemet's membrane into the parenchyma of the cornea, which absorbs them; but, owing to the fact that the aqueous is poor in dissolved matter, this process is certainly of very subordinate significance for the nutrition of the cornea. On the other hand, it is in just this way that the lens receives its nutrient material, i. e., from the aqueous and to a less extent from the vitreous, the dissolved matters from these passing by diffusion through the lens capsule. Definite preformed channels for the circulation of liquid within the lens do not exist.

While substances dissolved in the aqueous can enter the cornea, the aqueous itself cannot do so, its passage, which was formerly thought to occur by a sort of filtration, being prevented by the endothelium of the posterior corneal surface (Leber). If this is experimentally removed in the living eye at any spot, the cornea at the same spot becomes clouded because of the aqueous that gets in there. The same thing occurs under pathological conditions when the endothelium is injured, e. g., by inflammation in the cornea itself or by the deposition of inflammatory products upon the posterior corneal surface.

In quite the same way the capsular epithelium of the lens acts to prevent the entrance of the aqueous (see § 69), and the anterior epithelium of the cornea acts to prevent the entrance of the lacrimal fluid which bathes the anterior corneal surface.

Just as substances dissolved in the aqueous can pass by diosmosis forward into the cornea, so substances dissolved in the lacrimal fluid can pass backward and traversing the cornea reach the aqueous. In practice use has been made of this fact for a long time, solutions being put into the conjunctival sac when, as is the case more particularly with the mydriatics and miotics, it is desired that they shall act upon the iris. If we drop atropine into the conjunctival sac and then, as soon as the pupil has dilated, draw off the aqueous and drop this into another eye, dilatation of the pupil takes place in the latter also. This proves that the aqueous of the first eye contains atropine, and that, therefore, the atropine salt has passed from the lacrimal fluid into the aqueous by means of diosmosis. In the same way, when chemically active foreign bodies or an aggregation of bacteria are present in the anterior layers of the cornea, irritating substances pass from the cornea into the aqueous and act upon the iris, so that first hyperæmia of the latter and afterwards inflammation with exudation are set up. Both, therefore, are constant accompaniments of every violent inflammation of the cornea.

74. Nutrient Functions of Uvea.—The anterior segment of the uvea produces the aqueous and thus supplies nutrient material to the lens and to a slight extent to the cornea also (see § 73). The posterior segment effects the nutrition of the vitreous and in large part effects also the nutrition of the retina and the production of the constantly consumed visual substances.

To effect the latter objects the chorioid has a special arrangement of vessels. The large vessels, which do not allow nutrient matter to pass from them directly, are placed in the portion that is furthest from the retina, and, on the other hand, all the capillaries are united to form a single layer, which lies as close to the retina as possible.

IV. Circulation of the Lymph

75. Inflow.—With regard to the circulation of lymph in the eye, there must be considered the passage of nutritive material into the eye (inflow) and its discharge from the eye (excretion).

The inflow of the fluids of the eye takes place almost exclusively through the uvea. Besides the fluid that permeates the tissues there is found a pretty large accumulation of fluid called the aqueous in the anterior and posterior chambers of the eye, which are hence known as lymph spaces. To call the aqueous lymph, however, is incorrect, inasmuch as the aqueous in distinction from lymph, contains only traces of albumin and but few salts and furthermore does not clot when drawn from the eye by puncture of the cornea. Clotting occurs only under pathological conditions, when the aqueous at once acquires a large content of fibrin and often forms a clot even when still within the living eye.

For the production of the *aqueous* the iris and ciliary body are of moment. That the iris alone does not produce it is evident, since even in cases of absence of the iris—congenital or traumatic—the anterior chamber has its usual depth; and in such eyes, too, the aqueous when drawn off is rapidly replaced. But it does not follow from this that the iris has nothing at all to do with the production of the aqueous; a small quantity of the latter probably is derived from the anterior surface of the iris, even though this cannot be proved with certainty.

While it is generally agreed that the intra-ocular fluids are exuded from the uvea, opinions differ as to the way in which this takes place, some saying that there is a true secretion by the ciliary epithelium, others that there is a simple filtration, and others still that a combination of the two processes occurs (Elliot). According to Priestley Smith, it is likely that the aqueous proceeds mainly from the ciliary processes, the vitreous mainly from the orbiculus ciliaris.

The aqueous produced by the iris can leave the eye again by way of the ligamentum pectinatum. But that produced by the ciliary body has no direct exit. It must first pass through the pupil in order to get out through the angle of the aqueous chamber. Hence when in seclusion of the pupil the communication between the two chambers is interrupted, the liquid accumulates in the posterior chamber and pushes the iris forward at the expense of the depth of the anterior chamber, whose contents, however, can still flow off so long as the root of the iris is not jammed against the periphery of the cornea so that the ligamentum pectinatum is blocked.

In any event, since more aqueous is produced by the ciliary processes than by the anterior surface of the iris, a continuous flow of aqueous must take place from the posterior into the anterior chamber (and from here through the ligamentum pectinatum out of the eye). This flow is conceivable as taking place in two ways: either (1) the aqueous, being secreted in a uniform manner by the ciliary processes, flows quite uniformly and in an imperceptible current through the pupil; or (2) the aqueous does not flow forward through the pupil continuously, since the margin of the pupil is held against the lens by the pressure of the liquid in the anterior chamber or perhaps by the tone of the sphincter pupillæ, and is lifted away from the lens only from time to time when the pressure in the posterior chamber has slowly increased until it is somewhat higher than in the anterior chamber, the result being that the aqueous discharges itself at intervals through the pupil into the anterior chamber. In this latter case the pressure in the two chambers would be subject to differences with periodic equalization, although these differences in pressure must be slight. (See § 79.)

A continuous flow of liquid in the aqueous chamber results from the fact that the

aqueous that is in contact with the vascular iris grows warmer and hence ascends, while that in contact with the posterior surface of the cooler cornea becomes cooler and hence descends.

The poverty of the aqueous in *dissolved substances* arises from the fact that the blood plasma does not pass as such through the vessel walls, but that in this passage certain substances are kept back, first by the vessel wall itself, and also, so far as the ciliary body is concerned, by the epithelial wall of the latter. It is a matter of importance that, in common with the other substances, the protective bodies which are already naturally present in the body and which are increased in cases of disease, fail to pass from the blood into the aqueous. The want of such substances in the aqueous favors (in the same way that has been shown to hold good for the cornea) the outbreak of bacterial diseases. Thereafter, however, owing to the inflammatory irritation and the dilatation of the vessels that are associated with it, there results the passage not only of an increased amount of albumin and fibrin, but also of the above-mentioned protective bodies, into the aqueous, and thus the inflammation produces a coincident change in the conditions of secretion which is calculated to combat the harmful agencies.

With respect to the *quantity* of aqueous that is secreted in a given time, formerly very exaggerated ideas were entertained. Observers allowed themselves to be led astray by the fact that after the aqueous is drawn off the anterior chamber fills full again in about six minutes. But it has been shown that the newly accumulated aqueous is essentially different from the normal; that, in fact, it is very rich in albumin and fibrin. By the paracentesis the ocular tension is suddenly very much reduced; the blood flows in greater quantity than usual into the vessels, since they have been relieved of the pressure, and distends them, so that a liquid rich in albumin can pass through their walls. The stormy course of this process is proved by the changes found by Greef in the ciliary body, whose epithelium soon after the paracentesis is in places detached in the form of vesicles by the liquid which flows abundantly from the vessels. After paracentesis, moreover, just as in the case of inflammation, the protective bodies present in the blood pass over into the aqueous. We have, therefore, in the withdrawal of the aqueous a means of transferring these bodies into the aqueous, a fact by which is explained the therapeutic effect of paracentesis in many cases.

The rapid accumulation of aqueous after it has been drawn off is, therefore, favored by the artificial change of the pressure relations in the interior of the eye; but it would be a mistake to draw from this a conclusion as to the physiological secretion of aqueous. This certainly takes place with comparative slowness. According to Leber's observations, it takes three-quarters of an hour or perhaps longer for the aqueous to be completely renewed. According to others, the aqueous is renewed even more slowly and perhaps, indeed, is scarcely renewed at all (Hamburger).

76. Outflow.—The outflow of liquids from the eye must, obviously, so far as its quantity is concerned, be precisely equal to the inflow, as long as the intra-ocular pressure remains the same. As regards the path of the outflow, it must be premised that, both in the eyeball itself and in the orbit, lymph vessels are wanting, the conjunctiva of the eyeball alone possessing them. Hence the outflow, so far as it occurs in preformed channels at all, can take place only by means of lymph spaces.

The lymph spaces of the eye are in part recognizable anatomically, when for example they are lined by endothelium, which, however, is not the case for all tissue spaces in which lymph can flow. In that case injection with chemical substances or with colored solutions serves to demonstrate the presence of lymph passages. Thus by an injection of a solution of Prussian blue into the anterior chamber, it has been found that after a short time a blue coloration can be traced through the ligamentum pectinatum into Schlemm's canal, and from there into the ciliary veins that anastomose with it (Schwalbe, Leber).

77. Anterior Lymph Passages.—The lymph passages of the eye (Fig. 57) are distinguished into anterior and posterior.

The *anterior lymph passages* carry out the aqueous. This leaves the anterior chamber through the ligamentum pectinatum. The latter by the superposition of numerous fenestrated lamellæ forms a fine filter (Figs. 24 and 25) which does not transmit larger corpuscular elements, such as, for example, the red blood corpuscles, but only liquids and minute particles. According to the prevalent view these latter pass from here into Schlemm's

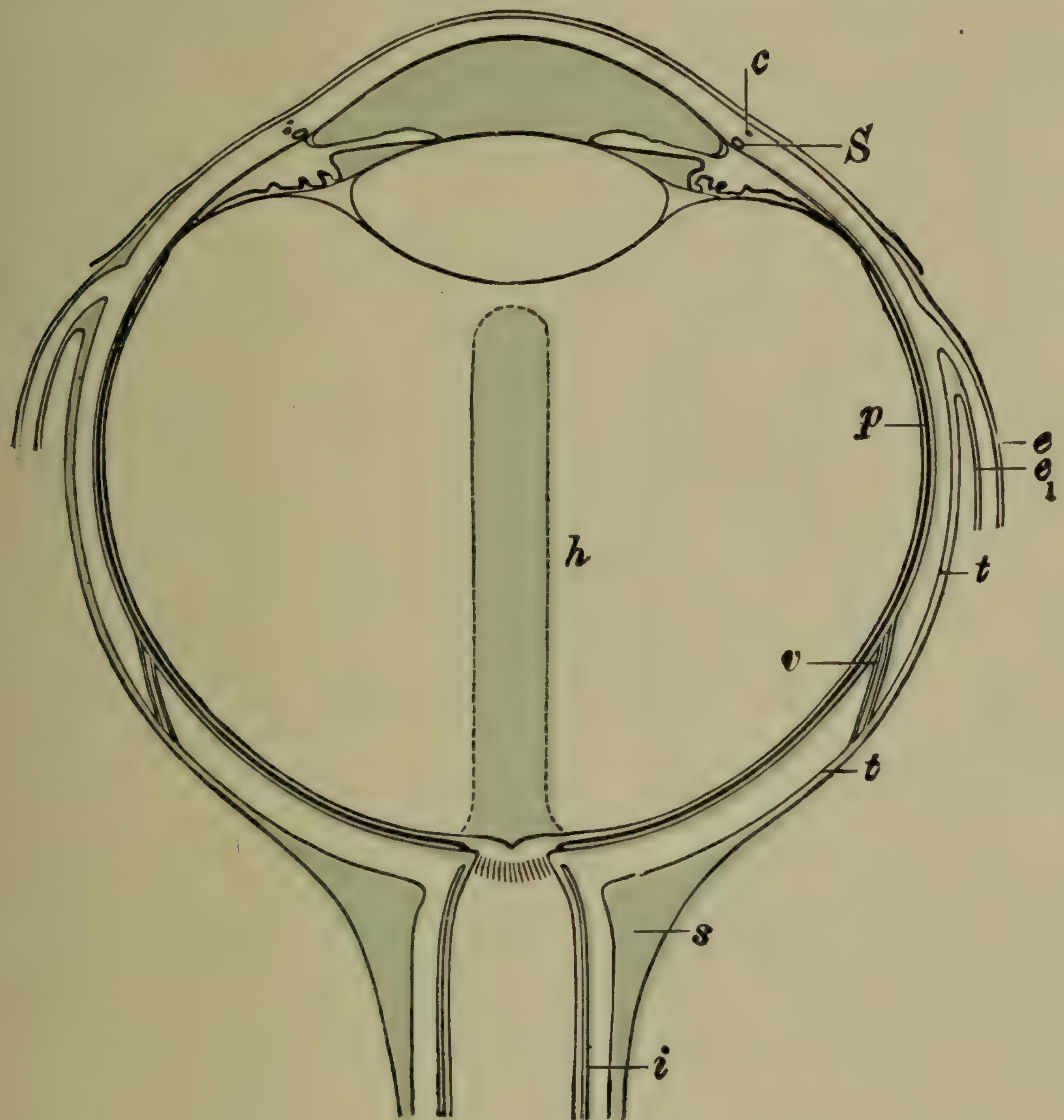


FIG. 57.—LYMPH PASSAGES OF THE EYE. (SCHEMATIC.)

S, Schlemm's canal; *c*, anterior ciliary veins; *h*, hyaloid canal; *p*, perichorioidal space, which communicates by means of the venæ vorticosæ, *v*, with Tenon's space, *t*, *t*; *s*, supravaginal space; *i*, intervaginal space; *ee*1, continuation of Tenon's capsule with the tendons of the ocular muscles (lateral invagination).

canal. Through the latter, therefore, and the ciliary veins that anastomose with it, by far the greatest part of the lymph leaves the eye.[1]

Since the ligamentum pectinatum is the main channel of outflow for the eye fluids, very serious troubles are produced by its occlusion. The fluid is retained in the eye, and increase of tension develops which leads to blindness. Injury to the filter of the ligamentum pectinatum may occur in general through the following different processes: (1) occlusion of the minute openings by corpuscular elements which stick fast in them or by clots; (2) transformation of the delicate network into a tough and impermeable tissue

[1] According to A. Thomson, the ciliary and iris muscles by their traction on the scleral spur (see Fig. 24) open Schlemm's canal and the spaces of the ligamentum pectinatum, and so pump the aqueous out of the anterior chamber.

by the remains of embryonic tissue or by inflammation; and finally (3) as a gross process the apposition of tissue, usually the iris, which is applied to the inner surface of the ligamentum pectinatum and thus cuts it off from the cavity of the anterior chamber (see §§ 841, 842, 849). These changes, which form the basis of increase of tension in the human body, can also in part be produced experimentally, and thus an artificial glaucoma can be set up.

The *iris* must be considered as a subsidiary channel of outflow from the anterior chamber. The injection of india ink in a state of minutest subdivision into the anterior chamber shows that the particles of ink penetrate into the anterior layers of the iris, where they are then taken up by the blood-vessels and carried off. Penetration into the iris takes place chiefly through the openings on its anterior surface, namely, the crypts, through which the tissue of the iris is in free communication with the cavity of the anterior chamber. Several clinical observations are explained by this fact. When, for example, after an iridectomy the anterior chamber is filled with blood, this usually disappears rapidly wherever it lies upon the iris, which in fact takes up the blood, while it stays for a long time upon the capsule of the lens in the area occupied by the pupil and the coloboma.

The part played by the iris in carrying off the aqueous is usually regarded as insignificant, yet according to some the iris is just the part by which most of the aqueous is taken up, and the latter, moreover, is not discharged into Schlemm's canal, but by way of the lymph sheaths of the anterior ciliary veins. This view seems less probable.

78. Posterior Lymph Passages.—As usually described, there are: (1) The perichorioidal space (Fig. 1, *p*), i.e., the space between the chorioid and sclera in which lie the lax lamellæ of the lamina suprachorioidea. This space is continued outward along the vessels that pass through the sclera (walls of the emissary canals, see § 38) and thus communicates with (2) Tenon's space (*t*), which lies between the sclera and Tenon's capsule. From this the lymph reaches the spaces which run along the optic nerve, namely, (3) the supravaginal space on the outer side of the dural sheath (*s*), and (4) the intervaginal space (*i*) between the sheaths of the optic nerve.

According to some, Tenon's space is not a lymph space at all.

Besides these there is assumed to be still another lymph passage in the posterior portion of the eye, which corresponds to the canalis hyaloideus or central canal of the vitreous (*h*) and which in the embryo lodges the hyaloid artery. Pathological processes argue the existence of such a lymph passage which runs in the vitreous straight back to the head of the optic nerve, for even in slight inflammations of the anterior portion of the eye the papilla of the optic nerve is found to be implicated, even when the posterior portion of the eye otherwise is still normal, so that we must assume that irritant substances from the focus of inflammation can get to the optic nerve first by a direct channel through the vitreous.

In any case, the posterior lymph passages carry only a very small fraction of the lymph out of the eye. Whether there are diseases which are referable to interference with this discharge of lymph posteriorly is uncertain.

V. Intra-Ocular Pressure

79. Pressure in Different Parts of Eye.—The intra-ocular pressure is intimately connected with the lymph circulation. Normally it is very nearly the same in the three cavities of the eye, namely, the anterior and posterior chambers and the vitreous.

In the posterior chamber it must be a little higher than in the anterior, in order to ensure the flow of liquid from the former into the latter (Elliot); but the difference in any case is but slight (cf. § 75). Any great differences in pressure between the cavity of the two chambers and the vitreous are impossible, even though a diaphragm—the lens and

the zonula—is interposed between the two, for this diaphragm can be displaced forward or backward too easily for such differences to occur, and, moreover, it is not perfectly impermeable to liquid.

Considerable differences of pressure between the separate cavities of the eye can occur only under pathological conditions. For example, it may occur in the region of the anterior and posterior chambers in seclusion of the pupil, when the pressure in the posterior chamber rises high above that in the anterior. Quite considerable differences in pressure between the cavity of the two chambers and the vitreous may occur when the diaphragm that separates the two is rendered unyielding and impermeable by cyclitic membranes.

So far as normal conditions are concerned, we may for the purpose of simplifying the study of the conditions of pressure leave the lens out of consideration, and think of the eyeball as a capsule which comprises a single cavity filled with fluid. The fluid contained in the capsule exerts upon the inner surface of the latter a pressure which, in accordance with the laws of hydrostatics,[2] is transmitted with the same intensity in every direction, and hence presses with the same weight upon every unit of surface of the wall. A square millimetre of the posterior surface of the cornea has, therefore, the same pressure to bear as a square millimetre of any portion of the sclera.

80. Factors Determining Pressure.—The intensity of the intra-ocular pressure depends upon the relation between the capacity of the capsule and the amount of its contents. If the former becomes smaller, or the latter greater, the pressure rises, and vice versa. The capacity of the capsule of the eye is dependent on the area and shape of the cornea and sclera and on the elasticity of these membranes. Under physiological conditions, it undergoes variations which though small cannot be altogether neglected. But the main variations in intra-ocular pressure are referable (1) to changes in the amount of *matter contained in the eyeball*, which may be increased or diminished. For example, the pressure at once sinks considerably when the aqueous has been discharged by puncture of the cornea.

Those portions of the contents of the eyeball whose amount is readily variable are the aqueous, the vitreous, and most of all the blood that circulates in the vessels of the inner tunics of the eye. Every increase or decrease of *blood pressure* in these vessels must result in a corresponding change in the ocular tension. It may be stated as a general proposition that the blood pressure in the vessels of the interior of the eye must always be a little higher than the intra-ocular pressure, because otherwise the arterial blood could not flow into the eye. When the blood pressure ceases, for example, at death, the tension of the eye drops at once. Owing to the small size of the vessels of the eye, the pulsatory variations of the blood pressure are so inconsiderable that the variations of the intra-ocular pressure which correspond to them are in large part compensated for by the elasticity of the fibrous coats of the eye. But even great and lasting changes in the blood pressure can affect the pressure in the eye for a moment only, and not permanently, because of the self-regulating action to be described later.

Under physiological conditions, variations in pressure can also be pro-

[2] As Elliot shows, the question is one of hydrodynamics rather than hydrostatics. In other words, we are dealing with a liquid mass in motion and not at rest. Nor is the eyeball a closed capsule, but one within which the pressure must vary, though under normal conditions very slightly, at different points. This fact does not invalidate the essential accuracy of the statements in the text.

duced by (2) the action of *external stress*. Every pressure exerted upon the eye from without increases the intra-ocular pressure by its own amount.

Hence, if with the finger we make gradually increasing pressure on the eyeball, and at the same time examine the eye with the ophthalmoscope, we see the retinal arteries become empty as soon as the pressure has reached a certain amount. For, owing to the external stress, the intra-ocular pressure has risen till it has become higher than the blood pressure in the central artery of the optic nerve, so that now both the latter and its branches are compressed. Under normal conditions the eye often undergoes pressure due to contraction of the eye muscles, and especially of the orbicularis palpebrarum when the lids are squeezed together. This, we may say, receives ocular demonstration in cases in which there is a thin place in the coats of the eye, for example, the thin floor of an ulcer or, better yet, a prolapse of the iris. Such a place, like a Marey's tambour, indicates by its protrusion and recession the variations of the intra-ocular pressure. In such cases we can see very clearly how in crying or coughing, or how when the lids are squeezed together, the thin spot distends, and under certain conditions actually ruptures. The more prominent the eyeball is, the more powerfully can the recti muscles, and especially the orbicularis, press on it, and hence cataract operations, for example, are particularly dangerous in very protruding eyes, because in them vitreous can be expelled from the wound very readily by pressure on the part of the patient.

Since external pressure on the eye produces increased outflow, the eye when the *pressure is released* is less full and hence softer than before. Hence, external pressure produced, e. g., by massage or a pressure bandage, temporarily reduces the intra-ocular pressure—according to Gradle by as much as 8-9 mm. of mercury. When the outflow is impeded, as in eyes that are glaucomatous or predisposed to glaucoma, this effect is less marked.

The pressure may also be affected by (3) *medicinal agents*.

The miotics, as is well known, are employed in order to diminish the tension in glaucomatous eyes, while the mydriatics, on the other hand, can set up a glaucomatous increase of tension in eyes predisposed to glaucoma. Even in normal eyes eserine or pilocarpine in ½-per-cent solution reduces the intra-ocular pressure by 3 to 8 mm. of mercury, the effect lasting from one to two hours or longer. Atropine has no effect on normal eyes. Cocaine diminishes the pressure from 1 to 4 mm. in normal eyes and still more in eyes that are hypotonic, while in glaucomatous eyes it acts like other mydriatics and increases the pressure. Epinephrine (adrenaline) sometimes occasions a rise in pressure, but if combined with cocaine is said to produce a decided fall (Shahan and Post). In glaucoma it quite often dilates the pupil and yet does not as a rule increase the tension (Knapp). Subconjunctival injections increase the tension, the effect lasting some hours (Lübs).

81. Regulation of Pressure.—Although persistent variations of the blood pressure and contractions of the muscles surrounding the eye influence the ocular pressure, the latter, nevertheless, except for minimal variations, remains constant. That is, a process of *self regulation* of the pressure occurs, inasmuch as the outflow of fluid through the lymph passages changes with the changing pressure. The rapidity of filtration of the aqueous of the eye depends, as in the case of any filter, upon the difference of pressure on the two sides of the filter. When, therefore, either from external stress or internal accumulation, the pressure in the interior of the eye rises, more aqueous runs out, and vice versa. Moreover, the ocular pressure is also regulated, although, to be sure, more gradually, by the fact that it itself affects the fulness of the vessels in the interior of the eye, and thus indirectly affects secretion. When the ocular pressure rises, less blood flows into the

interior of the eye, and because the vessels are less full the discharge of fluid into the interior of the eye diminishes. The reverse takes place when there is diminution of pressure. Such diminution is particularly great after escape of the aqueous. The direct consequence of this is a marked dilatation of the vessels in the interior of the eye, because the blood pressure in the latter is no longer supported in part by the intra-ocular pressure, but is supported simply by the vessel wall, which is not strong enough to withstand it and hence distends. Owing to the dilatation of the vessels the secretion of fluid in the eye increases so that the normal pressure is soon restored.

This self regulation of the ocular pressure explains why an eye in which the lens is absent is no softer, inasmuch as the absent lens is replaced by aqueous, and why, on the other hand, when the volume of the lens increases (cataracta intumescens), no increase of pressure occurs, inasmuch as a correspondingly less amount of aqueous is present in the eye.

Another factor besides the circulation of blood and of ocular fluids in the interior of the eye, that is of significance for the maintenance of the intra-ocular pressure, is found in the osmotic processes. If the salt content and, consequently, the osmotic pressure of the blood increase (e. g., by the injection of salt or by the injection of salt solution into a vein), water is withdrawn from the eye into the blood, and the eye, consequently, becomes softer (Hertel). The increase in tension which we get, if we inject dilute acids into Tenon's space (Fischer), is produced by swelling of the sclera.

A certain regulating influence upon the ocular pressure belongs also to the nerves, the trigeminus and the sympathetic. Irritation of these nerves increases the ocular pressure; section of them decreases it. Hence extirpation of the cervical ganglion of the sympathetic has been recommended as a remedy for increase of tension, but has not proved efficacious, because the diminution in tension following division of the sympathetic is but transient. Many cases of sudden diminution in tension which are not otherwise explainable (for example, those occurring after contusions, erosions, and inflammation of the cornea), are referable to the influence of the nerves.

82. Measurement of Pressure.—The intra-ocular pressure can be measured directly only by plunging a fine cannula into the interior of the eye and putting it in communication with a manometer. In practice, this method of measurement is not applicable, since it is associated with injury to the eye. If the coats of the eye are uninjured, however, we cannot test the ocular pressure, but only the *tension* to which the fibrous coat of the eye is exposed by the pressure acting upon it. For the methods of testing the tension of the eye and the normal values of the latter, see § 394.

CHAPTER IX

GENERAL ETIOLOGY OF EYE DISEASES

83. Since in the special part of the work the diseases of the eye will be discussed according to their site and the anatomical changes that they set up, here, in their general consideration, the *etiology* will be taken as a starting point. The injurious influences which lead to morbid changes are in a very general way either physical, mechanical, chemical, or parasitic. Their effects, to be sure, cannot by any means be sharply differentiated in any individual case, and in most cases several of these injurious influences act in combination. Finally, as additional causes of disease should be considered degenerative processes and a congenital faulty constitution, both of which conditions in the last analysis are referable to the injurious influences first mentioned without its being precisely known in what way these latter act.

I. Physical Sources of Injury

84. Radiant Energy.—The spectrum of any source of light along with the visible rays contains also those which are invisible. Adjoining the red end of the spectrum with its long wave-lengths are rays of still greater wave-length, the infra-red or heat rays. Beyond the violet end with its short waves lie the ultra-violet rays of still shorter wave-length, which are chemically active. But visible waves of the spectrum also have both a thermic and a chemical action. When, therefore, irritation or inflammation of a tissue develops as a result of intense radiant energy, the injurious effect may depend either on the action of light, in the narrower sense of the word, or on a thermic or a chemical action, or on all three together. This can be determined in many cases by experiment, by passing light through special filters which transmit only one variety of rays. Heat rays do, but the luminous or ultra-violet rays do not pass without hindrance through a blacked plate of rock salt. On the other hand, heat rays are absorbed by water, especially by a solution of alum, and hence are not transmitted. The ultra-violet rays pass through rock crystal, but are kept back even by ordinary glass and to a still greater degree by a glass that is colored red or yellow, and also by fluorescent substances (e. g., a solution of quinine) and by certain hydrocarbons. Thus we are able to study independently of each other the action of rays of long and short wave-lengths upon the skin or on the eye.

85. Effects of Heat.—The injurious effect of great heat in causing burns has always been known. To the higher degrees of radiant heat are exposed particularly those who work by a fire, for example, workmen at steel forges. The skin protects itself against radiant heat by abundant sweating. The evaporation associated with this withdraws heat from the

skin, and the same is true of the constantly moist surface of the eyeball, so that usually no injury of the superficial portions of the eye occurs. How is it with the deeper parts? Through the refractive media of the eye the heat rays are refracted just like light rays, and are united into a focus situated on the retina, and one would think that they might develop there a considerable heat effect, just as we can by means of a strong lens set fire to a match held at its focus. But, the media of the eye which contains a large amount of water absorb the heat rays to such an extent that they scarcely reach the retina at all. It is true that a large proportion of them—25 per cent according to Vogt—do pass through the cornea and sclera and reach the aqueous and superficial portions of the vitreous. Even so, they seem to cause trouble only when their action is very intense and is prolonged over a considerable period of time. Indeed, the only injury to the interior of the eye which has with certainty been traced to radiant heat (infra-red rays) is glass-blowers' cataract (see § 655).

Some, however, believe that other forms of cataract are due to nutritional changes set up by the action of the infra-red rays and that a large part of the irritations due to excessive light is produced by the same cause.

86. Effect of Visible Rays.—The visible rays, when they act too strongly, cause mainly a troublesome sensation of dazzling which produces blepharospasm and when it is of high degree is associated with actual pain in the eye. This pain due to dazzling cannot emanate from the retina because the latter has no sensory nerves. These are present only in the anterior portions of the middle coat of the eye, i. e., in the ciliary body and the iris, which are both rich in such nerves. The ciliary body itself is not impinged upon by the light directly, and yet it is possible that it is affected by light laterally diffused in the lens and that because of this or because of reflex irritation the ciliary muscle undergoes spasmodic contraction. Of a much greater moment doubtless is the effect on the iris, which contracts very energetically whenever the light acts strongly, so that the pain due to dazzling, like the pain that follows the instillation of eserine, is accounted for by the traction upon the nerves of the iris. Some of the discomfort produced by excessive and ill-directed light results from conjunctival irritation (causing "sandiness" of the lids) and possibly, too, from fatigue of the extra-ocular muscles (Ferree). Probably part of the discomfort, particularly the conjunctival and probably the ciliary irritation, is due to the ultra-violet and the infra-red rays (see §§ 85 and 87).

Differing from the pain of dazzling, is *dazzling* itself, which is appreciated as a disturbance of sight.

It occurs when either too great an amount of light or too much diffuse light falls upon the retina. Excessive diffusion occurs either because there is an opacity in the refracting media of the eye or because the light is too intense. In the former case the disturbance of vision that takes place is explained as follows: In the normal eye the images of the objects in the visual field lie upon the retina, side by side and sharply separated from each other, the bright and dark parts in contrast. Now, if by means of a spot of opacity upon the cornea light is diffused uniformly over the whole retina, the distinction between the light and dark portions of the retinal images becomes less striking. The following comparison may illustrate these conditions: In a well-taken

photograph all details are seen sharply and distinctly. But if it is rather highly glazed, and we look at it obliquely, the glazing shines so that the details of the photograph can no longer be distinguished. As the glazing is transparent, the rays emanating from the photograph still arrive at our retina and produce there sharp images of the details of the photograph. But in addition there come numerous rays reflected from the surface of the glazing which so flood the whole retina with light that sharp retinal images are, so to speak, drowned out.

Vision is reduced for the same reason, and a sensation of dazzling develops when the illumination is much too intense. How is the diffusion of light effected in this case? The normal cornea is not, as is ordinarily assumed, absolutely transparent. We can see this from the fact that a portion of the cornea, which has light concentrated upon it by focal illumination, looks gray, insomuch that the tyro might suppose that there was a pathological opacity of the cornea. The cornea, therefore, always reflects a certain quantity of light. The like is true of the lens, and, generally of all the refracting media of the eye. Because of this imperfect transparency of the refracting media, light diffusion takes place even in the normal eye, although, to be sure, under ordinary circumstances, it is too inconsiderable to excite notice. But when the illumination is very intense (or the pupil is very dilated) diffusion is disturbing because then an unusual amount of light diffuses over the retina. Moreover, if the illumination is very strong (or the eye lacks pigment) light also enters the interior of the eye through the non-transparent membranes (sclera and uvea) outside of the pupil, and adds to the dazzling.

Light that is too intense causes disturbance of sight not only by producing diffusion but also because the portions of the retina upon which it impinges become temporarily undersensitive, owing to the great consumption of visual substances. Everybody knows the troublesome after images which one gets so readily in looking at a sunset, and which cause disturbance in vision for some time. From prolonged looking at the sun, or other powerful sources of light (for example, electric light), a permanent disturbance of sight may result, there remaining for all time, at a point corresponding to the spot where the image of the sun was cast upon the retina (that is, as a rule, at the macula lutea), an area of indistinct vision, i. e., a central scotoma (scotoma helioplegicum), so that minute vision, such for example as is required in reading, is permanently impaired. In some cases a more considerable blindness is produced. In serious cases of this kind, moreover, we find with the ophthalmoscope pigment changes in the region of the macula lutea, proving that actual pathological alterations of tissue were set up (§ 789). After every eclipse of the sun, numerous cases of this sort are observed among persons who watched the eclipse with glasses which were not sufficiently smoked (*eclipse blindness*). As the infra-red and the ultra-violet rays are for the most part absorbed by the media of the eye, the injury here must be attributed mainly to the effect of the visible rays. Since under normal conditions these rays excite movement of the retinal cones and the retinal pigment, it can be understood how an increase of this process above a certain point may lead to pathological changes.

Another consequence of dazzling is *erythropsia* (see § 257).

Ocular discomfort, fatigue and drowsiness result from using the eyes by *insufficient illumination.*

87. Effect of Ultra-violet Rays.—The ultra-violet rays in sunlight are to a considerable extent absorbed by the atmosphere. Hence we find them in sunlight in greater amount the higher we are above the sea. The richness in ultra-violet rays of artificial sources of light varies. Poorest in this regard are the candle and the oil lamp, then follow in order the different kinds of Welsbach light, acetylene gas, the electric incandescent lights (the more so the more modern they are, i. e., the more light efficiency they have with the same strength of current), the arc lights, and lastly, the mercury-vapor lamp. The ultra-violet rays, if they enter the eye, are in large part absorbed by the refractive media of the latter. The lens, indeed, not only absorbs a portion

of the rays, but also transforms another portion into rays of greater wave-length—visible rays—so that the lens itself emits a grayish-green light (fluorescence of the lens). Hence, aphakic eyes receive more ultra-violet rays upon the retina than do those which contain a lens; consequently they see the spectrum prolonged at its violet end, and they are easily affected with erythropsia.

Since the lens absorbs or transforms so much ultra-violet light, it would be quite conceivable that it should be injured by rays of this sort, and Hess has actually been able to produce slight *opacities of the lens* by great and long-continued exposure of the eye to ultra-violet light. The *retina*, too, can be injured by ultra-violet light, but in man the injuries thus produced are usually transient even in aphakic eyes.

The opacities produced in the lens by the direct action of ultra-violet light differ in site and appearances from *ordinary cataract*. The latter, according to Van Der Hoeve, is produced by an indirect action of the ultra-violet rays, which are diffused laterally in the lens and so reach the ciliary body where they set up lesions that cause altered secretion and thus ultimately impaired nutrition in the lens (see § 655). According to this same author, cases in which no such diffusion takes place, so that the ultra-violet rays pass through the lens and are free to exert an injurious effect on the retina are especially apt to develop senile degenerative changes in the macular region (see § 743), while cases in which cataract develops are almost immune from changes of this sort.

On the exterior parts of the eye, the skin of the lids and the conjunctiva, ultra-violet light exerts a powerful action. It has been proved by experiment (Widmark) that what has been so long known as sunburn, i. e., the erythema of the skin which one gets from the prolonged action of the sunlight, is produced not by the heat rays nor the visible rays, but by the ultra-violet rays (according to Verhoeff and Bell, by the action of these combined with that of the visible rays). In travelling over the glaciers one is especially apt to get sunburn (called glacier-burn) because, at great elevations above the sea, the sunlight is richer in ultra-violet rays, and this, moreover, is particularly the case with light that is reflected from the snow. When glacier-burn affects the eyes it is called *snow blindness*, because those who are affected by it are scarcely able to open their swollen and inflamed eyes. The same symptoms may be excited by intense electric light (in working with the flaming arc light, or with the arc produced by a short circuit, etc.), when it bears the name of *ophthalmia electrica*. A less intense degree of acute conjunctivitis is produced by a few minutes' exposure to the intense light of the arc used in electric welding (Collis). These various conditions of irritation, which are called by Parsons collectively *photophthalmia*, have this feature in common, that the patient feels nothing at first, and the inflammatory symptoms begin only after a latent period of from half a day to a day. These symptoms consist of swelling and redness of the lids and conjunctiva, and sometimes also of erosions of the cornea and contraction of the pupil, caused by the irritation. Dangerous as the condition appears to the laity, on account of the violent pain and the photophobia, it nevertheless passes off in a few days. Cold compresses and the instillation of adrenaline form the best treatment.

Such violent inflammations apart, the ultra-violet rays when they

act less strongly seem also able to produce slighter changes of a chronic nature, e. g., hyperæmia of the conjunctiva and of the edges of the lids and a consequent sensation of heat and burning in the eyes. Thus it happened that many persons who previously could work well by artificial light got into trouble after the introduction of the electric light, especially one rich in ultra-violet rays.

The injurious action of light is obviated by protective glasses (see § 114).

88. Other Forms of Radiant Action.—Of the *Röntgen* and *radium* rays when there has been too prolonged action, there has been observed, not only in experiments upon animals but also upon man, injury to the eye, namely, inflammation of the exterior parts, cataractous changes in the lens, and inflammation or degeneration of the retina. The latent period in these cases is much longer than it is in the case of the ultra-violet rays, being from one to three weeks. In the therapeutic application of these rays to the adnexa of the eye, the eye itself should be protected by a shield of lead. When due precautions is observed the eye suffers no injury, being comparatively tolerant, especially of radium.

Lightning when it strikes the eye produces in it external injuries, chiefly burns and the inflammations that result from them. Moreover, in the interior of the eye there may occur opacities of the lens and atrophy of the optic nerve, but these conditions commonly do not become manifest until some time after the accident. They are produced not like the external injuries by the action of heat, but by the power possessed by the electric discharge to kill living cells even without the development of heat. Hess has proved this for the lens, showing that by electric shocks the epithelium of the lens capsule is made to die, the result being a clouding of the lens.

II. Mechanical Injuries

89. Mechanical injuries of acute onset are wounds; those that act slowly are pressure, traction, etc.

In considering the effect of injuries we should take account of one property of the eye, which belongs to it alone among all the organs of our body, and which leads to the most serious mechanical disturbances. The eye, in fact, is in large part a hollow structure filled with liquid which has no free communication externally, as the outflow of liquids takes place simply by filtration. In respect of their slight resilience, the envelopes of the eye may be compared with the capsule of the skull. Increase of tension, due to a disproportion between capacity and contents, is a frequent occurrence both in the capsule of the eyeball and in that of the skull, and the consequences are analogous in both. When in youth the capsule is still capable of expansion, it gives way and enlarges (the eye in the case of hydrophthalmus, the skull in the case of hydrocephalus), so that the contents have less to suffer from the pressure. On the other hand, such injury from pressure is the more marked the more rigid the capsule has become with advancing age (in the eye by greater rigidity of the sclera, in the skull by ossification of the sutures). Then as a result of the pressure the delicate nerve elements are the first to suffer (in the eye the optic nerve in the case of glaucoma; in the skull, the brain in the case of intracranial increase of pressure).

The lymph of the brain can flow off not only to the spinal cord but also forward in the intervaginal space of the optic nerve. Owing to the fact that in intracranial increase of tension lymph accumulates in this space under high pressure, there is afforded a mechanical source of injury, which frequently causes the blindness occurring in diseases of the brain (choked disc).

III. Chemical Sources of Injury

90. Chemical injuries are by far the most frequent cause of pathological processes. The vital phenomena going on in cells are complicated chemical processes which are affected by every alteration in the chemical composition of the liquid surrounding them. In a more extended sense of the term, parasitic diseases also act to produce chemical injury. In itself the bacterium would add as little harm to the tissues and would be tolerated with the same absence of reaction as a granule of india ink, provided that like the latter it were chemically indifferent. But by the production of toxic substances micro-organisms produce an injurious effect locally; by the diffusion of these substances they act on the neighboring parts; and by transmission of these substances through the lymph and blood-vessels they act on remote organs. By its toxic products the diphtheria bacillus sets up the local inflammation in the throat, and by the toxins that pass over into the circulation it causes the fever and the paralysis. Hence, diseases of chemical and parasitic origin cannot be sharply differentiated, and it is only from practical considerations that we will here regard as diseases of chemical origin those only in which the coincident action of microbes is, according to our present knowledge, excluded.

Chemically active substances working upon the surface of the eye produce in it corrosion or inflammation. In the same way, chemically active bodies (for example, chips of metal which penetrate into the interior of the eye) commonly produce severe inflammation.

Such inflammation is sometimes also set up by substances which in other places do not act to cause chemical irritation. For instance, atropine dropped into the conjunctival sac produces in some persons follicular catarrh or erythematous swelling of the lid. We must then suppose that there is a special individual susceptibility, which, in fact, makes itself so very evident in other parts also in the action of drugs (an example is the drug exanthems). This individual susceptibility plays a particularly great part in the case of organic poisons. It may be that some persons have in the fluids of their body substances which act to protect them against certain organic poisons and which are absent in other people. It also happens that some are unusually susceptible to a poison, because their tissues having once been affected by it are sensitized, or put into a state in which they react more readily to the poison than before. This sensitization, called *anaphylaxis,* is illustrated in the susceptibility of tuberculous subjects to doses of tuberculin, too small to affect the healthy (see § 140).

A good example of individual susceptibility and of anaphylaxis is the conjunctivitis which accompanies hay fever. This is produced by the pollen granules of certain plants which, passing through the air, get upon the conjunctiva and the nasal mucous membrane, and there cause inflammation by means of an organic toxic substance contained in them. The proof of the toxic nature of this inflammation is afforded by the fact that it is possible to prepare an antitoxin against it. But there are only a few people who are subject to hay fever, that is, who are susceptible to the corresponding toxin and whose susceptibility is actually increased as a result of having one attack of the disease. One who previously free from hay fever comes into a region where it is frequent and acquires it there, in the following years gets it again readily, even when at home. Other ocular diseases attributed to anaphylaxis are phlyctenular conjunctivitis (Verhoeff), parenchymatous keratitis (Derby), sympathetic ophthalmia (Elschnig) and endophthalmitis anaphylactica (Verhoeff).

Conversely, against other organic poisons a certain *immunity* develops in the course of time. This, for example, is true of the jequirity bean, the infusion of which when

placed upon the conjunctiva produces, as a result of the abrin contained in it, a violent conjunctivitis, which, in consonance with its toxic origin, does not set in until after a certain period of latency. Not only can we prepare an antitoxin combating the action of abrin (Roemer) but after a jequirity conjunctivitis has once occurred it is not possible to produce a conjunctivitis by a second application of the agent except after a rather long interval. The like is true of dionin.

A combined mechanical and chemical action probably lies at the bottom of those cases in which conjunctivitis is set up by small foreign bodies of a special sort (caterpillar hairs, the hairs from certain species of primula, dust from hyacinth bulbs).

In other cases, the chemical noxæ do not make their attack upon the surface, but from the blood, from which they are thrown out into the tissues that become affected. Two agents that are employed in skin disease, chrysarobin and naphthalin, are good examples of this. Chrysarobin often causes conjunctivitis, not only when it comes into contact with the conjunctiva locally but also when, starting from remote parts of the body, it undergoes absorption. Naphthalin, when ingested into the body, causes retinitis and opacity of the lens.

Chronic inflammations or degenerative processes are produced by chemical poisons more frequently than are acute inflammations (so in the case of alcohol, tobacco and many other poisons).

91. Autotoxis.—In addition to the poisons that are brought into it, the body is also harmed by those which it itself produces. A transition form between the introduced and the self-produced poisons are those which are developed in the digestive canal by abnormal processes of decomposition from nutrient materials which have been introduced there and are not in themselves toxic. By some, the cause of many diseases has been regarded as consisting in such *intestinal auto-intoxications.*

Intestinal intoxication is probably not a frequent cause of eye disease, yet it seems certain that it sometimes occasions iritis and other diseases of the uvea, including glaucoma. It is often a question, however, whether the uveal disease in this case is due to the absorption of toxic food products, produced by faulty metabolism in the intestines, or to absorption of the toxins of the bacteria which cause the faulty metabolism.

Apparently more frequent are the cases in which various structural diseases of the eye, such as keratitis, chronic iritis, cyclitis, chorioiditis, and retinal hæmorrhages, and also various functional eye disorders (asthenopia, insufficiency of accommodation and convergence) are caused by bad teeth, or by disease of the tonsils or the accessory cavities of the nose. Here, however, we have to do in most cases, not with the poisons produced by derangement of metabolism, but with bacterial toxins (endogenous infection, § 99). Better and longer known is the etiologic part played by the metabolic diseases proper, like diabetes, even though the precise way in which the general disorder of metabolism causes the local disease (e. g., the iritis or retinitis in the case of the eye) is still obscure.

Many *diseases of individual organs* lead to diseases of the eye, which not infrequently are the first thing that directs the attention of the physician to the causal affection. It is an acquisition of most recent times to have recognized that in many cases these interrelations depend upon altered chemical action. We owe this knowledge in part to the discovery of the function of the endocrine glands; disease being caused both by abnormal increase of their secretion and by its cessation. Again, examination with sera has shown how, by utilizing the roundabout passage through another animal, we can study the interchange of action in the organs of the same body. In this way it has been demonstrated that diseased organs can deposit in the blood the products of their metabolism or their decomposition which cause, almost like a regular chemical reaction, definite changes in other organs or tissues, among them the eye.

Of diseases of the eye due to perverted action of the endocrine glands the following may be cited. Upon hypersecretion of the thyroid gland depends Basedow's disease.

which produces important symptoms in the eye, and it may be that disturbance of thyroid function is one of the factors in producing glaucoma. According to Dunn, acquired hypothyroidism causes certain forms of cyclitis especially that occurring in interstitial keratitis due to hereditary syphilis. Cessation of function of the parathyroid glands is the cause of tetany, among the consequences of which are opacity of the lens (particularly lamellar cataract) and inflammation of the optic nerve. Grave disturbances of nutrition, above all acromegaly, are produced by degeneration of the hypophysis, in which case the eye suffers, although, to be sure, indirectly, owing to pressure of the enlarged gland upon the optic nerve.

According to prevalent ideas it is by a similar sort of chemical influence that the eye is implicated in disease of organs other than the endocrine glands and especially in the inflammation of the retina occurring in *nephritis*. In this condition this view would negative the old one that the connection between the two states is found in vascular changes or in the increase of blood pressure, conditions which both usually accompany chronic nephritis, or in the retention in the body of sodium chloride, due to its insufficient excretion by the kidneys. But recent researches indicate that in the retina vascular (arteriosclerotic) rather than toxic conditions are after all the primary factors in renal retinitis (Cohen). The main injury to the retina is attributed to toxic matter which is formed by the decomposition of kidney substance and which possesses a particular affinity for the tissues of the retina.

Not only may actual disease of the eye be set up by decomposition products originating in the patient's own organism, but also an excessive susceptibility to disease may be so induced (*auto-anaphylaxis*).

Diseases of the eye sometimes develop not only as a result of diseases of individual organs, but also in consequence of *physiological processes* taking place in them. As an example may be adduced the inflammation of the optic nerve which sometimes occurs in the course of pregnancy or lactation, without there being any other demonstrable disease of the body. It is conjectured that in this case also the fault lies with the action of toxins, which either have been formed as an exceptional thing in the course of the physiological processes occurring in the organs concerned, or toward which the eye does not possess its normal immunity.

IV. Parasitic Causes of Injury

92. Varieties of Infection.—Inflammation of the eye develops most frequently as a result of infections. In almost every person with a high fever we find the conjunctiva reddened and the eye glued up; i. e., we find a slight conjunctivitis. To a particularly great extent is this the case in measles. Many acute infectious diseases, however, entail for the eye consequences that are considerably more serious, namely, metastatic inflammation of the uvea and retina and inflammation of the optic nerve. The chronic infectious diseases implicate the eye even more frequently than do the acute. It is well known what a prominent part as regards eye diseases falls to the share of tuberculosis (including scrofula) and syphilis. Sometimes the eye complication follows the primary disease after a number of years, as, for example, in the case of the optic nerve atrophy in tabes, whose cause (syphilis) has apparently been cured long before.

The micro-organisms may either attack the eye itself primarily or make an entry at some other portion of the body, so that the eye is implicated secondarily. The former is denoted as ectogenous, the latter as endogenous infection.

In both cases a distinction must be made between the direct action of micro-organisms in situ and their remote effects. In the direct infections of the eye we are pretty well informed regarding these relations. In ulcus serpens, for example, the sup-

puration in the cornea is excited by the bacteria which are there present, but the associated iritis and the accumulation of pus in the anterior chamber are produced by remote action, the bacterial toxins, which are diffused through the cornea into the anterior chamber, causing the iris to become inflamed. Much less known are the relations in cases of the second kind, in which the eye simply participates in a general infection. This is especially the case in the very frequent tuberculous and syphilitic affections of the eye. In some cases of this sort the tubercle bacilli or the spirochætæ are found in the tissues of the eye, but in cases still more numerous their demonstration is not as yet feasible, so that we cannot tell whether we are dealing with a direct or a remote action of the pathogenic organism.

93. (*a*) **Ectogenous Infection.**—The exterior portions of the eye, the margins of the lids, the conjunctiva, and the cornea, can be attacked by micro-organisms directly, and these may either get a foothold upon the intact surface or may require a lesion of the epithelium in order to penetrate into the tissues and injure the latter. The interior of the eye is not accessible to ectogenous infection until traumatism or ulcerative perforation enables the micro-organisms to enter.

94. Ectogenous Infection of Lids and Conjunctiva.—The border of the *lids* presents favorable conditions for the lodgement of microbes. Between the cilia there collect the desquamated epithelium and also the secretion of the Meibomian glands, both of which in pathological cases form scales and crusts, and as an additional factor there is the moistening by tears. The border of the lid, like the skin in general, quite regularly harbors the staphylococcus albus and also the xerosis bacillus. The latter is a bacillus of the group of diphtheria bacilli, and can be distinguished from the latter with certainty only by animal experimentation, which shows whether the injected cultures have a pathological action or not. Both the staphylococcus albus and the xerosis bacillus are usually held to be non-pathogenic for the eye, but the former not infrequently does excite inflammation. In the case of inflammatory disease, such as blepharitis ulcerosa and acne pustule of the border of the lid (hordeolum), the staphylococcus aureus is also present. Moreover, being a part of the external skin, the border of the lid may participate in all diseases of the skin and may then harbor the morbific germs which characterize the latter.

The *conjunctiva* of the eyeball and the cornea form a moist surface which is exposed to the air and upon which the particles of dust and the germs which are conveyed through the air remain and adhere. Hence the conjunctival sac is almost never free from germs and, indeed, at times the most varied kinds of germs, saprophytic and pathogenic, occur there. But they do not multiply in the conjunctiva, because by the winking movement of the lids the surface of the eye is swept at regular intervals, and the tears together with the germs contained in them are carried in to the nose. This can be shown if cultures of readily demonstrable non-pathogenic bacteria (e. g., the bacterium prodigiosum) are placed in the conjunctival sac. Soon these can be demonstrated in greater and greater quantity in the nasal mucus, while they disappear at the same rate from the conjunctival sac. Furthermore, the constant slight current of tears passing down through the lacrimo-nasal duct prevents germs from ascending against the current

from the nose into the conjunctival sac. When, owing to deficient winking, the drainage of tears is arrested, the amount of germs contained in the conjunctival sac at once increases. This amount is, therefore, greater in the morning than in the evening, and is particularly apt to be increased by a bandage because the latter checks the movement of the lids. By increasing the germs prolonged bandaging of the eye suffices of itself to produce a catarrh of the conjunctiva. We see this often in the clinic when after a cataract operation the other eye, which has not been operated upon, is bandaged for one or two days, whereupon this eye often shows a catarrh of the conjunctiva which was not present before. Hence, many operators before every major eye operation apply a test bandage for a day to see whether the eye will react to this with a catarrhal irritation.

Although the most various kinds of germs fall from the air into the conjunctival sac, yet, as is the case on the border of the lids, the white staphylococci and the xerosis bacilli are the only germs found as regular inhabitants of the conjunctival sac, and they actually occur in smaller quantities in the conjunctival sac just because they are continually removed by the tears. Of bacteria usually regarded as pathogenic only the pneumococcus and the streptococcus occur with any degree of frequency on the conjunctiva (in at most 5 per cent of the cases). When, as is usually the case, the conjunctiva is not affected by these germs, they do not as a rule lead to infections of wounds on the eyeball. A healthy organism, owing to its normal protective forces is, in fact, a match for some few germs even if they are pathogenic. Hence, even in the pre-aseptic time it was comparatively seldom that one had to complain of infection in operations on the eyeball. If such an infection did set in, this usually took place, as we know from experiment, not from the germs in the conjunctival sac, but from dirty instruments. Even when the conjunctiva is not normal, infection of wounds of the eyeball is comparatively rare. This is shown by the results of cataract operations in Palestine, where, although there is scarcely an eye that is free from trachoma or conjunctivitis, post-operative infection is rare (Butler).

The *inflammations of the conjunctiva* are in very great part caused by special micro-organisms, some of which have the peculiarity of not being pathogenic either for other mucous membranes in man or for animals at all (some monkeys, perhaps, excepted). In this category belong the ordinary exciting agents of acute conjunctival catarrh, namely, the bacillus of Koch-Weeks and the diplobacillus of Morax-Axenfeld. Others are dangerous for a few other mucous membranes besides the conjunctiva and mainly for the mucous membrane of the uro-genital tract, as is the case with the gonococcus. Of germs which are generally pathogenic the pneumococcus, the micrococcus catarrhalis, and the staphylococcus aureus are fairly frequent causes of conjunctivitis, while others like the streptococci, diphtheria bacilli, etc., take a subordinate place. Two very frequent infections, specific for the eye, namely, trachoma and inclusion blennorrhœa, differ from all the others in that they can be transmitted by a filtrable virus, i. e., one that contains no demonstrable micro-organism (Axenfeld).

Besides ectogenous, endogenous infection occurs as cause of conjunctivitis (the conjunctivitis of measles has been adduced above as an example of this, and another example is the metastatic gonorrhœal conjunctivitis), but these cases are rare in comparison with those of ectogenous infection.

95. Ectogenous Infection of Lacrimal Sac.—Without there being any special infection from without, the amount of germs contained in the conjunctiva is greatly increased by the stagnation of tears, which accompanies diseases of the lacrimal sac. In the healthy sac, inasmuch as the tears do not stay in it, disease of its mucous membrane is not produced by the germs which constantly arrive with the tears from the conjunctiva. In fact, even in inflammations of the conjunctiva, the lacrimal sac is usually not infected by the germs coming from the conjunctiva. This condition changes at once when, owing to stenosis of the lacrimo-nasal duct, the tears stagnate in the lacrimal passages. Then the germs multiply, and cause a catarrhal disease of the mucous membrane of the lacrimal tract (dacryocystitis chronica). Then in the contents of the lacrimal sac there are found germs, the pneumococci being the most numerous, and after them the staphylococci with other pyogenic bacteria. Owing to the fact that the tears now stagnate in the conjunctival sac, too, the latter contains the same bacterial flora, so that wounds accidentally made become infected readily (ulcus serpens) and so, too, operations on the eye are readily followed by suppuration.

96. Ectogenous Infection of Cornea.—The *cornea*, being a tissue placed upon the surface of the body, is, just like the conjunctiva, more in danger of ectogenous than endogenous infection. From the former, develop in the first place many of the corneal ulcers which are so frequent. With regard to ectogenous infection there is an apparent contrast between the conjunctiva and the cornea, in that the bacteria which get into the conjunctival sac are able to infect the conjunctiva, even when intact, while it is assumed that for the cornea a defect of the epithelium is necessary for infection to take place. It would follow from this that the epithelium of the cornea is more resistant to germs than is the epithelium of the conjunctiva. But perhaps the difference is only apparent. For while in the conjunctival sac the germs can linger for a time, they are at once swept by the lids off the smooth surface of the cornea, and it is only when depressions are made in the latter, as a result of small defects in the surface, that the germs can remain lying in these. These epithelial defects may be caused by a slight traumatism. The cornea, just like the rest of the surface of the body, is exposed to external injuries, but it has a most delicate epithelium, which is quite soft and besides is seated on the smooth Bowman's membrane, so that it can be readily stripped off en masse. Epithelial lesions of the cornea therefore, are among the most frequent of occurrences.

The body that causes the injury may itself inoculate the cornea directly with bacteria, as is probably to be assumed for many cases of deep or actually perforating wounds of the cornea, to which suppuration of the edges of the wound succeeds. In mere epithelial lesions, a simultaneous inoculation with the micro-organism probably occurs but rarely (e. g., in the infection of the cornea by aspergillus fumigatus). Ordinarily we have to deal with a secondary invasion of bacteria into the small wound, for we find the infection generally caused by bacteria which scarcely ever occur on the

foreign bodies that cause the injury, but occur very commonly in the conjunctival sac. The most frequent of these bacteria requiring mention is the pneumococcus, which, especially in disease of the lacrimal sac, is fairly often found in the conjunctival sac, and is the usual exciting cause of ulcus serpens. Of other bacteria which occur in the diseased conjunctival sac and which then also can excite inflammation of the cornea, should be particularly mentioned the diplobacilli of Morax-Axenfeld and in Palestine and Egypt the Koch-Weeks bacillus and the extra-genital gonococcus. In isolated instances other bacteria have occasionally been found. Among them occur these which elsewhere are not pathogenic at all (e. g., the bacillus subtilis) or which have been found only in the cornea (bacillus of Zur Nedden).

We call the cases in which an inflammation of the cornea sets in as a result of a conjunctival lesion *secondary* keratitis, in contrast with *primary* keratitis, in which the cornea is the first point of attack of the injurious agent. In the cases of secondary keratitis traumatism is not required in order to produce a defect of the epithelium, but this defect may also develop because, owing to the abundant secretion in the conjunctival sac, the corneal epithelium is macerated and in places exfoliated, so that access to the corneal tissues is opened to the germs present in the tissue. Hence we particularly often find secondary corneal ulcers near the margin of the cornea. For in a severe case of conjunctivitis the swollen limbus rises steeply from the cornea, so that here a dead angle is produced, in which the secretion remains lying because it cannot be brushed away by the movement of the lids. Consequently the corneal epithelium is macerated in this spot most of all. Infection of the cornea may be produced by the exciting cause of the simultaneously present conjunctivitis, but may also be produced by any other pyogenic agent. Thus in conjunctivitis gonorrhoica and diphtheria, we commonly find in the corneal ulcers not the specific bacteria but the ordinary pyogenic germs. (For the way in which bacteria produce suppurative keratitis see § 554.)

97. Ectogenous Infection of Interior of Eye.—This as a rule takes place only when the eyeball is laid open, either by perforating injury or by a perforating ulcer of the cornea. In exceptional cases, a very thin place in the envelopes of the eye may enable the bacteria to enter, this, for example, happening in subconjunctival rupture of the sclera, in which defects of the epithelium and minute splits in the conjunctiva might form the portal of entry. Particularly dangerous are old scars of the cornea, with inclusion of the iris, when the scar is protruding and thinned. By the movement of the lids the epithelium may be injured at the most prominent portion of the scar, and the germs which enter there get not into the cornea but at once into the included iris and thus into the interior of the eye (*late infection* of the eye).

In recent injuries either the body that causes the injury carries the germs into the eye (*primary infection*), or these germs during the succeeding days, as long as the wound remains unclosed, enter from the conjunctiva (*secondary infection*). In perforating injuries primary infection is the more frequent process, in contradistinction to the slight traumatisms (erosions) of the cornea, in which, as above mentioned, secondary infection is the rule. Whether a primary or a secondary infection lies at the basis of an inflammation can in part be determined by the period at which the inflammation sets in. In a primary infection of the eye by pyogenic agents, a severe inflammation begins in twenty-four or at most forty-eight hours. A later outbreak of the inflammation argues a later onset of infection, i.e., a secondary infection.

98. Infection from Perforating Injuries.—The eye is not a single cavity but is divided by a diaphragm consisting of the lens and the zonula into an anterior and a posterior segment. As regards infection the two behave in different ways.

Perforating wounds in the *anterior division of the eye* often heal without inflammation, even when they are produced by foreign bodies that are not sterile, because the aqueous which gushes out at once effects a natural irrigation and cleansing of the wound. (Thus it is explained why minute perforating punctured wounds of the cornea, in which the aqueous does not escape, are more dangerous than large incised wounds. In the pre-aseptic time discission of a secondary cataract was in the hands of some operators a more dangerous procedure than the cataract operation itself.) Even the very frequently occurring perforation of the cornea by ulcers but rarely leads to purulent infection of the interior of the eye, because, from the moment of perforation up to the closure of the opening, aqueous continually oozes out and impedes the progress of germs in the opposite direction.

Quite different is the behavior of the *posterior division of the eye* with regard to a perforating injury. The vitreous does not flow out in any great quantity, but lies in the wound. The latter, therefore, is not irrigated—in fact, the protruding vitreous affords subsequently a convenient path for the entrance of germs. The bacteria arriving in the vitreous find here an excellent nutrient material, the more so since the natural protective substances are absent from the vitreous. Hence many bacteria which otherwise are not pathogenic for the body, i. e., which are saprophytes, excite severe inflammation when introduced into the vitreous. The bacillus subtilis (hay bacillus) is the saprophyte that oftenest causes a severe inflammation (usually a panophthalmitis).

Not only does infection develop more readily in the posterior division of the eye than in the anterior, but it has much more serious results in the former. Even a severe inflammation in the anterior segment does not necessarily lead to blindness. Its consequences, posterior synechiæ and pupillary membranes and even a secondary opacity of the lens, can in many cases be removed by operation. An infection in the vitreous cavity leads in slight cases to a plastic, in severe cases to a purulent, inflammation. In the latter case the pus may either become encapsulated (abscess of the vitreous) or break through to the outside (panophthalmitis). Even in the slightest cases, those of plastic inflammation, the eye usually becomes blind, because the exudates subsequently shrink and detach the retina from its bed.

From what has been said it can be understood that operations in the anterior segment of the eye could be made with good results even in the pre-aseptic time, while any manipulation in the vitreous cavity was scouted, because, as experience showed, it was always followed by suppuration of the eye. The aseptic method has made a transformation in this regard, but for traumatic lesions the old law still holds good.

While infection of the interior of the eye commonly affects only one of the two segments of the eyeball, this is not true of the subsequent inflammation. With regard to

this, three cases occur: 1. The inflammation remains absolutely confined to one segment. We may see in the depth of the vitreous an abscess develop about a foreign body while the anterior segment remains quite normal. To be sure, cases of this kind in ectogenous infection are very rare; in endogenous infection they occur more often. Particularly in the metastatic inflammation, which is produced by the meningococcus and which accompanies cerebrospinal meningitis, we not so very rarely see the yellow reflex of pus in the depth of the eye while the anterior segment is normal. Of course, too, the converse may occur (i. e., suppuration in the confines of the anterior chamber while the posterior division is intact), only this cannot commonly be proved, since a view into the deeper parts is prevented by the exudate. 2. In infection of one segment there develops in the other a somewhat less severe inflammation due to the action of toxins which diffuse from the primary focus of inflammation through the diaphragm into the other segment. This is the most frequent case. With infections of the vitreous cavity there is present in the anterior segment an iritis. Similarly with infection of the anterior segment there develops a non-purulent exudate in the anterior portion of the vitreous, or, in the lightest cases, there develops at least hyperæmia of the retina and optic nerve. This latter is found at times as a remote effect even in non-perforating ulcers of the cornea. 3. The agents which excite infection actually pass from one segment to the other and lead to a severe inflammation there.

Which of the above-mentioned cases occurs depends mainly on two circumstances, the virulence of the infection and the density of the diaphragm. The latter is diminished to the greatest extent in case of absence of the lens (e. g., in an eye that has been operated on for cataract) and to a less extent in the event of an injury to the lens. In cases of the latter sort, there is the additional factor that the swelling lens masses give a much better nutrient material for bacteria than does the aqueous. It has been shown by experiment that the same infection situated in the region of the anterior chamber has much more serious consequences if the capsule of the lens has previously been ruptured. It is an old experience that perforating injuries with opening of the lens capsule are more often followed by severe inflammation than are precisely similar injuries with an intact lens. For the same reason in the pre-aseptic time the operation for cataract was followed in a certain number of cases by purulent infection, while this almost never took place after an iridectomy. According to Verhoeff, this inflammation of the eye due to injury of the lens capsule may be due to a peculiar form of anaphylaxis (see § 714.)

The bacteria that enter the anterior chamber multiply in the aqueous and often also grow out into the iris. The bacteria which get into the vitreous grow abundantly in the latter, but do not ordinarily pass into the inner coats of the eye. The purulent inflammation of the latter depends, therefore, on the remote action of the bacteria present in the vitreous. The inflammation consequently affects mainly the inner surface of these membranes, i.e., the retinal coating of the ciliary body and the retina itself, on which account the name endophthalmitis is a proper one for this sort of inflammation. When bacteria are particularly abundant or virulent, necrosis of the coats of the eye develops, starting from their inner surface. Necrosis of the cornea leads to the entrance into it of leucocytes and to a demarcating inflammation with the clinical picture of a ring abscess. Necrosis of the inner coats of the eye in the posterior segment affects first the retina, then, when of great intensity, the chorioid also, and last of all the sclera, and thus paves the way for perforation of the coats of the eye in panophthalmitis.

Apart from the question of its situation (anterior chamber or vitreous) and of individual susceptibility, the severity of the inflammation depends mainly on the quantity and virulence of the invading bacteria. In the traumatic inflammations the most frequent exciting cause of purulent inflammation is the streptococcus and secondly the bacillus subtilis; in operative injuries the pneumococcus comes first and the streptococcus second.

Slighter inflammations also occur after injuries and operations. Such inflammations set in only after a rather long period of incubation, sometimes indeed not till after the lapse of weeks, and run a milder course. In place of the purulent exudate there appears a delicate plastic exudate or precipitate. However, even these cases of insidious inflammation may lead ultimately to the destruction of the eye. In exceptional instances

such mild inflammations may be produced by the ordinary pyogenic germs, while in other cases are found bacteria which are less virulent or which in other situations are not pathogenic at all. In other cases, again, it is not possible to demonstrate the presence of any bacteria whatever.

99. (*b*) **Endogenous Infection; Metastatic Ophthalmia.**—Endogenous infection consists in this, that pathogenic germs get into the blood and are carried with it into the vessels of the eye, where they settle. The nature of the inflammation depends mainly upon the nature of the germ. The pyogenic germs cause a purulent inflammation, which generally passes over into panophthalmitis. It is these inflammations that constitute metastatic ophthalmia in the narrower sense of the term.

Metastatic ophthalmia is produced either by ordinary pyogenic germs, among which the streptococci rank first and the pneumococci second, or by bacteria which are specific for certain definite diseases (e. g., the meningococcus, the pneumobaccillus, the influenza bacillus, the typhoid bacillus, etc.). Moreover, a mixed infection may also occur. The diseases caused by these bacteria, and above all puerperal fever and ordinary pyæmia, are the main cause of metastatic ophthalmia. But sometimes—as, for example, in the case of an inconsiderable attack of influenza or a very small purulent focus somewhere in the body—the original disease may develop in so mild a guise that it is overlooked, and the panophthalmitis apparently develops spontaneously (cryptogenous metastatic ophthalmia).

Although it is the rule that endogenous infection with pyogenic germs produces a purulent inflammation of the eye, yet, just as has been stated to hold good for ectogenous infection, sometimes the inflammation shows a light character, and in fact may be limited to individual foci in the chorioid or retina which go on to healing. This, indeed, seems to be even more the case in endogenous infection; germs that would cause suppuration, if injected into the eye, producing only chronic plastic inflammation when reaching the latter by endogenous paths (Selenkowsky). Examples of this are plastic iritis and similar affections caused by endogenous infection with germs of nasal, tonsillar and dental origin (see § 91) and with influenza bacilli. A mild course is comparatively frequent in cerebrospinal meningitis, and it is the rule in relapsing fever, whose exciting cause, however, the spirillum, is not one of the pyogenic germs. In relapsing fever the eye is implicated usually under the form of a benign irido-cyclitis. These cases form a transition to the second group of cases of endogenous infection, which are caused by the germs that excite chronic infectious diseases.

100. Non-Purulent Endogenous Infections.—Of *chronic infectious diseases* must be mentioned in the first place tuberculosis (lepra resembles it), syphilis, and gonorrhœa, when by entrance into the blood channels it has led to general infection; also the so-called rheumatic affections, which are probably in most instances due to infection either with the gonococcus or certain varieties of streptococcus. These diseases are complicated by eye inflammations which, in conformity with the pathogenic properties of their exciting germs, are not acute and purulent but chronic and non-purulent. The different membranes of the eye are attacked by these inflammations with different degrees of violence, and this does not depend simply on the vascularity of the membrane, for the non-vascular cornea is often affected. It is assumed that the microbes in question or their toxins possess a dif-

fering affinity for the individual membranes of the eye. By far the oftenest to suffer is the uvea, and in this the iris and ciliary body suffer the most, the chorioid less. Tuberculous, syphilitic, and gonorrhœal irido-cyclitis are frequent diseases.

The way in which the uvea participates in the general infection, whether by the presence of the bacteria themselves in the tissue or by the mere action of toxins, has been but little investigated as yet and is probably not always the same. In tuberculosis at times iritis occurs with evident tubercle nodules which without doubt are caused by the bacilli themselves; but still more often we find a chronic iritis which neither clinically nor microscopically shows tubercle nodules nor allows us to demonstrate bacilli by the microscope or by inoculation, so that we should be inclined to attribute the iritis to a simple toxin action. But the possibility is by no means excluded that in such cases the tubercle bacilli themselves, although in scanty number and in a weakened state, may be the cause of the inflammation.

The cornea becomes diseased under the guise of keratitis parenchymatosa, phlyctenular keratitis, and sclerosing keratitis. The first disease is in most cases to be attributed to hereditary syphilis, the two latter affections to tuberculosis. Here, again, it is not determined whether the disease of the cornea depends upon the direct action of the microbes which may get into the cornea from its marginal vessels or upon the action of toxins. In animals, at all events, there can be produced by the inoculation of syphilitic material an inflammation which resembles parenchymatous keratitis and in which spirochætæ can be demonstrated in the cornea. A similar keratitis can be produced experimentally, by injecting the trypanosomes, which are akin to the spirochætæ. The retina, in which the metastases that occur in the course of acute infectious diseases are most often localized, is rarely affected primarily, but very often secondarily in chronic infectious diseases.

By *endogenous infection of wounds* we understand the lodgement of microbes, which are present in the circulating blood, in those parts of the body that have been injured by traumatism. In animals an infection of this sort can be set up experimentally by injecting pyogenic germs into the blood channel and then injuring the eye; and from some observations that have been carefully made it is probable that the same process occurs at times in man.

V. Degenerative Processes

101. The line cannot be sharply drawn between simple degenerative processes and inflammatory processes that run a very chronic course. Simple degeneration has its purest embodiment in the changes which *old age* brings with it. Rather arbitrarily some of these changes are called physiological, others pathological, according as they are present in the majority or only in the minority of old people, or, even less rightly, according as they proceed without or with disturbance of the function of the eye. The pinguecula and gerontoxon are regarded as physiological changes, but senile cataract as pathological, although quite small opacities of the lens are but rarely missing in very old people.

A form of senile degeneration which is disseminated over the whole body is the hyaline degeneration of connective tissue and of the elastic fibres. In the eye this degeneration is the origin of the pinguecula and also of that thickening of the pupillary margin by which its motility is impaired. Hyaline exudations and thickenings are found in old persons in Descemet's membrane, and also in the lamina vitrea of the uvea where they sometimes become visible with the ophthalmoscope, as so-called "druses," and may even give rise to disturbance of sight, if they actually occupy the region of the macula lutea.

Another senile change at the macula is the central senile retino-chorioiditis, which occupies a mid-place between degenerative and inflammatory processes, and which, since it leads to a central scotoma, is a not infrequent cause of the impaired sight of old people. The cystic degeneration of the retina occurring in old people affects only the region of the ora serrata and does not, therefore, impair the sight.

Very often the eye becomes diseased indirectly, as a result of senile change in the vessels. Arteriosclerosis may lead, on the one hand, to rupture and, on the other hand, to narrowing or occlusion of the blood-vessels in the retina, the results of which are hæmorrhages and degenerative processes in the retina. In sclerosis of the main trunk of the ophthalmic artery or the internal carotid, the optic nerve may be so compressed by the hard vessel wall that it atrophies.

Physical influences, without exceeding their customary limits, may lead to degenerative changes, if either they act in a cumulative way for a long period of time, or if a tissue that has too little resistance is opposed to them. The former is the case with the pinguecula, which, although it is a senile phenomenon, yet develops only at that portion of the cornea which is exposed to the air; the latter is true of the zonular opacity of the cornea which develops in the region of the palpebral fissure in eyes which have suffered in nutrition, owing to some grave disease. In exceptional instances, zonular opacity of the cornea occurs as a simple senile change, in which case age constitutes the cause of the diminished nutrition of the cornea.

Poisons attack first of all the most sensitive portion of the eye, the retina, and in this again the ganglion cells which are particularly susceptible to poison. These cells are simply killed by the poison, and as a necessary consequence there is developed an ascending atrophy of the nerve-fibre layer of the retina and optic nerve. This process has been demonstrated experimentally for a series of poisons (quinine, filix mas, etc.) and by some is asserted to occur in the case of other poisons also (tobacco, alcohol, etc.).

For a series of degenerative processes we do not, up to the present time, know the cause. Only a few of these processes need be mentioned here: In the cornea, nodular and lattice-shaped opacity and keratoconus; in the lens, many forms of partial opacity; in the retina and optic nerve, retinitis pigmentosa and those changes which characterize Sachs's family amaurotic idiocy. These degenerations occur rather often with an hereditary or family distribution, so that probably they are founded on some *congenital defective structure* of the tissues, which later in the course of life becomes manifest as an actual disease. This hereditary structural defect Gowers and Collins describe as an *abiotrophy* (abiosis), i. e., a lack of vital force in a structure causing the latter to begin to degenerate and die before its time. Examples of abiotrophy are ocular paralyses of hereditary origin but developing and progressing after birth; retinitis pigmentosa and other retinal degenerations, including amaurotic family idiocy; Leber's disease; lattice-like and similar degenerations of the cornea; ordinary cataract (Collins).

VI. Congenital Defects, Heredity

102. Malformations and the Results of Fetal Disease.—We call many a thing a congenital defect of the eye without being able to offer sure proof that it actually was present at the very moment of birth. Gross changes in the eye of a new-born child, to be sure, excite the attention at once, but changes in the background of the eye are naturally not discovered until much later, on the occasion of some ophthalmoscopic examination, and even complete opacities of the lens are usually not noticed until some weeks

or months after birth. So it happens that with regard to one of the most frequent forms of cataract, the cataracta perinuclearis, we do not yet know for certain whether it is congenital or is acquired in the first years of life. One anomaly, the medullated fibres of the retina, which is called congenital, we know certainly is not so because at the time of birth the optic nerve itself possesses no medullary sheaths, these being formed first in extra-uterine life. The laity proceed in a still more inconsequent fashion, changes which develop soon after birth (e. g., those due to ophthalmia neonatorum) being often alleged to be congenital.

Congenital defects are of two kinds. One kind depends on an interference of development (malformations, in the narrower sense of the word), the other on some disease of the fetus. A sharp line of distinction between the two cannot be drawn in all cases.

The *disturbances of development* are attributable either to a defective constitution of the germ-cell itself, having its origin in the father or the mother, or to the action of the neighboring parts upon the developing germ-cell.

A good example of the first case is congenital ptosis, which sometimes is inherited through several generations. It is due to entire absence or defective development of the levator palpebræ. The same is the case with colobomata or complete absence of the iris, with congenital displacement of the lens, etc. Malformations based upon a defective germ-cell show typical forms which find their explanation in embryology.

Injurious effects of the surrounding parts on the germ-cell are shown in the case of pressure of the amnion upon the fetus, or in the presence of amniotic bands, by which many anomalies in the lids and conjunctiva are accounted for.

Fetal diseases arise either from diseases of parents (especially syphilis) or from traumatic injuries which affect the fetus while still in utero. In new-born children, we may find either the results of diseases that have already run their course—such as opacities of the cornea or staphylomata following keratitis, adhesion of the pupil or atypical colobomata following iritis, etc.—or diseases (for instance, retinitis pigmentosa) which are still recent and which undergo further development in extra-uterine life. Congenital tumors (for example, angiomata, nævi, gliomata) also occur, which grow larger in after life.

In this text-book, the malformations proper, being the really typical changes, are the conditions mainly pictured. Some of them, e. g., albinism, form but one of the symptoms of a general inhibition of development or they are at least accompanied by other congenital anomalies of the body; but generally the malformation of the eye is the only congenital defect present. Most malformations can be attributed to inhibition of development. The earlier this sets in the more marked are the consequences. Thus in inhibition occurring in the earliest stage of existence there is anophthalmus, in which case only a scarcely recognizable rudiment of the eyeball is present. In the human being after birth, we find either the actual condition set up by the inhibition of development or else its consequences. Thus, for example, the cysts, which sometimes hang upon the microphthalmic eyeball and are usually considerably larger than the latter, are an after-development due to ectasis of the insufficiently closed spot in the wall of the eye In such a way as this an organ which was originally well developed may be secondarily altered.

Many malformations are caused by the persistence of the fetal structures, which otherwise undergo retrogression even before birth (e. g., a persistent hyaloid artery, or a

persistant pupillary membrane). Hydrophthalmus is attributed to persistence of the fetal ligamentum pectinatum, so that the tissue in the angle of the anterior chamber is made more dense. This interferes with the filtration of the aqueous outward, so that there are developed increase of tension and, afterward, enlargement of the entire eye. Here, too, the conspicuous thing is not the malformation proper but its final result, the enlargement of the eyeball.

103. Heredity.—The most important cause of the malformations proper is a defective constitution of the germ-cell, which is acquired through heredity. In the wider sense of the term, heredity takes place in two ways, by transmission of a defective tendency which sooner or later develops into a malformation or a disease, or by the transmission of the disease itself. The most frequent example of the latter case (so-called *pseudo-heredity*) is the inheritance of syphilis, which takes place by the direct passage of spirochætæ from the mother to the fetus; furthermore, chemical substances (e. g., alcohol) circulating in the blood may cause the fetus to be diseased. But by *heredity in the true sense* we understand simply the transmission of a defective tendency to the descendant. The defective tendency thus transmitted may be simply an exaggerated sensitiveness to special forms of injury (hereditary anaphylaxis).

Weissmann has formulated the following hypothesis with regard to heredity in general: The chromatin substance of the nucleus of the ovum which has just been fertilized, and which consists of the combined paternal and maternal chromatin, is not completely consumed in building up a new organism by a process of continuous subdivision, but a small portion of it is stored up and remains as the germ-plasm. This, in the developed organism, is deposited in the germ-cells themselves, and from it springs the next generation (continuity of germ-plasm). When, therefore, father and son have the same hereditary defects, this arises from the same defective germ-plasm. For the first in the line of ancestors who shows the defect at all, it must be assumed that for unknown reasons the germ-plasm from which he was derived was constituted differently from that of his predecessors (primary germ variation). But when this abnormal constitution of the germ-plasm has once developed, it can, in accordance with the continuity of germ-plasm, be transmitted to all succeeding generations, as the production by animal breeders of new varieties of animals from a single abnormally formed individual proves.

Heredity is called *direct* if the defect is transmitted immediately from one generation to the next, and *indirect* or *discontinuous* (atavism) if the defect skips one or more generations. The common type of a direct heredity is that called *dominant*, in which the defect is transmitted equally to males and females and never to the descendants, immediate or remote, of a sound individual. In contrast with this, what is called *sex-linked* heredity is usually discontinuous. In this form males are predominantly affected, yet rarely transmit the defect to their children, while females, who are just the ones through whom it is usually transmitted, are rarely affected themselves. Hence a man affected with a hereditary malady of this sort would regularly have a sound father and mother and sound children, but some or all of his daughter's sons would be diseased. Altogether discontinuous is *recessive* heredity, in which several generations in succession remain exempt, and then a number of individuals, including males and females indiscriminately, are attacked in one generation.

This skipping of generations may actually go so far that the like structure is found only in our ancestors among the animals. For example, the presence of hyaline cartilage in the semilunar fold, which is extremely rare in Europeans and somewhat more frequent in the lower races of mankind, represents the cartilage in the membrana nictitans of the mammalia.

A defective tendency in one progenitor, which has thus remained latent for generations, may become manifest in the descendant, if, by chance, the other progenitor posses-

ses the same latent tendency, so that the two tendencies are added together, and thus from two healthy parents diseased descendants are produced (potentized heredity). Of course, a faulty tendency having the same trend is most apt to be found in those persons who have the same descent, for which reason degenerated children are more frequently derived from the marriage of blood relations than from other marriages. Hence in recessive heredity consanguinity of the parents has a considerable effect in developing a morbid tendency, but in dominant and sex-linked heredity it seems to have little influence.

The way in which *acquired diseases*, in the narrower sense of the word, are inherited is still more obscure than is inheritance in general. The occurrence of such inheritance is established, but in any case it is much less common than is generally assumed.

The inherited anomalies or diseases may (1) already be present at birth, e. g., in the case of ptosis, coloboma or absence of the iris, ectopia of the lens, congenital cataract. (2) They are present at birth only as a tendency and do not develop until later on, instances of this being Leber's disease (heredity optic neuritis), retinitis pigmentosa, and many kinds of cataract. According to Treacher Collins these are examples of abiotrophy (§ 101). Finally (3) the faulty disposition dependent on heredity is not necessarily a disease at all, but may be only a variety of anatomical construction which predisposes to disease when other injurious influences are added—examples of this being the yielding character in the sclera that predisposes to myopia and the smallness of the eyeball that predisposes to glaucoma.

Hereditary defects include congenital anomalies of the iris (§§ 735-740), glaucoma and buphthalmus (§§ 836, 849), cataract (§ 654), ectopia lentis (§ 666), amaurotic family idiocy (§ 795), degenerative conditions of the retina (§§ 794, 795), Leber's disease (§ 815), color blindness (§ 254), congenital ptosis (§ 362), paralyses of the eye muscles (§ 325), squint, and occasionally nystagmus (§§ 334, 356), and refractive anomalies (myopia, or rather the predisposition to myopia). Mostly in these cases the heredity is direct (dominant), but in Leber's disease, in color blindness, and sometimes in other conditions it is sex-linked. This is the case to a certain extent also in retinitis pigmentosa, in which, moreover, the heredity is more frequently recessive than direct (Leber).

Hereditary defects usually affect both eyes but may be unilateral (Lutz).

CHAPTER X

SYMPTOMATOLOGY OF EYE DISEASES

104. Altered Secretion.—The *secretion* in inflammation of the conjunctiva is either mucus or purulent, according to the intensity of the inflammation. By the drying of the secretion the lids stick together, especially over night. Only in phlyctenular conjunctivitis the secretion of mucus is little marked in comparison with the profuse secretion of tears, because this disease in its pure form affects only the conjunctiva bulbi. Secretion of tears is also associated with diseases of the eyeball itself, including the inflammations of the cornea, iris, and ciliary body. However, both these diseases and phlyctenular conjunctivitis, if they last a long time, readily lead to catarrhal inflammation of the conjunctiva and hence also to a secretion of mucus in addition to the secretion of tears.

Since many tears flow down into the nose when the secretion of tears is profuse, a patient often has to blow his nose, and hence thinks that he is suffering from a cold. At the beginning this is incorrect, but when profuse lacrimation has lasted for a long time the mucous membrane of the nose does finally get into a catarrhal state, owing to the irritation produced by the tears.

105. Glandular Swelling and Fever.—Normally, the *pre-auricular lymph gland* is not perceptible to the touch. When it can be felt through the skin this proves that there is some swelling of the gland. Such swelling is associated with eye diseases dependent upon infection of the eye. The conditions that most frequently give rise to it are, on the one hand, the phlegmonous inflammations (hordeolum, panophthalmitis, phlegmons of the orbit), and also inflammations that are particularly virulent (vaccine blepharitis, gonorrhœal and diphtheritic conjunctivitis). A marked glandular swelling is especially characteristic of Parinaud's conjunctivitis (see § 477). Among chronic infectious diseases, the tuberculous and syphilitic inflammations of the eye and its adnexa lead to swelling of the pre-auricular gland.

Fever is but rarely produced by an inflammation of the eyes—most frequently by the above named phlegmonous inflammations and by inflammations that are specially virulent.

106. Photophobia[1] is a term used to denote both the discomfort and the sense of dazzling and blinding produced by light (see § 86). The discomfort varies from a sense of burning, smarting, and sandiness of the lids or smarting of the eyeball to a severe cramping pain. It is associated with nictitation or when severe with blepharospasm. Photophobia originates both in the terminal fibres of the trigeminus and also in the retina. Most of the nerve endings of the cornea are in the epithelium, hence foreign bodies, small erosions, and superficial inflammations of the cornea are accompanied by marked photophobia, particularly if the nerve terminals

[1] From φῶς, light, and φόβος, fear.

are irritated by frequent winking. (Bandaging of the eye therefore, produces relief.) Deep ulcers of the cornea, in which the superficial nerves have been destroyed, or deep wounds of the cornea produce much less photophobia, so that we may almost say that the photophobia is often in inverse relation to the gravity of the corneal change. Inflammations of the iris, which is very rich in sensory nerves, are also associated with marked photophobia. In all these cases, the mere opening of the lids, even when the illumination is weak, suffices to evoke reflex blepharospasm.

The case is different with the photophobia that emanates from the retina. This photophobia occurs only when there is a great amount of light falling upon the eye (e. g., when one looks at the sun), but in that case affects healthy eyes also. In the case of the retina, it is not sensory fibres (since these are not present in it) but optical fibres that convey the reflex to the sphincter of the lids and of the pupil.

The grittiness and burning of the lids seem to be due to the direct action of light, especially the ultra-violet (perhaps also the infra-red) rays, on the conjunctiva; and the sense of fatigue or strain produced by excessive or misdirected light (asthenopia photogena) is probably attributable to tiring of the ciliary or extra-ocular muscles. (Specially trying is light changing rapidly in intensity or the attempt to use the eyes in a very unequally illuminated room.) Similar symptoms with a sense of drowsiness are caused by insufficient illumination.

A symptom that frequently accompanies photophobia, whether emanating from the trigeminus or the optic nerve, is reflex *sneezing*. Particularly in phlyctenular conjunctivitis with marked photophobia sneezing sets in with great regularity whenever the attempt is made to open the eyes.

107. Pain.—Pain is associated only with the inflammations of the anterior portions of the eye which possess sensory nerves; the chorioid, retina and optic nerve are incapable of exciting pain (the pain in acute retrobulbar neuritis comes not from the optic nerve itself but from its sheaths). The pain bears no definite relation to the severity of the inflammation, but in intensity apparently keeps pace with the photophobia and the secretion of tears, as it obviously should do since all three are excited by irritation of the trigeminus.

The inflammations of the conjunctiva do not produce any violent pain, but only a sensation of a foreign body or of moderate burning and pressure. Sharp pain in the course of a conjunctivitis points to an involvement of the cornea. The inflammations of the cornea and sclera cause pain which is felt chiefly in the eye itself. On the other hand, in inflammation of the uvea and in increase of tension the pain usually radiates to the neighboring parts, and chiefly to the forehead, less often to the ears or the teeth of the upper jaw. This sort of pain is known as ciliary neuralgia, and in fact sometimes assumes a neuralgic character, since it occurs in attacks, and between the attacks there are intervals in which the pain is absent, even though the condition of the eye shows no recognizable variation. Indeed, not infrequently, just as in the case of true neuralgia, the attacks of pain recur every day at the same time (especially in the evening or at some hour of the night), and are then often favorably influenced by quinine. The radiation of the pain to the neighboring parts, especially to the forehead, often causes the

patient himself to localize the starting point of the pain incorrectly, since he states that he feels no pain in the eye itself. If, then, there are no striking changes in the eye, the eye trouble itself may readily be overlooked and the morbid condition be regarded as a true neuralgia.

The kind of pain may give a valuable clew to the diagnosis. Beginners readily confound an iritis accompanied by marked injection, but without evident exudation, with a conjunctival catarrh. If the patient states that he has pain in the bones over the eye, we may be sure that it is not a simple conjunctival catarrh that is present, but an iritis.

The pain in severe inflammation of the eye often has the unpleasant character of becoming particularly violent just at night and depriving the patient of his night's rest. In irido-cyclitis, as in acute glaucoma, pains occur which are among the most severe that a man can possibly have, and which (especially in glaucoma) can actually excite symptoms of irritation of the brain, e. g., vomiting.

108. Headache.—Apart from pain which radiates from the eye to the head, actual headache may also emanate from the eyes even when the latter are not really diseased, but simply subjected to undue strain, as in ametropia. If, therefore, the patient complains of frequent headaches, the physician treating him should always think of the possibility of their originating from the eyes.

Headache emanating from the eye can easily be recognized when we find the eye *inflamed*, weeping, photophobic, or sensitive to pressure. But often the objective changes in the eye are slight; in fact, they may be actually absent at the moment of examination.

This is particularly the case in prodromal *glaucoma*. Since the prodromal attacks usually occur in the evening, the physician often chances to see the patient in his office after the attack has elapsed and finds nothing abnormal in the eye. Then a precise history must afford assistance, a history which is particularly characteristic when the prodromal stage has lasted a long time. The headaches, the patient says, come on less frequently at first; afterward more and more often all the time; in summer he is almost free from them, in winter suffers much more often. The pain usually comes on in the afternoon or evening, and ceases with sleep; the patient is never waked by it from sleep. The headache is often produced by excitement, the visit of company, or by going to the theatre, etc. In addition there is the associated disturbance of vision, consisting of some clouding of the sight and the appearance of colored rings about a light. But these disturbances are often slight and if they are limited to one eye they are readily overlooked by the patient. The examination of the eye then usually shows a strikingly shallow anterior chamber and perhaps arterial pulsation (in the retina) when even very slight pressure is made by the finger upon the eye. But as long as no glaucomatous excavation is present a certain diagnosis can be made only by having the patient visit us just when he is having an attack of headache. We shall then find in the eye the characteristic, even if slight, changes of a prodromal attack of glaucoma, and upon dropping in pilocarpine the symptoms pass off in about a quarter of an hour.

Headaches in which there is nothing to be seen in the eye externally originate in errors of *refraction* (hypermetropia and astigmatism), in anomalies of accommodation, and in disturbances of *muscular* equilibrium. Headaches of this sort occur especially after undue or prolonged exertion of the eyes. They are often relieved by sleep, although in some cases on the

contrary, they attack the patient when he first wakes in the morning, especially if he has been overusing the eyes the night before. Infrequent in very young children, they occur very often between the ages of ten and twenty, when to the stress imposed by study are added the nervous disturbances caused by developmental changes in the body and eye.

As in the case of asthenopia (see § 111), headache due to refractive or motor anomalies is often excited by looking at a series of moving objects (*panorama headache*).

Neurasthenics, too, when they strain their eyes have pain which they locate sometimes in the lids, sometimes in the eyes themselves, or behind the eyes, or in the head. The discomfort often sets in, within a few minutes, sometimes after reading a few lines, while in disorders of refraction or of the muscles, it does not generally set in until after rather prolonged exertion of the eyes.

109. Headache not Originating from the Eyes.—Headaches which by the patient are often referred to the eyes but which have another origin are migraine, the headaches occurring in disease of the frontal sinus, and supra-orbital neuralgia.

In *migraine* the pain as a rule is unilateral. It is violent, boring and situated over the eye, in the forehead and above it, but sometimes also in the eye itself. The attack is often introduced by a disturbance of vision (scintillating scotoma); during the attack the patient feels best if he can lie quietly in a darkened room; glaring light pains him; there is a sort of photophobia. All of these circumstances may cause the patient to believe that his eye is affected. In distinction from the headaches that really emanate from the eye, the migraine attack often shows a quite typical course. Beginning with or without a scintillating scotoma, it rises pretty rapidly to a considerable pitch and usually lasts for from half a day to a whole day; then nausea ensues, and frequently vomiting, whereupon the the patient feels better and the attack comes to an end. Typical, also, is the way in which the attacks recur at certain intervals. They occur usually not oftener than once or twice a month (in women, who suffer from migraine more than men do, commonly in conjunction with menstruation), and with increasing age grow less frequent and milder. In a person who suffers from migraine, a true migrainous attack can be set up by straining of the eyes, just as it can by many other injurious influences.

In empyema of the *frontal sinus*, there are also frontal headaches, but they are always on the same side, which is commonly not the case in migraine. When drainage into the nasal cavity is free, the pain is not particularly violent, but is more like a dull pressure. On the other hand, upon closure of the excretory duct and accumulation of the secretion, very violent, boring or hammering pains set in. The pain often comes in attacks, or sometimes shows a periodical increase, especially in the morning, when the secretion has accumulated during the night. Sometimes the pain increases when the head is bent forward. Local symptoms are tenderness of the upper margin of the orbit to pressure or percussion, tenderness to pressure of the supra-orbital nerve, and in acute cases a slight œdema of

the skin over the frontal sinus. In addition there is a history of frequent colds in the head or blocking of the nasal cavity on the side of the pain. A certain diagnosis, however, can be made only on the basis of a rhinological examination.

Lastly, supra-orbital *neuralgia* has, in common with the two other kinds of headache, its localization in the forehead; and the tenderness of the supra-orbital nerve to touch occurs in the disorders above mentioned, too. Its character of recurring in attacks neuralgia shares with migraine, and the unilateral character of the pain it shares with disease of the frontal sinus. During the attack the eye of the affected side often is less wide open and weeps, and is even slightly injected, so that the suspicion of there being some eye trouble might be excited. It is a characteristic sign of supra-orbital neuralgia that the pain is particularly violent and that the tenderness of the nerve, when touched during the attack, is so great that the patient draws his head back at once when this is done. The attacks set in suddenly after an interval of entire freedom from pain and sometimes are of but short duration, but recur either every day at certain hours or in irregular fashion several times a day.

Headaches of a persistent character, but with temporary exacerbations, accompany uræmia. *Nephritics*, therefore, suffer much from headaches, which are deep-seated and are boring in character. Sometimes there are vertigo and vomiting. In chronic nephritis frequent headaches are often the only thing of which the patient complains. In increase of *brain-pressure* (chronic hydrocephalus, meningitis serosa, brain tumor) the pain often has a similar character, but is still more often accompanied by vertigo and vomiting; often also there is tenderness of the skull to percussion at the point where the severest pain is situated. Hence in patients who complain of frequent headaches, the examination of the urine for albumin and of the eyes for changes at the optic-nerve entrance should never be neglected. These latter changes may last a long time without there being any interference with sight, so that it would be a mistake to believe that an ophthalmoscopic examination is superfluous if the patient sees well.

In *syphilitics* deep boring headaches occur which undergo exacerbation chiefly at night. In *gout* and in rheumatism (rheumatic nodes on the branches of the trigeminus) there is present a quickly varying and boring or tearing pain in the orbit or about it or in the forehead. *Arteriosclerosis*, general *anæmia* and its opposite, *plethora*, give rise to headache. In some cases a persistent headache occurs without any assignable cause. Such a headache, which is called *habit* headache, is found especially in young persons and then usually diminishes or disappears as the patient grows up. But it is unsafe to make the diagnosis of habit headache without making a very searching examination for errors of refraction and motility, since such errors, even when very slight may cause persistent headache, especially in young persons in whom the nervous system has not yet acquired stability.

110. Vertigo.—Vertigo is another very widespread symptom, on account of which the patients often visit the ophthalmologist with the idea that the vertigo starts from the eyes. It is advisable, therefore, to enum-

erate, just as was done with regard to headache, the different kinds and causes of vertigo, and to show how we can tell whether the vertigo is to be attributed to the eyes.

Vertigo, like headache, is a subjective symptom not to be recognized from any objective changes, so that the physician is dependent on the statements of the patient. These statements are much less precise than in the case of headache, partly because most men are not able to describe their symptoms intelligently anyhow, and partly because they are really dealing with sensations which are very difficult to define.

We find indeed, when we question patients closely that disturbances of the most varied kind are called vertigo, there being included along with true vertigo various sensations of distress and disorders of vision. The disagreeable sensations caused by sudden noises and by heights (in looking down from a height) are quite generally called dizziness, but have nothing to do with real vertigo. Of visual disorders it is mainly the transient sort that are erroneously called vertigo by the patients, who complain that they are "dizzy" when owing to hypermetropia the print swims after they have read a long time or when, owing to weakness of convergence, diplopia occurs, and the lines run into each other. Muscæ volitantes, a prodromal attack of glaucoma, scintillating scotoma, the momentary obscurations occurring in choked disc, are also often denoted as vertigo.

Vertigo in the narrower sense of the word either takes its origin from some visual derangement, or it develops independently of the latter, although even then not infrequently it is accompanied by visual derangment. The sensations which the patient has in the different forms of vertigo may on precise analysis be differentiated into the following varieties:

1. Uncertainty in movement, as in grasping at objects and still more in walking, going up stairs, etc. This derangement arises from *false localization*, the object that is grasped at or the spot on which it is desired to plant the foot being seen in the wrong place. To a very slight extent, everyone has this sensation who for the first time in his life wears glasses, when he walks about. He sees the floor in front of him raised or, on the contrary, depressed, and is afraid of falling. This phenomenon very soon passes off as the patient becomes habituated to his glasses, and only in the case of strong glasses, such as, for example, patients wear who have been operated on for cataract, does it happen that some cannot get accustomed to them. For the glass, when the patient does not look through its centre acts like a prism, and the more so the stronger it is, and, owing to prismatic deflection, the object sighted appears in the wrong place. Hence, too, the trouble is the greatest when the patient looks quite obliquely through the glass, e. g., in looking down when he goes down stairs.

To a much greater degree this same trouble is produced by false localization in paralyses of the eye muscles, so that even slight degrees of the latter may be very troublesome. An insignificant paresis of the trochlearis, which becomes manifest only in looking far down, may actually make the act of going down stairs dangerous, unless the patient himself remembers to close the paralyzed eye, whereupon the vertigo at once disappears. So too a very marked and persistent asthenopia is caused sometimes by the very slightest amount of paresis in the inferior rectus, a paresis which ordinary tests fail to reveal.

2. The *false movement* of objects produced by false localization (§ 313). It constitutes even more than the latter the cause of the vertigo occurring in paralysis of the eye muscles. This vertigo is often extremely great, but disappears at once on closure of the paralyzed eye. It is especially the peripheral paralyses that produce marked vertigo, while in paralyses of central origin the vertigo is often quite inconsiderable, and even the diplopia scarcely reaches the consciousness.

A false movement of objects occurs when the images on the retina are displaced without the patient's being conscious of a movement of his eyes corresponding to the movement of the images. In paralyses of the eye muscles this happens because, without the patient's knowing it, voluntary movements of the eye are absent; but in other cases because, conversely, movements of the eye occur which do not reach the patient's consciousness. These latter involuntary and unconscious movements occur in nystagmus and vertigo produced by turning round. In the ordinary sort of nystagmus originating in childhood, no false movement occurs, because the patient is habituated to his normal eye movements and has learned to take them into account in judging of the displacement of the images on his retina. In nystagmus that develops later—miners' nystagmus, nystagmus originating in the ear, and nystagmus in disseminated sclerosis—false movement of objects and consequently vertigo do occur. Unconscious movements of the eyes also develop in the attempt to follow objects that are continually passing by, as, for instance, when one looks from a bridge at the water flowing beneath. When in that case the gaze falls upon objects which do not alter their position with regard to the eye (the flooring of the bridge, the bank), these appear to be moving in the opposite direction. The same thing occurs when the ojects are still but the man is moving, e. g. when he is looking from a moving railway train.

3. The sensation of *disturbed equilibrium*, of falling or of being turned in a certain direction, so that by a movement of the body, which compensates for this imaginary disturbance, a fall in the opposite direction may easily result.

This sensation originates in an irritation of the nerve terminals in the semicircular canals. To the highest degree such vertigo is present in Ménière's disease; to a less extent it is produced in patients with ear trouble by syringing the ear, the use of the air douche, etc. Even in healthy ears the vestibular apparatus can be irritated and thus a vertigo be produced by whirling round, by swinging, by syringing the ear, or by galvanization. This irritation is regularly associated with nystagmic movements of the eyes (§ 356) which in themselves are calculated to set up vertigo. But the main cause of the latter lies in the irritation of the vestibular nerve, since the vertigo is evoked though to a less extent even when the eyes are closed.

4. Vertigo forms with headache and vomiting the triad which belongs with the symptoms that regularly accompany *intracranial increase of tension.* In addition, but not of quite such regular occurrence, is a fourth symptom, choked disc. Vertigo can be made available as a focal symptom only when it develops in a particularly intense way and is combined with ataxic symptoms, as is the case in involvement of certain portions of the brain, especially the cerebellum and the vermis.

5. A sensation in the head that cannot be precisely described, frequently combined with a transient feeling that everything is becoming

black before the eyes, is among the consequences of sudden changes of *circulation* in the brain, occurring thus in anæmic persons when they sit up suddenly, and occurring as a precursor of syncope and in arteriosclerosis of the arteries in the brain.

It is known that disorders of the *stomach* are sometimes associated with vertigo, which can sometimes be excited voluntarily by pressure on the epigastrium. The way in which vertigo is excited by the stomach has not yet been made clear. Caution, moreover, must rule in making the diagnosis of stomach vertigo, since any vertigo, if intense, may lead to vomiting, which should not then without further consideration be regarded as a disease of the stomach.

111. Asthenopia.—Asthenopia [2] is a sense of strain and weariness in the eyes and head, set up by the use of the eyes. The patient with asthenopia says that the eyes "give out." It may occur alone (asthenopia simplex), or be associated with pain in the eyes (asthenopia dolens), headache (asthenopia cephalalgica), or irritation, redness, and burning of the eyes or lids (asthenopia irritans). It may be accommodative, i. e., caused by refractive errors, especially hyperopia (see § 197) and astigmatism; muscular, i. e., caused by anomalies of the exterior muscles (see § 340); nervous, i. e., caused by hysteria or neurasthenia (see § 245); photogenous i. e. caused by excessive or improper illumination (see § 87); or reflex, i. e., produced by morbid conditions of other organs, especially the nose and its accessory sinuses and the teeth. In those predisposed by refractive or motor anomalies, the fatigue of the exterior and interior muscles of the eye occasioned by watching a series of moving objects, e. g., in the theatre, at a ball game, in shopping, walking in crowded streets, motoring, or riding on a train often causes asthenopia (panorama asthenopia). This may be accompanied by headache (see § 108). Asthenopia is the commonest of all eye symptoms and the one that is most readily relieved by proper treatment, which consists essentially in the removal of its cause.

112. Other Symptoms.—Other symptoms of which the eye patient may complain are:

1. *Blurred vision.* This occurs in opacity of the media or disease of the fundus of the eye and also in errors of refraction and accommodation. It may be permanent or, especially in hyperopia and beginning presbyopia may be transient the patient noticing that the print he is reading suddenly runs together and then becomes clear again. Blurred vision, especially when transient, is also caused by motor anomalies—in this case signifying that the patient sees double but that the double images are so close together that they overlap.

2. Double images (monocular or binocular *diplopia*; see §§ 171, 285 and 288).

3. Multiple vision (*polyopia*, see § 171).

4. Tendency to shut one eye in order to see clearly (*monoblepsia*). This is sometimes an evidence of great inequality of refraction (anisometropia) or other condition causing marked inequality of vision in the two

[2] From ἀσθενής, weak, and ὤψ, sight.

eyes. But commonly such conditions do not cause it, and in the great majority of cases it is due to some motor anomaly causing double vision. For these it is of considerable diagnostic significance.

5. *Metamorphopsia, micropsia,* and *macropsia* (see § 238).

6. Flashes of light before the eyes (*photopsiæ*). These are due to irritation of the retina from pressure, traction, etc. Not infrequently they indicate a beginning detachment of the retina.

7. *Chromatopsia,* (erythropsia, etc., see § 257).

8. Floating specks before the eyes (*muscæ volitantes,* see § 668).

9. *Drowsiness* or *aprosexia*; a frequent symptom in cases of poor vision, insufficiency of convergence or accommodation, and asthenopia due to reflex disturbances.

10. Slow *reaction time.*

11. *Reflex disturbances* (remote pains, nausea, tics of various kinds). These are not infrequently set up by refractive and muscular anomalies.

12. *Head-tilting* (sometimes amounting to actual torticollis). This is thought to occur in astigmatism (being then usually slight), and occurs in motor anomalies, particularly pareses of the ocular muscles (see § 316).

CHAPTER XI

GENERAL THERAPEUTICS OF THE EYE

I. Hygienic Regulations

113. General Hygienic Regulations.—These consist in the prescription of a light diet and in the case of inflammatory eye affections also in the withdrawal of alchohol. But from very old people who have been accustomed to take a little alcohol regularly, the latter should not be withdrawn, and as little ought we without further consideration to take alcohol away from actual drunkards. Such a rule applied after an operation, for example an operation for cataract, would be directly calculated to provoke an attack of delirium tremens. A good night's rest is important; if this is disturbed by severe pain, which very often sets in just at night, an anodyne must be administered in the evening. Of the ordinary remedies acetylico-salicylic acid (aspirin) has a particularly good effect in the pain caused by inflammation of the eye. With very violent pain, however, a morphine injection is the only thing that helps. As far as possible we should permit the patient to go out every day into the fresh air, but if he is very photophobic not till after sundown. He should stay in an atmosphere free from dust and smoke (avoiding restaurants filled with smoke, etc.). With any serious deep affection of the eye (inflammation, hæmorrhage, traumatism —including operations), abstention from work is required, including both heavy physical work and also any work that would strain the eyes. It is important, therefore, in these cases to keep the patient in bed, so that as small demands as possible shall be made on the general and intraocular circulation, and eye and body shall have less chance to move about. Sometimes in inflammations and usually after operations, in order to rest and protect the eyes it is necessary to use protective glasses or a bandage (see §§ 114, 116). In general, such protection must be applied to both eyes, even though but one is affected.

The patients often think that if they bandage the affected eye they can with impunity read with the sound eye. But it is not possible to exert one eye alone. Accommodation, and convergence, and the contraction of the pupils that is associated with them always take place in both eyes at the same time and to an equal extent, and in experiments upon animals the illumination of one retina suffices to cause contraction of the cones and advancement of the pigment in the non-illuminated eye as well. If, therefore, a diseased eye is to be protected from the light, it is not sufficient to bandage it alone; both eyes must receive protection.

Absolute exclusion of light by bandaging both eyes or staying in a very dark room is scarcely ever required. We have abandoned treatment in the dark, which formerly was often employed and which depressed the patients physically and mentally. The like is true of the bilateral bandage which formerly was applied for a series of days, especially after cataract operations. A not infrequent consequence of complete exclusion of light in old persons who are predisposed to dementia is mental derangement (see § 900). In a person who has been operated on for caratact the two eyes are bandaged

simply on the day of the operation, and from the first day the patient is allowed to stay —and without harm to him, either—in quite a light room (Fuchs).

114. Protective Glasses.—As stated above, when protection from light is indicated, this should be applied to both eyes. For this purpose protective glasses are used. For the visible rays we use glasses of a neutral gray color. These are indicated for healthy eyes when an excess of light has to be kept out (in travelling over snow, in the tropics, and in certain industrial pursuits).[1] They are indicated for diseased eyes, to protect them against even ordinary daylight, when there is either great sensitiveness to light (photophobia) or disease of the retina or uvea. Since the organs are stimulated by light to the performance of their function, the shutting out of light is the first condition for putting them in a state of rest.

Since all kinds of glass keep out much of the ultra-violet light, the ordinary smoked glasses also give in most cases a sufficient protection against the ultra-violet rays. Hence, such glasses are always indicated when with much ultra-violet light very dazzling visible rays are present (in dazzling from snow, in work with the arc light, etc.).[2] If we have to do with a light that is less intense, these glasses by cutting off the visible rays impair the ability to see minute objects. For such cases it is desirable to have a glass which keeps out the visible rays either little or not at all, but keeps out the ultra-violet rays as completely as possible. Glasses having this property have usually a yellowish, amber, or greenish tinge and a special chemical composition. They go under various trade names, such as euphos, chlorophyll, etc. Having very little color tinge but quite effective in excluding the ultra-violet rays is the Crookes' glass.

115. Eye Guards.—For purposes of prophylaxis against eye injuries eye guards are used. These should protect the eye not only in front but also at the side. On the other hand, they should not exclude the air from the eye altogether, because by so doing an unpleasant feeling of heat is engendered, and the glasses themselves become covered on their inner surface with moisture. Hence the lateral protection must be effected by means of a fine wire mesh, which permits the circulation of air. To further coolness and circulation of air, an open space can be left between the upper rim of the guard and the brow since in civil life foreign bodies practically never reach the eye from above (Collis). The transparent part lying right in front of the eye is made of glass or, on account of their being less fragile, of celluloid, mica, or wire tissue. But glass is the only material transparent enough to afford really good sight. These spectacles are hot, and readily become covered with sweat, moisture, dust, or smoke, and unfortunately, therefore, are worn by the workmen with much reluctance. The glass also soon becomes clouded from the constant impact of metallic and other particles. Hence the guard should be so arranged that the glass can readily be removed and replaced (Collis).

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[1] Also in looking at the sun, especially through a telescope. For this purpose and also in certain forms of welding sometimes deeply colored glasses are used, e. g., a red glass backed with a green or a red glass backed with a blue.

[2] When smoked or colored glasses are ground in the form of convex or concave lenses, the thickest part, whether edge or centre, looks darker than the rest. This can be obviated by grinding a plano-convex or plano-concave lens of white glass and cementing to it a plane slab of the desired color.

116. Eye Dressings.—A dressing for the eye may be either a protective or a pressure dressing. The object of the *protective dressing* is simply to keep the lids closed and still. By this means erosions, ulcers, or wounds on the surface of the eye are kept from being scoured by the movement of the lids, a process by which pain is set up and the regeneration of the epithelium and the precise apposition of the lips of the wound are interfered with. Hence in major operations we usually bandage both eyes on the first day in order to put the lids in a state of complete rest. When there is much secretion from the eye, a dressing is not indicated. A bandage should also be used with circumspection for an eye that is predisposed to glaucoma, since the pupil dilates when the eye is covered and the mydriasis so caused may set up a glaucomatous attack. The protective dressing should be as light as possible. First, a bit of gauze is laid on the closed lids, then the hollow over the eye is packed flush with cotton, and the cotton held in place by strips of plaster or a bandage. In children, from whose heads an ordinary bandage always keeps slipping because of their continual restlessness, an immovable dressing made of a starch bandage is indicated. If the lids stick together beneath the dressing we smear the layer of gauze which lies upon the eye with an ointment containing boric acid or ichthyol. If it is a question of protecting the eye against a blow from the hand, which might force open a recently united wound (such a thing may readily occur in children in any time, and adults during sleep), a protective wire cage or an aluminum shell (Snellen) is fastened over the eye. (See also Ring's mask, Fig. 397.) A moist dressing in which the cotton is soaked in boric-acid solution and the like, and which has a piece of waterproof material placed over it, readily produces eczema of the skin of the lids.

The *pressure dressing* is made by applying more cotton to the eye and drawing the bandage tighter. When such a bandage is taken off after being on some time, the eye is somewhat softer than usual; in fact, some times the cornea is thrown into fine wrinkles, and the eye weeps and shows some ciliary injection. The pressure acting on the eye from without has increased the intra-ocular pressure by its own amount, so that now the aqueous flows out under a higher filtration pressure, and the eye thus becomes softer. The application of the pressure dressing is made with the expectation that pathological liquids, e. g., the subretinal liquid in detachment of the retina, will like the aqueous, more readily leave the eye. But very frequently the pressure dressing is badly borne; the eye becomes suddenly very soft, the anterior chamber unusually deep, and the ciliary injection may actually increase into an iritis. We must, therefore, proceed very circumspectly in applying a pressure dressing. The sensitiveness of many eyes with detachment of the retina towards a pressure dressing, arises from the fact that in such eyes the vitreous is not healthy and under the pressure dressing its volume rapidly decreases. Eyes which are sound in their deeper parts usually bear the pressure bandage well. We can, therefore, apply it without danger and even with the additional help of an elastic bandage, if we are dealing with ectasiæ of the external coats of the eye. An ectasia which has already developed cannot probably be relieved thus, but probably also the forma-

tion of such an ectasia can be prevented. If the yielding spot (the thinned floor of an ulcer in the cornea, a prolapsing iris, a softening of the cornea due to pannus or severe parenchymatous keratitis, a softened portion of the sclera) can no longer resist the normal intra-ocular pressure, it is possible by diminishing this pressure with a pressure bandage for a protrusion at this spot to be averted until the place has become sufficiently firm. With this object, an elastic bandage is applied once or twice a day for as long a time as the patient can bear it without pain, which is usually for some hours (Fuchs).

In all cases where there is either the thinned floor of an ulcer, a recently united wound, or a still open perforation of the eyeball, sudden increase of pressure may lead to bursting open of the weak spot and to escape of the contents of the eye. Therefore, everything must be avoided which may produce a sudden *increase of pressure*, such as squeezing the lids, bodily labor like lifting a heavy weight, even stooping, straining at stool, chewing, and sneezing. Sneezing can be avoided if pressure is made at the right moment with the finger on the hard palate in the region of the foramen incisium. When perforation threatens, rest in bed is indicated.

II. Symptomatic Treatment

117. Physical Agencies.—Apart from this so-called hygienic treatment of the eye, disease of the eye requires special treatment adapted to it, which may be both symptomatic and causal. For symptomatic treatment both physical and medicinal remedies are at our disposal. The physical agencies applied are as follows:

118. Heat and Cold.—The effect of heat and cold is usually secured by means of cold or hot compresses. Because of the thinness of the lids, the effect of these compresses extends not only to the conjunctival sac but also to the eyeball itself, in fact even to the tissues of the orbit. The effect of iced compresses is more thorough than that of warm compresses. The former can depress the temperature by about 4° C., the latter can raise it 1° or more. The effect of *cold* compresses is to contract the blood-vessels and thus combat the hyperæmia. They are chiefly used in recent injuries, after the action of corrosive agents and of severe cauterization of the conjunctiva, or after the expression of trachoma granulations and finally in gonorrhœal conjunctivitis during the first stage. They diminish the feeling of heat and the pain. Furthermore, since cold retards physical and chemical processes, they are applied to diminish the swelling of the lens when it threatens to develop too violently after wounding of the lens capsule. Much more in use are *hot* compresses. Heat dilates the blood-vessels, increases the hyperæmia, and accelerates metabolism, so that the natural protective substances of the blood are brought right to the spot. Phagocytosis by the leucocytes that have migrated from the blood-vessels is increased. Heat, therefore, reinforces the curative efforts made by nature in combating the lesion. Hence hot compresses find extensive application in inflammations of all sorts in the anterior portions of the eye, and often

also act to reduce pain. But theoretical considerations apart, it must be determined by observation how in individual cases the application of cold or heat is borne.

The compresses should be applied thus: The patient lies on his back, and the compress is kept on the closed lids by its own weight, and must not be pressed hard or tied tight upon the eyes. In order, also, that it may not press on the eye, it should not be too heavy. The best way of proceeding is to take a piece of linen or gauze, which is folded several times, so as just to cover the eye and its immediate vicinity; over this, a piece of water-proof material is applied in order to prevent too rapid alteration of the temperature. For cold compresses, the material is placed in cold water or upon a block of ice. Instead of plain water, lead water or a sublimate solution is employed in some cases. Hot compresses are made with hot water. The compresses, hot or cold, must be changed every minute or two. To obviate this, some have advised an ice-bag instead of the cold compresses and thermophores or Leiter's tubes instead of the hot ones; but most of these appliances have the disadvantage of being too heavy for the eyes.

Hot compresses are usually applied continuously for a certain period (say ten minutes), then intermitted for another period of twenty to fifty minutes. Cold compresses give the best results if applied without any intermission at all (De Schweinitz).

Hot moist applications to the closed lids may be replaced by *hot salt solution dropped directly on the eye* itself and especially on the cornea. For frequently repeated or semi-continuous applications, used in keratitis, scleritis, irido-cyclitis, and uveitis, the solution may be heated only to 40° to 46° C.[3] In this case the eye need not be anæsthetized, and the applications can be made very frequently. To produce an intense effect (sterilization of corneal ulcers) a solution heated to 70° or more and dropped directly on the ulcer has been used with good effect (Lippincott). In this case the cornea must be anæsthetized with cocaine. This is also necessary when in other ways intense heat is applied directly to the eyeball. Such an application is made in four ways. Either a jet of *hot air* is propelled by the action of a rubber bulb from a metal tube that has been heated over an alcohol lamp (Bourgeois); or a jet of *steam* or *alcohol vapor*, imparting a heat of 98° C and 78° C, respectively, is used (Wessely); or, in the method of *chauffage,* a galvano-cautery point kept at a suitable temperature by means of a rheostat and held close to the eye but not in contact with it is moved slowly in front of the affected part for the space of a minute (Weekers); or the metal head of a *thermophore* heated to a determinate temperature (up to 60° C) is held in contact with the sclero-corneal margin for some three minutes (Shahan). Dry heat applied by these different methods has proved effective in checking the progress of corneal ulcers and ulcus serpens, and, according to Shahan, the contact thermophore may diminish the tension in glaucoma.

Hot applications may be replaced by *diathermy,* or the passage through the parts of a high frequency electric current, which by the resistance of the tissues is converted into heat.

Heat is also applied either by the actual *cautery* or galvano-cautery to destroy an infecting area, e. g., the germ-laden margin of a corneal ulcer (see § 66).

119. Phototherapy and Actinotherapy.—*Phototherapy,* or the application of light, especially light very rich in ultra-violet rays, has been used in corneal ulcers (Hertel, Schanz).

[3] The temperature should be tested by dropping the liquid first on the back of the hand, but it will be found that in many cases the eye will tolerate liquid too hot for either the skin, the lids, or the palpebral conjunctiva to bear.

Actinotherapy by means of the x-rays or radium is used in tuberculosis, trachoma, and spring catarrh of the conjunctiva, and occasionally in keratitis; with good result also in angioma and epithelioma of the lids, sarcoma of the orbit, and some cases of epibulbar epithelioma or sarcoma. It should not replace enucleation in the case of intra-ocular sarcoma or glioma (except perhaps in certain cases of localized sarcoma of the iris), but should be used after enucleation for intra-ocular tumor and after removal of an orbital sarcoma to prevent recurrence and also may be used after recurrence has taken place.

In superficial lesions of the eye or adnexa the radium is applied in contact with the parts, the applicators being so shaped as to parallel the latter, in order that the radium may exert a uniform action. The cornea in this case is sheltered by a lead screen. In tumors, when possible, the radium or radium emanation is buried in the centre of the mass. Single large doses are of more value than repeated small ones (Janeway); indeed an initial small dose may aggravate the condition (New and Benedict).

In applying either radium or x-rays it is important that the α-rays and β-rays should be filtered out, as they act harmfully on the superficial parts. The γ-rays do not do this, and at the same time they penetrate deeper, and act much more energetically on morbid growths than do the α-rays and β-rays (Leber).

120. Electricity.—Electricity gives good service in paralysis or spasm of the orbicularis. It is also applied in paralyses of the eye muscles. We are not, however, able to make an eye muscle contract by means of the faradic current, unless the muscle is exposed, and it is, therefore, a question whether faradization is of any therapeutic value at all in paralyses of the eye muscles. The case is scarcely better for the galvanic current, which has but very slight current density when it flows through an eye muscle. So, too, the therapeutic results of galvanization in ordinary atrophies of the optic nerve are very doubtful. Better authenticated is the action of electricity as an analgesic in eye inflammations. A saucer-shaped electrode is placed on the closed lids, the other electrode is put in the patient's hand, and a weak faradic current is passed (Reuss). Electricity achieves veritable triumphs in all cases in which the action of suggestion is concerned, that is, in the numerous cases of neurasthenic and nervous asthenopia and amblyopia. The way in which we apply the electricity is of less consequence than our ability to impart to the patient the conviction that electricity is a very powerful curative agent which will surely relieve him. Electricity can also be applied for the purpose of making remedies (especially iodine and mercury) which are dissolved in water, penetrate into the tissues of the eye by means of the galvanic current (cataphoresis).

Differing from cataphoresis, in which the substance as such is conveyed into the tissues, is *electrolysis*, in which a chemical compound is dissociated, one component of it (ion) collecting at the anode, the other at the cathode. Conveyance of ions by this means is called *iontophoresis* or *ionic medication.* A current of not over 2 milliamperes is used. Substances applied by iontophoresis are zinc sulphate in ⅕- to ½-per-cent solution (in ulcus serpens, dendritic keratitis, and herpes corneæ) and quinine sulphate in herpes zoster. In a more mechanical way electrolysis is used to remove cilia and shrivel up vascular tumors of the lids (see §§ 922, 926).

121. Suction Hyperæmia.—Bier's hyperæmia can be induced in the eye by suction.

A glass of the shape of a cupping glass is placed with its mouth upon the lids, and the air in it is rarefied by suction made with a rubber bulb. There are produced œdema of the lids and conjunctiva, some exophthalmus, and evidently also an exit of fluid from the eye itself, for the latter directly after the procedure is softer, although the diminution in tension is not lasting. The method is employed with advantage in purulent processes in the lids.

122. Blood-letting.—Blood-letting is accomplished by means of leeches, or with Heurteloup's apparatus. Leeches to the number of from 6 to 10 are placed on the temple, not too close to the lids, as otherwise the latter become œdematous and swollen. Blood-letting is proved to have a particularly favorable action in severe irido-cyclitis.

In inflammation of the deep parts (chorioid, retina, optic nerve) it is used but rarely now. In such cases we withdraw the blood from a point over the mastoid process because an emissary vein of Santorini, which carries off the blood from the transverse sinus and thus indirectly from the cavernous sinus and the ophthalmic vein, empties here.

Maddox, observing the favorable effect of epistaxis in congestive conditions of the eye, recommends that blood-letting be effected by incising the nasal mucous membrane with a slender keratome. The same author has found local blood-letting useful as a prophylactic against expulsive hæmorrhage (see § 888).

123. Massage.—Massage is used to absorb scleritic nodules and, combined with the application of yellow oxide of mercury ointment, to clear up corneal opacities; to reduce the tension of the eye either before or after an operation for glaucoma (see §§ 81 and 846); and in retinal embolism to dislodge the embolus. It may be applied directly to the eye, which must then be anæsthetized, but is usually applied to the eye through the closed lids.

Massage is usually applied with the fingers. In recent embolism it is made as far back as possible on different portions of the eyeball; or the operator may squeeze the ball between two fingers placed on opposite sides of the globe, and then suddenly release the pressure—a manœuvre which causes the blood to flow suddenly into the vessels. To promote filtration and reduce the tension in glaucoma, two forms of manipulation are used (Elliot). In the first, which may be called palpation massage, the surgeon stands behind the patient and directs the latter to look down and gently close the eyes; then makes pressure through the upper lid upon the globe with the index fingers of the two hands, applying each finger alternately and directing the pressure towards the centre of the eye. The massage is made at first for about half a minute and with slow movements; later it may be prolonged over a period of three or five minutes, and the movements are made more rapidly. In the second form of manipulation, (rotary massage), three fingers of one hand are applied to the closed lids with a gentle, circular, smoothing motion for fifteen or twenty seconds.

124. Diaphoresis.—Diaphoresis is employed for inflammations (scleritis, irido-cyclitis, chorioiditis, retinitis, retro-bulbar neuritis), and for the absorption of blood, exudate, opacities of the vitreous, and subretinal

liquid. Diaphoresis by the subcutaneous injection of pilocarpine is efficacious but so disagreeable for the patient that it has largely been given up. The most usual means of producing sweating are: (*a*) the taking of 1-2 grammes of aspirin with hot lemonade, and then covering up warm in bed; (*b*) hot baths followed by wrapping in a dry woolen blanket; (*c*) a hot-air bath in bed (in this the air is heated by an alcohol lamp and is carried by a tube beneath the bed-clothes, which are raised somewhat by means of a cradle); (*d*) vapor baths; (*e*) the electric-light bath. We select any one of these procedures, depending upon the patients's wishes and the local conditions, and we produce diaphoresis every day or at longer intervals according to the severity of the case and the patient's strength.

Sweating has a therapeutic value only when it is very profuse. If it remains only in the stage of congestion which precedes the sweating, or if the skin becomes but slightly moist, we get simply the disadvantages of the procedure without its advantages. If by sweating we wish to remove pathological products from the eye, the patient should take altogether very little liquid, since what we are trying to effect is the absorption of pathological effusions, by removing a large quantity of water. If, on the other hand, it is a question of removing toxic substances from the body, we make the patient drink water in abundance, since the excretion of sweat increases proportionally, and the injurious substances are the more abundantly eliminated.

Diaphoretic treatment is contraindicated in arteriosclerosis and heart disease, in patients who are in a very reduced state (especially tuberculous patients), and in pregnancy.

125. Medicinal Remedies; Modes of Applications.—Medicinal remedies are applied in solution or as ointments or in the form of powder. *Watery solutions* are intended to be dropped into the conjunctival sac once a day or oftener. Solutions which irritate and hence are followed by rather profuse secretion, should not be instilled directly before the patient goes to sleep, because the resulting reaction interferes with his going to sleep, and the increased secretion is prevented by the closed lids from escaping freely. On the other hand, remedies are to be instilled in the evening when we wish them to act particularly by night (thus, for example in the case of the miotics in glaucoma). If after being used a long time the solution shows cloudiness or flocculi it is to be renewed. In eyes with recent wounds due to injury or operation, only sterile solutions should be instilled. Slightly warm solutions pain less than cold solutions when they are instilled and are also rather more easily absorbed. It is useless to instil the solution in large quantities at any one time, for only a little remains in the conjunctival sac, and the rest at once flows off. One good drop is sufficient; if while we are dropping it in we make the patient look up and at the same time draw down the lower lid, the solution stays longer in the conjunctival sac and is absorbed in greater amount.[4] Drugs which alter the size of the pupil arrive by diffusion through the cornea into the aqueous, and thence act upon the iris (see § 73). A part of the solution is also absorbed by the conjunctiva and by the nasal mucous membrane, the solution reaching the latter through the tears. Ordinarily, this absorption is too slight to result in any general

[4] This is particularly the case if, after the patient looks up and while we are still holding the lower lid away from the eye, we direct him to look down so that the cornea is immersed in the fluid lying in the conjunctival sac.

effect (symptoms of poisoning). This occurs only when the instillation is made very often, or when very concentrated solutions are used.

Medicaments may be introduced into the conjunctival sac in *oily* as well as in aqueous solution and also in the form of soluble *tabloids*. *Ointments* are used in the conjunctival sac when either a long continued action is desired, or there is a profuse flow of tears which would wash out an aqueous solution at once, or when more or less irritant substances are to be applied.

The chief agents applied in the form of ointment are atropine, cocaine, and holocaine; boric acid, argyrol, or occasionally iodoform (after operations and for wounds or ulcers); copper sulphate or citrate (in trachoma); and white or yellow oxide of mercury (in corneal affections).

Just as in the case of irritant collyria, the copper and mercury ointments, owing to their irritant action, should not be introduced into the eye at night before the patient goes to sleep. Ointments intended for the conjunctival sac should not contain any coarse particles which would act as a mechanical irritant. The medicinal substance must, therefore, be in the very finest powder, and must be rubbed up with the ointment bases with special care so as to form a uniform mixture. (This is particularly the case with the yellow oxide of mercury ointment. This should be kept in black jars, since it is gradually decomposed by the light.)

As a soothing application, especially in abrasions of the cornea, simple white vaselin squeezed out of a tube is useful.

An ointment designed for the conjunctival sac should be taken up on a clean glass rod, warmed nearly to the melting point, and then deposited gently between the lids.

Ointments intended for the margin of the lid are rubbed in upon the closed lids in the evening before the patient goes to sleep, so as to act during the night. Crusts and scales are previously to be removed as far as possible by washing them with tepid water or water and soap, (§ 430). Since sometimes it is impossible absolutely to prevent some of the ointment from getting into the conjunctival sac and upon the cornea, very irritant ointments which are well borne by the skin cannot be applied to the eye. For lid ointments a fat should be chosen as a basis which is somewhat hydrous, for the water as it gradually evaporates cools the inflamed borders of the lid (cooling ointment). We accordingly select cold cream or, as this soon becomes rancid, vaselin with which a small quantity of hydrous lanolin is mixed.

Powdered remedies must be in a very fine powder. Calomel, gallicin, and dionin are sprinkled with a brush upon the conjunctiva of the depressed lower lid, not upon the cornea; iodoform is sprinkled upon the raw spot in the eye.

126. Remedies Used; Atropine.—The remedies most used in eye diseases are as follows:

Atropine, this paralyzes the oculo-motor nerve terminals in the sphincter iridis and in the ciliary muscle, and hence produces dilatation and immobility of the pupil and loss of accommodation. The effect of the atropine makes its appearance in from ten to fifteen minutes after the instillation, and soon reaches its maximum. Commencing with the third day it begins to decrease again, but does not disappear completely until after the lapse of a week or longer. Quite as long lasts the disturbance of sight which is

caused partly by the dilatation of the pupil, but mainly by the paralysis of the accommodation, and hence makes itself apparent most of all in near vision (causing inability to read).

In infants atropine, even in poisonous doses, often exerts very little effect on the pupil. In others the width of the atropinized pupil averages 7.5 mm., but varies from 6 to 9 mm., the least dilatation being found in those over 40 years of age.

Atropine is such an efficient mydriatic that the millionth part of a gramme suffices to dilate the pupil. If we instil atropine into another person's eye, and in so doing wet our finger, and then through carelessness touch our own eye with it, this is enough to produce dilatation of our own pupil.

The instillation of atropine and still more its application in substance (see infra) sometimes cause symptoms of *poisoning*. These consist in a troublesome feeling of dryness in the throat, nausea, reddening of the face, and an acceleration of the pulse; also excitement and tremor, or, in case of severe poisoning, loss of consciousness. In marked poisoning the pupil of the other eye that has not been treated with atropine is dilated, too. Like evidences of general poisoning with dilatation of the pupil and reduction of the accommodation, causing dazzling and poor vision for near objects, are also produced by the internal use of atropine or remedies containing it (belladonna), sometimes by the application of belladonna plasters. Infants are especially susceptible to the poisonous effects of atropine, and in them it should be used sparingly or not at all—the more so as it does not produce in these cases the desired dilatation of the pupil.

Some persons show an *intolerance of atropine*. This makes itself apparent in various ways: (*a*) By the appearance of symptoms of poisoning, like dryness of the throat and nausea, even with small doses. This occurs particularly after long-continued use of atropine. (*b*) By exciting a catarrh (atropine catarrh) which is generally marked by the formation of numerous follicles. For this also a prolonged use of atropine is usually requisite. (*c*) In some persons a single drop of atropine produces great redness and swelling of the lids, like an attack of erysipelas.

Atropine is used to put the iris at rest in case of irritation or inflammation of this organ, and is also used to produce paralysis of the accommodation, in order to get a precise determination of the refraction. Generally a 1-per-cent solution of atrophine sulphate is used. When a particularly great effect is to be attained, we place a granule of the atropine salt in substance in the conjunctival sac, where it dissolves in the tears, and affords a concentrated solution. Still greater is the effect if we combine atropine with cocaine so that contraction of the dilatator of the pupil is added to paralysis of the sphincter. We cocainize the eye just as for an operation, and after anæsthesia has set in, place a granule of atropine sulphate in the conjunctival sac.

In applying atropine (especially atropine in substance) to the eye we preclude symptoms of poisoning if we prevent the tears from flowing down into the nose. With this object we draw the lower lid away from the eyeball for a little while, so that the tears pour out over the cheek, or we compress the lacrimal sac with the finger and if the patient is a child depress the head toward the shoulder so that the tears shall not run down into the mouth. In cases of severe poisoning a subcutaneous injection of morphine is indicated as an antidote.

Atropine is contraindicated in old persons with shallow anterior chamber, because in them glaucoma might be set up by the dilatation of the pupil, and obviously it is rigorously forbidden when glaucoma has already broken

out. (The same is true of homatropine, euphthalmine, and cocaine.) It must farther be discarded in persons who show an intolerance of its effects.

In such cases the atropine, according to circumstances, must either be simply given up or it must be replaced by another mydriatic. Of these the most suitable is *scopolamine hydrobromide*, and in ¼- to ½-per-cent solution, for it is more poisonous but also more active than atropine. By some this in ⅕-per-cent solution is used regularly instead of either atropine or homatropine in determining the refraction (see § 217).

Since atropine in ordinary dosage causes disturbances of sight lasting a week or more, we should never instil it without substantial reasons. For the same reason we do not use atropine when it is simply a question of dilating the pupil to facilitate examination with the ophthalmoscope, but employ homatropine, euphthalmine or even cocaine.

Too much caution cannot be inculcated in regard to the senseless way in which *atropine* is often used, as it still is, unfortunately, by many general practitioners, who instil it in every kind of eye disease. In many cases—e. g., in conjunctival catarrh—atropine is not only superfluous, but also causes the patient annoyance through the disturbance of vision produced by its use; and in eyes which have a tendency to glaucoma, atropine may actually inflict great injury by determining an attack of acute glaucoma. Accordingly, atropine should be employed only upon quite specific indications, and should be applied no oftener than is requisite to obtain just the result desired.

127. Homatropine.—This is used in 2-per-cent or 3-per-cent solution to paralyze the accommodation and thus facilitate the determination of the refraction (see § 217). The effect on the pupil and vision in this case regularly equals that produced by atropine in the same case, lasts several days, and is not promptly controllable by pilocarpine or even eserine. Hence when we do not wish to produce such a thorough going action, but simply to dilate the pupil in order to make an ophthalmoscopic examination, we use a ½- or 1-per-cent solution, the effect of which lasts less then twelve hours and can be abrogated by one or more instillations of 1-per-cent pilocarpine. It is particularly important not to use the stronger solution in case there is any suspicion of glaucoma.

Homatropine in 2-per-cent solution may also be used as a tentative application instead of atropine in abrasions and slight infiltrations of the cornea (see §§ 541, 591) and as a means of diagnosis in suspected iritis (§ 682). In 1-per-cent solution it may be used to diagnosticate a suspected glaucoma (§ 830) and to ascertain the site of election for an optical iridectomy (§ 951).

128. Euphthalmine.—Euphthalmine in a 2-per-cent solution produces a comparatively transient dilatation of the pupil, which can be abrogated by 1-per-cent pilocarpine. It is hence used for much the same purposes as the 1-per-cent homatropine solution. In 5-per-cent solution it produces a much more lasting effect, which pilocarpine may not suffice to counteract.

129. Miotics.—*Eserine* (also called *physostigmine*) has an action exactly the opposite of that of atropine, since it places the sphincter iridis and the ciliary muscle in a state of tonic contraction. Consequently, miosis develops so that the pupil is about the size of a pin's head, with adjustment of the eye for a near point, as if marked myopia were present. Eserine diminishes the intra-ocular pressure somewhat especially in hypertonic eyes. *Pilocarpine* acts in a similar way but less energetically.

The action of miotics is of shorter duration than that of the mydriatics, and is also less powerful. Hence a pupil contracted by eserine or pilocarpine can be dilated by atropine, but a pupil dilated by atropine cannot be contracted by a miotic.

Eserine frequently excites severe pain in the eye and head, painful contractions in the lids, and even nausea and vomiting. These are not symptoms of poisoning, but are the results of the great narrowing of the pupil and contraction of the ciliary muscle, by which the ciliary nerves are pulled upon. This bad by-effect, therefore, usually is absent when, for any cause, the eserine fails to produce any marked contraction of the pupil.

A solution of eserine after being a few days in the light turns red (for which reason it ought to be prescribed in dark or paraffined bottles), without, however, suffering any impairment of its activity.

Eserine and pilocarpine may be used in paresis of the iris and ciliary muscle, but, as their action is transient, are effective only when the paresis is well on the wane (see § 380) or when the paresis itself is transient, e. g., when due to homatropine or euphthalmine. When used for this purpose they should be given in full doses, i. e., the eserine in ¼-to ½-per-cent (in certain cases even 1-per-cent) solutions, and the pilocarpine in 1- or 2-per-cent solutions.

Of much greater value are these agents in glaucoma, in which they are used in a strength and frequency varying according to the acuteness of the glaucomatous process and the results of the treatment (see § 846)

130. Cocaine.—Cocaine dilates the pupil—not, however, as in the case of atropine and its congeners by paralyzing the sphincter pupillæ, but by contracting the dilatator. The dilatation of the pupil is therefore only a moderate one unless a strong solution or repeated instillations are used, and the reaction of the pupil to light persists; moreover, mydriatics and miotics still produce an effect. If cocaine is instilled into an eye the pupil of which has been dilated by atropine, the dilatation increases somewhat; hence the mydriasis produced by the simultaneous action of atropine and cocaine is the most considerable that can possibly be attained. The accommodation is not paralyzed by cocaine, but only somewhat weakened.

Besides acting upon the iris, cocaine produces also the following effects: The conjunctiva becomes very pale, and at the same time the patient has a feeling of cold and dryness in the eye. The palpebral fissure is more widely open and the act of winking is less frequent. Sometimes the eye is protruded somewhat forward and the intra-ocular tension slightly diminished (see § 80). The practically important phenomenon, however, is the anæsthesia shown by the superficial tissues of the eye (cornea and conjunctiva).

The effects of cocaine are best explained upon the assumption that it acts as a stimulant to the fibres of the sympathetic. The contraction of the vessels thus produced causes the pallor of the conjunctiva. The sympathetic also innervates the musculus tarsalis superior and inferior (see § 3) and the dilatator pupillæ, and the contraction of these muscles causes the dilatation of the palpebral fissure and of the pupil. The anæsthesia of the surface of the eyeball has nothing to do with the sympathetic, but depends on the paralysis of the sensory nerves.

In applying cocaine we generally use a 2-to-4-per-cent solution of the hydrochloride, distilled several times at short intervals. This is used: (1) To produce a transient dilatation of the pupil, especially in suspected glaucoma or other conditions in which we are afraid to use a mydriatic which is not so readily counteracted by eserine. (2) To reinforce the

action of atropine or homatropine. (3) To reduce the congestion of an inflamed eye and so promote the absorption of a mydriatic or miotic subsequently introduced. (4) To shrink a turgid mucous membrane, e. g., that of the tear duct, in order to make the latter more patent. (In uses 3 and 4 it can be combined with or replaced by adrenaline). (5) To diagnosticate a paralysis of the sympathetic (see § 374). (6) To produce anæsthesia. If we instil a solution several times into the conjunctival sac, we obtain complete insensibility of the conjunctiva and cornea in a few minutes while the sensitiveness of the iris is not abolished until after fifteen minutes or so and then often not completely. The anæsthetic effect lasts ten minutes or more. It is most frequently used as a preliminary step to any painful manipulation of the eye, such as the removal of foreign bodies and actual operations. For the latter purpose it may be introduced not only by instillation, but also by subcutaneous or subconjunctival injection (§ 893) or by cataphoresis (§ 120). It also serves to diminish photophobia and blepharospasm, and hence facilitates examination. Finally, cocaine is frequently prescribed for instillation in painful inflammations of the eye. But since it renders only the superficial portions of the eye insensible, it acts to relieve pain only in inflammations of the cornea, but not in irido-cyclitis, glaucoma, etc. But it is precisely in lesions of the cornea that cocaine can also *do harm*. We can convince ourselves of this fact when we instil cocaine a number of times in succession before an operation. The epithelium of the cornea then often becomes opaque and dull, or may even be exfoliated in places.

There are two reasons for this; first, the direct injurious action of the cocaine on the epithelial cells and, second, the drying of the surface of the cornea which occurs because, as a result of the insensitiveness of the cornea, the lids do not wink enough. We should not, therefore, apply cocaine for too long a time and, while we are applying it, should take care that the patient keeps his eyes shut after each instillation; then we shall rarely have to complain of "cocaine opacity." If we entrust a patient with a cocaine solution, to remedy some painful inflammation of the eye, he is easily induced to drop it in often because the analgesic action of the cocaine lasts only a short time. This frequency of instillation because of the injurious action of cocaine on the corneal epithelium may readily exert an unfavorable influence on the course of a keratitis. Holocaine, especially in ointment or in oily solution is a better drug than cocaine in such cases, and dionin or acoine is usually much better than either.

The mydriasis produced by cocainization remains for some hours and may annoy the patient by the disturbance of sight that is associated with it. Both the mydriasis and the disturbance of sight can be speedily relieved by the instillation of a drop of pilocarpine. Part of the disturbance of sight is caused by dryness and consequent unevenness of the cornea. This can be relieved if the patient shuts his eyes at intervals so as to moisten the cornea.

131. Other Local Anæsthetics.—Of these the most important are:

Holocaine (*phenacaine*). This is more poisonous than cocaine. It seems to have no injurious action on the cornea, does not contract but rather congests the blood-vessels and causes little or no dilatation of the pupil. In 1-per-cent solution it is a very effective anæsthetic, used particularly when we wish to avoid acting on the pupil or tension (hence, especially in tonometry, in operating for glaucoma, and in the removal of foreign bodies).

All the other agents are less poisonous than cocaine. Of these—

Procaine (or *novocaine*) is similar in action to cocaine but is weaker. It is the best anæsthetic to use (in 2-per-cent solution) for subconjunctival injections, and with the addition of adrenaline it can be used to anæsthetize the eye for enucleation.

Tropococaine and *stovaine* dilate the pupil less than cocaine; with the former the conjunctival vessels are unchanged, the latter dilates them somewhat. Stovaine is used in a 4-per-cent, tropococaine in a 1- to 3-per-cent solution.

Eucaine B and *alypine* leave the pupil quite unaltered but dilate the blood-vessels somewhat. Eucaine is used in a 1- to 3-per-cent, alypine in a 2- to 4-per-cent or, if injected subconjunctivally, in 1-per-cent solution.

Acoine has no effect on the vessels and the pupil. It has been used mainly as an addition to solutions which are to be injected under the conjunctiva; but procaine is better for this purpose. A 1-per-cent solution of acoine in castor oil may relieve for a number of hours the sharp pain produced by erosions or abrasions of the cornea (especially those remaining after the removal of a foreign body).

Oil of rose (beta-phenylethylol), which since the time of Dioscorides has been used as an adjuvant to collyria, has an anæsthetic effect about as great as that of cocaine when applied to the conjunctiva and is even superior to cocaine when used intracutaneously. It is much less toxic than cocaine (Barbour). *Benzoyl carbinol* would seem to be equally or even more effective as it is also but slightly toxic. The like is true of *saligenin.*

Butyn in 2-per-cent solution acts more powerfully than 4-per-cent cocaine, and appears to be less toxic (Bulson). It does not affect the cornea or pupil. In ½-to 1-per-cent solution it can be used for subconjunctival or subcutaneous injection.

132. Adrenaline.—*Adrenaline, suprarenin, paranephrin, epinephrine,* are names given to an extract of the supra-renal capsules, and *tonogen* and *epinine* are synthetic preparations which have the property of causing the smooth muscle fibres supplied by the sympathetic to contract, and hence act primarily as vaso-constrictors. If one of these drugs is dropped into a very much reddened eye, the latter after a few seconds becomes pale as that of a corpse and remains so for an hour or longer. The large conjunctival vessels and the deep (scleral) vessels are not usually affected. Adrenaline may hence be used to differentiate between a deep and a superficial injection (in suspected iritis, etc.), and to show the character of tumefactions of the conjunctiva, since it reduced that part of the tumefaction due to vascular engorgement, but leaves unchanged that due to exudates. On account of its transient action it is not adapted for the treatment of inflammation of the eye, but only for the production of a temporary ischæmia. If we have an inflamed eye to operate on, it reinforces the action of the cocaine and diminishes the bleeding during the operation. It may also be instilled during an operation, in order to diminish a troublesome bleeding. For the same purpose we add it, in the proportion of two or three drops to the cubic centimetre, to solutions of cocaine or other anæsthetics intended for subcutaneous or subconjunctival injection. Finally it is used to diminish the turgescence of the mucous membrane of the tear duct and so to render the latter patulous (see § 903).

Adrenaline occasionally produces mydriasis by its stimulating action on the dilatator pupillæ. This seems to occur particularly in glaucomatous eyes or in those predisposed to glaucoma, and in such eyes, contrary to the rule that mydriatics are harmful

n glaucoma, the tension is rarely elevated by its use (A. Knapp). It may, therefore e used to confirm the diagnosis of glaucoma in doubtful cases and in actual glaucoma to lilate the pupil for purposes of examination.

133. Dionin.—Dionin (ethyl-morphine hydrochloride), when introluced in powder or in a 5-per-cent solution into the conjunctival sac, produces, first, much burning and injection of the eye, after which, in a few ninutes, a marked hot œdema of the conjunctiva develops which may go on o a tense chemosis with puffing of the lids and with sneezing. Some recomnend beginning with a 2½-per-cent solution and gradually increasing to 10-per-cent. The œdema disappears again in a few hours. The great yperæmia of the conjunctiva causes the natural protective substances of the blood to pass into the tissue of the conjunctiva and into the interior of he eye. A further curative factor is the fact that the escape of bloodserum in quantities into the conjunctiva can act to modify the circulation n the eye. We, therefore, attribute to dionin an effect in promoting absorption, for which reason we employ it for clearing corneal opacities. In scleritis and irido-cyclitis, dionin ameliorates the violent pain and also little by little diminishes the injection of the eye. Furthermore, it acts very well to counteract marked photophobia in phlyctenular conjunctivitis, parenchymatous keratitis, etc. In detachment of the retina and other affections of the deeper parts it may be used by subconjunctival injection (see below). On repeated application less and less œdema is produced and the therapeutic effect of the drug ceases too. Then further application produces only a useless sensation of burning without any curative effect.

Dionin is said occasionally to produce rise of tension without causing mydriasis. Hence in doubtful cases it may be well to combine with it a miotic (Elliot).

134. Hypertonic Solutions.—These, applied in the form of an eyebath for five minutes every two or three hours, have been used to promote osmosis and increased lymph flow in congestive and inflammatory conditions, especially those associated with chemosis (corneal ulcers, gonorrhœal ophthalmia). Specially recommended is magnesium sulphate in solutions ranging from 8 per cent up to saturation (Kirkpatrick).

135. Subconjunctival Injections.—The injection of active liquids beneath the conjunctiva has a twofold effect, namely, irritation of the tissue and alteration in the conditions of diffusion. The irritation of the tissue manifests itself at once by pain and by redness at the site of injection. A more remote consequence of the irritation is the passage of the natural protective substances from the blood into the tissues and spaces of the eye, and upon this probably depends part of the curative effect of the injection, especially in corneal ulcers. The alteration of the conditions of diffusion occurs because after the injection there is present beneath the conjunctiva a salt solution (e. g., a 5-per-cent solution of sodium chloride) having a concentration quite different from that which belongs to the liquid in the vitreous. The differing osmotic tension tends to become equalized through the animal membranes which lie between the two liquids (sclera, chorioid, retina). Thus there is given a stimulus to metabolism,

which may promote the absorption of subretinal liquids in detachment of the retina, of exudates in the inner membranes of the eye, and of hæmorrhages and vitreous opacities. The injections are made with a hypodermic syringe after the eye has been cocainized. In corneal diseases they are made beneath the anterior portion of the conjunctiva (but not too near the limbus). In diseases of the posterior segment of the eyeball they are made in Tenon's space but not far back. The redness and swelling that set in after the injection disappear after one or more days; and according to the way this occurs the injection can be made either every day or at longer intervals. After long-continued injections especially if combined with acoine, there is formed an adhesion between conjunctiva and sclera, which becomes more and more solid all the time. The substances which are oftenest used for injection are mercury oxycyanide (1 to 3 minims of a solution varying in strength from 1:1000 to 1:4000) and a 5- to 10-per-cent sterilized salt solution (one-half to one syringeful); also dionin in from ½- to 5-per-cent solution. In order to make the injection less painful cocaine, or better 0.2 gm. of a 4-per-cent solution of procaine or of a 1-per-cent solution of alypine are added. In corneal ulcers, in which it is a question of causing an irritant effect, it is better to use the mercury solution, and the salt solution in diseases of the deep parts where the excitation of diffusion is the thing to be considered (Fuchs). Not painful and apparently efficacious are injections of 0.7-per-cent salt solutions (Wilmer). In inflammatory irritation of the eye injections are not well borne. It must be remembered also that the injections tend to increase the intra-ocular tension, so that they should not be used if there is a suspicion of glaucoma.

136. Strychnine.—This exerts an excitant action upon the optic nerve, so that even in normal eyes it produces a slight although not permanent increase in the visual acuity and an enlargement of the field of vision. For therapeutic purposes as much as 5 mg. may be injected once a day beneath the skin of the temple, and this dose may be increased to 10 or even 12 mg. especially in paresis of the iris or ciliary muscle, in which condition it may give good results.

Strychnine acts best in disturbances of vision unattended by changes visible with the ophthalmoscope, especially in hysterical and neurasthenic forms, which, however, generally afford a good prognosis anyway. In serious lesions of the optic nerve, as in progressive atrophy, we often obtain with it an improvement in the sight and especially an enlargement of the field of vision; but these changes are not usually permanent.

III. Causal Treatment

137. Local Treatment.—Causal treatment is conducted in accordance with the etiology of the individual case. Hence it admits of a general consideration only so far as infectious diseases are concerned. The warfare upon the microbes and upon the effect they produce in the tissues can be accomplished by local and by general treatment. *Local* treatment consists in applying antiseptics, by which germs lying on the surface of the eye are killed or rendered inert; in applying detergent solutions, by which the germs are washed away; or in applying caustics, by which an infected portion of tissue is destroyed along with the germs that it contains. In the case of the

eye, these measures can be used in infection of the superficial structures only, i. e., of the conjunctiva, cornea, and occasionally the anterior chamber.

The chief *antiseptics* used in the eye are zinc sulphate, which is specifically injurious to the Morax-Axenfeld bacillus (§§ 453, 457); silver nitrate and other silver preparations, which attack various germs, but especially the gonococcus (§§ 453, 465, 466); optochin (§ 453), which attacks the pneumococcus; hypochlorite solution (Dakin's solution) or solution of chloramine (§ 860), and corrosive sublimate. The last named in 1:3000 solution, although it does not affect germs lying beneath the surface or hidden in remote recesses, does render inert the germs lying free in the conjunctival sac. It is therefore used before operations, when it suffices to make the field sterile during the time that the operation wound is being made and until it is sealed. Collargol in 1-per-cent ointment may be used for the same purpose (Salzmann).

The *detergents* are the ordinary normal salt and boric acid solutions and argyrol, which owing to the way in which it penetrates in to the recesses of the conjunctival sac and descends into the lacrimal duct exerts a continuous detergent action, which is probably of as much consequence as its antiseptic effect.

Caustics are applied only when we have to do with a germ mass in the superficial membranes of the eye and above all in the cornea. The germ mass must not have too great an extent, as, otherwise, too widespread destruction would have to be made, and the caustic must be applied so that its action shall be precisely limited to the affected spot. This is true of the eye more than of other organs, because, for example, in the treatment of corneal ulcers every square millimetre of transparent cornea is of importance. Hence, liquid caustics or those which dissolve quickly are unsuitable, because they would flow over the neighboring parts and destroy them. The action of nitric and pure carbolic acid however, can be sharply localized, and these two agents are often used to limit corneal suppurations. Still better answering the requirements is the actual cautery which is usually employed under the form of a fine galvano-cautery loop. It is used above all in ulcus serpens and other rapidly advancing corneal ulcers. Weaker caustics are a stick of copper sulphate, whittled to a sharp point, which is employed to advantage in keratitis dendritica, and tincture of iodine which is applied with a swab to the floor of an ulcer, after curetting away the necrotic portions.

Where caustics are not advisable, the attempt must be made to strengthen the natural *resisting power* of the tissues against bacteria. Serviceable in this way are the application of heat, subconjunctival injections, and paracentesis of the cornea, measures to which belongs the property of favoring the passage of protective substances into the tissues of the eye, and most of all into the cornea.

Under the head of local treatment may be included the removal of a focus of *infection remote from the eye*, but causing disease of the latter. Such foci are found in the teeth, tonsils, accessory nasal sinuses, and intestinal tract, and cause all sorts of eye disorders (prominently keratitis, iritis and uveitis, optic nerve disease, weakness of

accommodation, and asthenopia). After the infection from these foci has become generalized, even thoroughgoing removal of the latter does not suffice and may have to be combined with a vaccine treatment (see § 140).

138. General Treatment.—General treatment has to reinforce local treatment and is the only form of therapy possible when the bacterial focus is not directly accessible. Besides the remedies, like diaphoresis, mercury, and iodine, which are applied generally in infectious diseases of various kinds, there are also those which possess a quite specific action for certain morbific agents. This action is based on the fact that by passing through one infection one acquires an immunity, varying in degree, against a new infection by the same morbific agent, because the body forms specific protective substances against the latter. The action of the protective substances may consist either in their killing the bacteria themselves or in their neutralizing the poisons produced by the bacteria. Such an immunity may also be produced artificially, and that either in a direct or indirect way. The former is *active immunization* or vaccine therapy, which consists in injecting into the patient dead or weakened cultures of bacteria of the same kind as those by which he has been made ill, so that he himself may form in his own body substances protecting against these bacteria. The type of such an active immunization is the injection of tuberculin. The indirect method is called *passive immunization* or serum therapy, which consists in subjecting an animal to active immunization, so that its serum forms protective substances which then are employed for rendering the patient immune. The protective substances contained in this serum may be either antitoxic or bactericidal. The oldest example of the first case is the antidiphtherial serum, while the pneumococcus serum, for example, contains bactericidal substances.

The protective substances are of avail only against that kind of bacterium with which the immunization was produced. Since some bacteria act differently in different strains, some, in order to secure a specific immunization, try to immunize an individual case of disease by means of cultures which they have obtained from the morbific germs derived from the case itself. But the preparation of cultures in sufficient quantities demands so much time that in acute cases one would usually be too late to effect a cure. In acute cases, therefore, while the *autogenous* vaccines, i.e., those derived from the patient himself, are preparing, *heterogenous* vaccines of similar character derived from other sources are used. Or an autogenous serum may be used derived from a blister raised on the patient's skin (Römer). Another way of getting a specific serum with certainty consists in procuring it by immunizing animals with different strains of the same species of bacterium, so that one may expect that the special kind of strain by which the patient is rendered ill should be represented in it. Such a serum has been called *polyvalent* or better *multivalent.*

When a specific germ cannot be isolated from the lesion in the eye itself or from the blood, *complement fixation tests* are used in order to determine the specific organism producing the disease and the one that should be used in immunizations. The oldest of these tests (that for the spirochæta pallida) is the Wassermann test, but in recent years complement fixation tests for the gonococcus, pneumococcus, various strains of staphylococcus and streptococcus, the influenza bacillus, the colon bacillus, and the micrococcus catarrhalis have been applied (Hastings, Reber).

In ophthalmology the chief agents used have been diptheria and tetanus antitoxin for effecting passive immunization and tuberculin for effecting active immunization.

139 Diphtheria and Tetanus Antitoxin.—*Diphtheria antitoxin* has been successfully employed in diphtheria of the conjunctiva and various other infections, too, both by subcutaneous injection and by instillation into the conjunctival sac. *Tetanus antitoxin* is used to ward off tetanus in contaminated wounds of the eye or adnexa.

Other sera have been used with varying success e.g., anti-pneumococcus serum in ulcus serpens. Since it has been found that diphtheria antitoxin acts to combat other infections beside diphtheria, the efficacy of this and other sera has been attributed to the proteins they contain rather than to any specific antibodies. Acting on this hypothesis some have given and with considerable success intramuscular injections of *milk* to check infections of the cornea or other organs due to gonococci, etc. Five to ten cubic centimetres are injected usually into the gluteal muscle. Local, focal, and general reactions are produced as in the case of tuberculin (see § 140).

140. Tuberculin.—This is used extensively in ophthalmology, both for diagnostic and for therapeutic purposes. Its *diagnostic* application is designed to determine whether the patient is tuberculous at all and in particular whether his eye disease depends on his tuberculosis. The former is the case if the patient reacts in a postitive way, i. e., with a rise of temperature, to the tuberculin injection; the second, if a focal reaction occurs in the diseased eye.

The diagnostic application of tuberculin is performed by injecting the old tuberculin of Koch. Since in positive cases this excites fever, it is to be omitted as superfluous, if the presence of tuberculosis has been already made certain by the physical examination of the patient or by the examination of the sputum. It is specially important to make sure that there is no cavity in the lungs nor active tuberculous process in the joints or vertebræ, since in such cases the use of the tuberculin is dangerous (Wilder). Active tuberculosis being excluded, we place the patient in a hospital, and take his temperature every two hours. If after 24 hours the temperature is normal, we inject 1.0 mg. of tuberculin subcutaneously (preferably at 9 P. M., so that the reaction, which usually takes place within 8 or 9 hours, shall not occur at night and so escape us.—Török). Positive *general reaction* is denoted by a rise of temperature. If none occurs in 48 hours, 3 mg. should be injected, and if in 48 hours more there is no reaction 5 mg. may be given. If then there is no reaction, active tuberculosis may be assumed to be absent. It is important to place the injections thus close together, since, if they are put too far apart, anaphylaxis may develop. Besides the rise of temperature, there frequently develops, if tuberculosis is present, a *local reaction* at the site of injection, namely, an infiltration of the subcutaneous cellular tissue at the place where the tuberculin passed from the point of the needle into the tissue. The skin at this place is a little reddened and beneath the skin is felt a hard and rather tender spot. A *focal reaction* in the eye itself may also occur. This consists either in increased injection or in fresh exudations, i. e., it denotes an aggravation of the eye disease which in some situations, especially the cornea, may be quite dangerous. (For this reason it is unwise to use tuberculin as a diagnostic agent when both eyes are suspected of containing a tuberculous focus). In many cases the focal reaction occurs in the form of exudates in the chorioid or retina. To determine whether a focal reaction is present or not, we should examine the eye exteriorly and with the ophthalmoscope before and after each injection (Jackson). If a focal reaction is absent, when a general reaction is present, the diagnosis that there is a local tuberculous trouble is only a diagnosis of probability and it is in just such cases, too, that the eye condition seems less likely to respond to treatment by tuberculin. In such cases a

careful general examination should be made and if no other tuberculous focus is found and no other cause for the eye trouble, the latter may probably be regarded as tuberculous (Török).

Other diagnostic methods of employing tuberculin are the cutaneous methods in which the tuberculin is introduced, not beneath the skin, but into it and excites in the skin a local reaction without causing fever. In Pirquet's method the epidermis is scarified and tuberculin is placed on it; in Moro's method a tuberculin ointment is rubbed upon the intact skin.

The Pirquet reaction occurs not only in active tuberculosis but also in the presence of old healed tuberculous lesions. It is therefore usually positive in adults, since the great majority of these have been infected with tuberculosis at some time in life. Its use is hence confined to children. Even in them it gives no clue as to the site of the tuberculous process.

The *therapeutic application* of tuberculin is performed by means of subcutaneous injections, for which we take either old tuberculin, new tuberculin (TR), or the preparation known as bacillus emulsion. In this method, in contrast to what obtains in the diagnostic application, care must be taken that the patient does not get fever. We therefore begin with a very small dose, and increase it, but very gradually; and if the patient, in spite of this, does get fever, we return to the former smaller dose. We should do the same or lengthen the interval between injections if there is a focal reaction in the eye.

The initial dose should be 1: 10,000, 1: 5000 or at most 1: 2000 mg. and the dose should be increased at each injection, the injections being made at intervals of a week or so. At the Knapp Memorial Hospital the custom is to begin with 0.0001 mg. and give the injections every four days; adding to each injection 0.0001 mg. until a dose of 0.001 mg. is reached; and then adding 0.001 mg. till 0.01 is reached; then at weekly intervals adding 0.01 mg. till 0.1 mg. is reached; then every two weeks adding 0.1 mg. till 1 mg. is reached. This dose, which is not exceeded, is repeated once a month. In any event, whatever routine is adopted, the dose and interval must be regulated by the effect. In some cases we have to continue these injections for many weeks in order to attain success and in any event, even after apparent cure, an injection should be given every three or four months for years.

In some cases a lesion not apparently tuberculous is benefited by tuberculin treatment.

Other Forms of Vaccine Treatments.—*Other infections* in which vaccine treatment has been applied are those produced by the gonococcus, various strains of the staphylococci and streptococci, the influenza bacillus, and the pneumococcus. Such treatment has been particularly applied in uveitis of gonorrhœal origin or derived from dental infection and the like; also to arrest a beginning suppuration after operations. As above stated, complement fixation tests may be employed to determine the specific organism involved and to indicate the culture that should be used in forming the vaccine. As in using tuberculin, the amount of vaccine used and the interval between injections are regulated by the effect i. e., the presence or absence of a local reaction at the site of injection or a focal reaction in the eye.

141. Treatment in Syphilis.—Against the widespread parasitic disease, syphilis, no immunizing procedure has so far been found. Fortunately, in salvarsan (arsphenamin) and neosalvarsan, mercury, and iodine we possess specifically acting agents with which to combat this disease.

Treatment in syphilis is largely regulated by the complement fixation or *Wassermann test.* A positive Wassermann may be found as early as the first week, but usually not before the fourth week of the disease. In secondary syphilis and in tertiary syphillis with lesions it is almost invariably present, but may be banished for the time being by treatment. Per contra, when absent it may sometimes be elicited if we give an injection of mercury or salvarsan (*provocative Wassermann*). It must be noted that it is found in some conditions besides syphilis (yaws, trypanosomiasis, some forms of malaria, scarlet fever). But this usually causes no confusion.

In syphilis of the nervous system, including general paresis and tabes, the blood examination may prove negative, and yet the spinal fluid, obtained by lumbar puncture, will almost always give a positive Wassermann reaction. The spinal fluid at the same time usually shows lymphocytosis and a positive globulin reaction (Noguchi's *butyric-acid reaction*) and also exhibits characteristic changes (so-called *luetic curve*) when a series of dilutions of the fluid are treated with colloidal gold.

The Wassermann reaction can be replaced by Noguchi's *luetin test* (cutaneous reaction produced by the intradermic injection of dead spirochætæ pallidæ).

Salvarsan is used in doses of 0.25 to 0.30 gm., increased if need be, given by intravenous injection every one or two weeks. In syphilis of the nervous system if salvarsan given intravenously is not effective, salvarsan or serum obtained from the patient himself after an intravenous injection of salvarsan may be administered by intraspinous injection (Fordyce). Certain forms of tuberculous eye trouble, in which tuberculin fails, are benefited by salvarsan (Benedict).

142. Other Forms of General Treatment.—Besides treatment addressed to the infections there is that form of general treatment which is concerned with the metabolic processes and has to do with the regulation of the digestive functions and of the internal secretions (stimulation or inhibition of the endocrine glands); furthermore treatment designed to improve the conditions of circulation of the blood and lymph or to improve the sensory, motor, or trophic functions of the nerves. Many diseases of the eye require for their successful treatment careful consideration of one or more of these factors.

We may simply mention here the use of thyroid extract in parenchymatous keratitis and other forms of corneal disease; the use of small doses of calomel and careful regulation of the diet in phlyctenular keratitis and in uveitis; the prolonged administration of iodides to absorb vitreous and other opacities; and the exhibition of arsenic in disorders traceable to perverted nerve action (herpes zoster, corneal dystrophies).

PART III

ANOMALIES OF REFRACTION AND ACCOMMODATION

ANOMALIES OF REFRACTION AND ACCOMMODATION

143. The function of the refractive media of the eye, which form a part of the enveloping or protective system of the latter, is so to conduct and concentrate the light that impinges on it as to set up appropriate stimuli in the sensory organ proper, the retina. The eye, which is composed of these two structures, one subserving protection, nutrition, and conduction, the other subserving sensation, is constructed upon the principle of a camera obscura. This consists of a box blackened on the inside, the anterior wall of which contains a strong convex lens, which throws upon the posterior wall an inverted image of the objects that may chance to be in front of the camera. In the human eye we find instead of the convex lens quite a number of refracting surfaces constituted by the surfaces bounding the refracting media of the eye, namely, the cornea, aqueous humor, lens, and vitreous; and in place of the posterior wall is found the retina, which not only receives the image, but also at the same time perceives it. Hence a diminution of visual power may be produced by two different causes: either the dioptric (conductive) apparatus of the eye is defective, so that a sharp image is not thrown upon the retina, or the retina (sensory apparatus) is at fault so that the image is not properly perceived.

In order to throw a sharp image upon the retina the dioptric apparatus of the eye must fulfill two conditions. In the first place, the refracting media must be perfectly transparent. Hence opacities of the cornea, lens, etc., make distinct vision impossible. The second condition is, that the refractive power of the media should be such that they project an image of external objects which is both perfectly distinct and also lies precisely upon the retina. The variations from this rule we designate as errors of optical adjustment or as errors of refraction and accommodation. The theory of these errors, as we have it to-day, forming a harmonious, well-compacted whole, is chiefly Donder's work. It is these errors that we shall consider in the present part of the book, reserving for Part IV the consideration of the disorders of the sensory apparatus proper.

ANOMALIES OF REFRACTION AND ACCOMMODATION[1]

CHAPTER XII

THE THEORY OF GLASSES

144. Prisms.—In optics the name prism is given to a wedge-shaped block of glass or similar material, used to deflect the rays of light. It has an apex (B, Fig. 58), a base, AC, and sides, AB and BC. ABC is the apical or *refracting angle.*

Since the material of which the prism is made is more refractive (optically denser) than air, a ray of light, OD, impinging on the side AB is, according to the laws of optics, bent toward the perpendicular, GDE; i. e.,

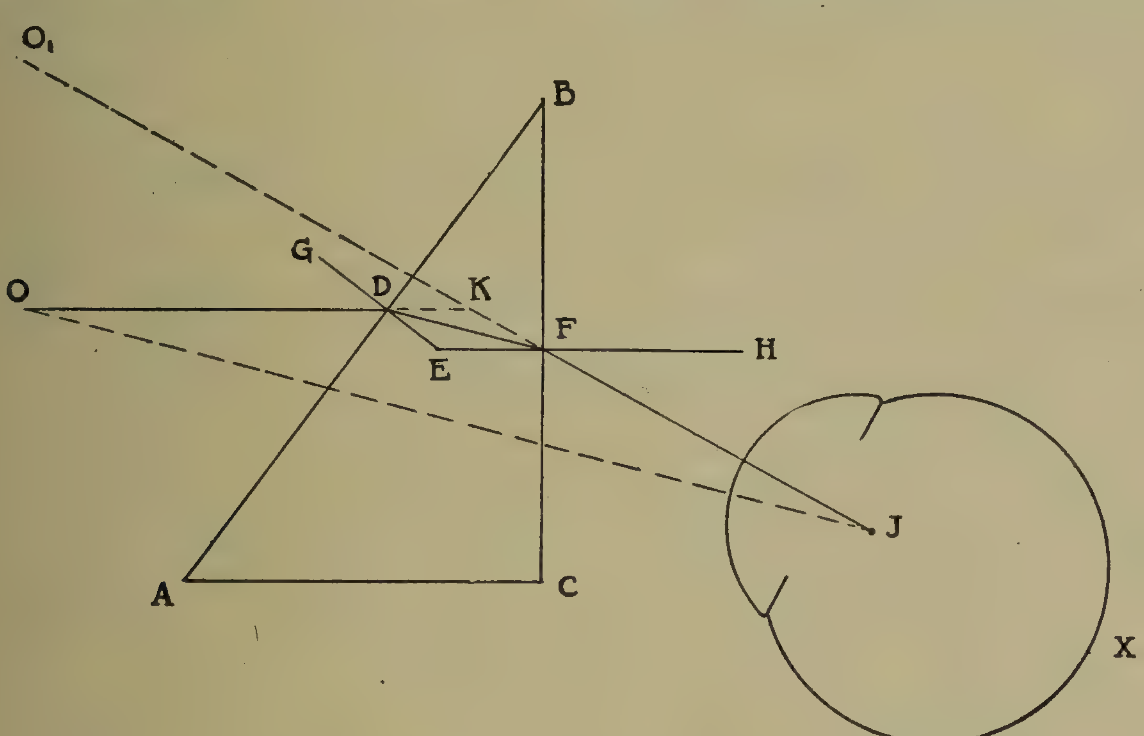


Fig. 58.—Refraction Through a Prism.

it takes a course DF such that the angle of refraction, KDF, is less than the angle of incidence, ODG. After traversing the prism the ray emerging on the other side BC, is now according to the same optical law deflected away from the perpendicular, EFH, and takes the course FJ, such that the angle of refraction, HFJ is now greater than the angle of incidence, DFE. The net result is that the refracted ray, KJ, which originally had the direction OD, now has the direction FJ, i. e., has been bent toward the base of the prism. The angle OKO_1 between the original and the final directions is called the *angle of deviation.*

The optical density of the material composing the prism is measured by what is called the *index of refraction* of the material. This index really represents the relative speed of the light rays in a vacuum, or what is practically the same thing, in air, com-

[1] This whole part has been completely revised and remodeled by the translator, who therefore must be regarded as responsible for the statements made.

pared with their speed in the material. Thus when we say that the index of refraction of ordinary optical glass is 1.54, we mean that light travels 1.54 times as fast in air as it does in glass. If n denotes the index of refraction of a medium, e. g., of the glass composing the prism ABC, then in all cases the value of the angle of refraction KDF $(=r)$ as compared with the angle of incidence OGD $(=i)$ is given by the equation

$$\sin i = n.\sin r$$

This is called the *law of sines*, and is the basis of the whole theory of refraction.

145. Deflection Produced by Prisms; Minimum Deviation.—The ratio between the refractive angle of a prism (ABC) and the deviation (OKO_1) varies with (1) the optical density of the glass; (2) the size of the refracting angle; and (3) the amount of the angle of incidence OGD i. e., with the position in which the prism is held. The last named factor has a considerable influence, and to avoid the variations due to it care should be taken that when prisms are used for clinical purposes they are always held in one definite position. The best position theoretically is that producing a minimum deviation. Practically identical with this is the position in which the posterior surface of the prism (the one turned toward the patient) is perpendicular to the incident ray or line of sight OD (see Fig. 58 in which the prism is so placed and the table below). This is the position which should be invariably used in practice. Particular care should be taken not to hold the prism with its front face perpendicular to the line of sight.

The amount of deviation produced is shown in the following table.

DEVIATION PRODUCED BY PRISMS OF DIFFERENT REFRACTING ANGLES.

(Back face perpendicular to line of sight; index of refraction 1.54).

Refracting Angle	Deviation	Refracting Angle	Deviation	Refracting Angle	Deviation
1°	0.5	10	5.4	25	14.1
2	1.1	11	6.0	30	17.2
3	1.6	12	6.5	35	20.5
4	2.2	13	7.1	40	24.0
5	2.7	14	7.7	45	27.9
6	3.2	15	8.2	50	32.1
7	3.8	16	8.8	55	36.7
8	4.3	18	9.9	60	42.1
9	4.9	20	11.1		

It will be observed that for weak prisms the deviation is a little over one half the refracting angle.

146. Numbering of Prisms.—Prisms are numbered either according to their *refracting angles* or according to the *deviation* they produce. In the latter case the numbering may be by actual degrees of minimum deviation effected (Jackson), by centrads (Dennett), or by prism dioptries (Prentice). Numeration by centrads and prism dioptries is practically equivalent to numeration by refracting angles, at least for prisms of ordinary strength. To distinguish degrees of minimum deviation from degrees of refracting angle, the former are denoted by the symbol d.

A centrad is a deviation of 0.57°, or the hundredth part of a radian. A *prism dioptry* is a deviation that produces a deflection of 1 cm. in an object situated 1 metre from the eye. By far the best method of numeration is that of *minimum deviation,* since this corresponds to the actual angular effect of the prism in measuring or correcting a deviation of the eyes and gives a better idea of the real position of the eyes. Thus if a person is overcoming a prism of 25° (or 25 centrads), base out, he is not converging through an angle of 25°, but one of a little over 14°; but if he is overcoming a prism of 25° minimum deviation (25 *d*) he is actually converging 25°. Since for certain purposes (summation of prisms) it is necessary to know the refracting angle, this should be marked on the prism along with the degree of minimum deviation.

147. Displacement in Apparent Position of Objects Produced by Prisms.—A prism not only deflects the rays of light coming from an object O, but also to an eye looking through it makes O appear in a different place. For according to the laws of projection the eye will see O, not as would be the case if the prism were absent, in the direction of the line JO connecting the object with the eye, but in the direction of the line JF. The object will therefore appear to be somewhere along this line, e. g., at O_1. That is, it will be displaced toward the apex of the prism.

We may thus use a prism to displace the apparent position of an object up, down, in, or out, according to the direction in which the apex of the prism is set before the eye. In this way we produce any desired kind or amount of double vision. We may also correct double vision when it exists, and thus measure the deviation that causes it, or make one eye swing in any given direction in order to get an image on the macula. See §§ 287, 290. The angle OJO_1 is called the *angle of displacement.* It is rather less than the angle of deviation, but if the prism is held close to the eye the difference is usually negligible.

148. Lenses as Aggregations of Prisms.—If we place a series of truncated prisms of gradually increasing strength one on top of the other, the rays of light passing through the series will be deflected as shown in Fig. 59, i. e., not at all by the central segment and in increasing amount by each successive segment from the centre outward. We see how if the successive prisms are of proper strength the rays passing through them can all be converged to the same point, F, which is hence called the focus (principal focus) of the system. If now we multiply the number of segments (component prisms) indefinitely, we have in effect a double convex lens.

Just as rays falling upon the lens in a parallel direction ultimately reach the principal focus, F, so also rays which go in the opposite direction, from F, and impinge upon the lens, will emerge from the latter in a parallel direction (principle of conjugate foci—see § 154).

A concave lens is composed of a like series of prisms, only now the apices of the latter are directed toward the centre (Fig. 60) instead of away from it. The rays passing through these component prisms consequently are deflected away from each other and do not meet at all. They do, however, diverge from a common point, F, situated in front of the lens. This point is also designated by the name of focus, but in this case is called *virtual* to distinguish it from the *real* focus of a convex lens. Here, precisely as in the case of convex lenses, the law holds good that the path of the rays is the same when the direction is reversed. If rays impinge upon the lens from its

posterior aspect with a convergence such that they are directed to ward F, they will be parallel upon their emergence from the lens at its anterior aspect.

149. Varieties of Lenses.—The lenses shown in Figs. 59 and 60 are biconvex and biconcave. In addition to these, plano-convex (Fig. 61 A)

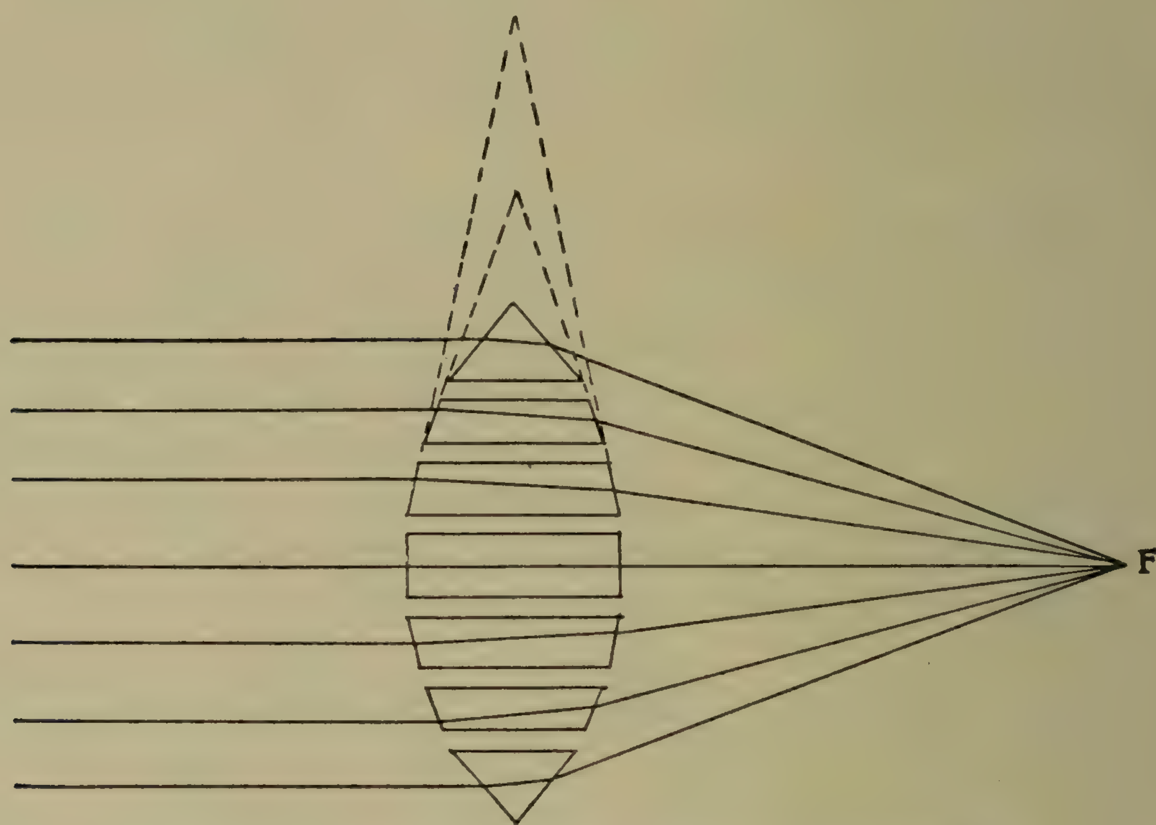


Fig. 59.—Aggregation of Prisms Forming a Biconvex Lens. F, real focus.

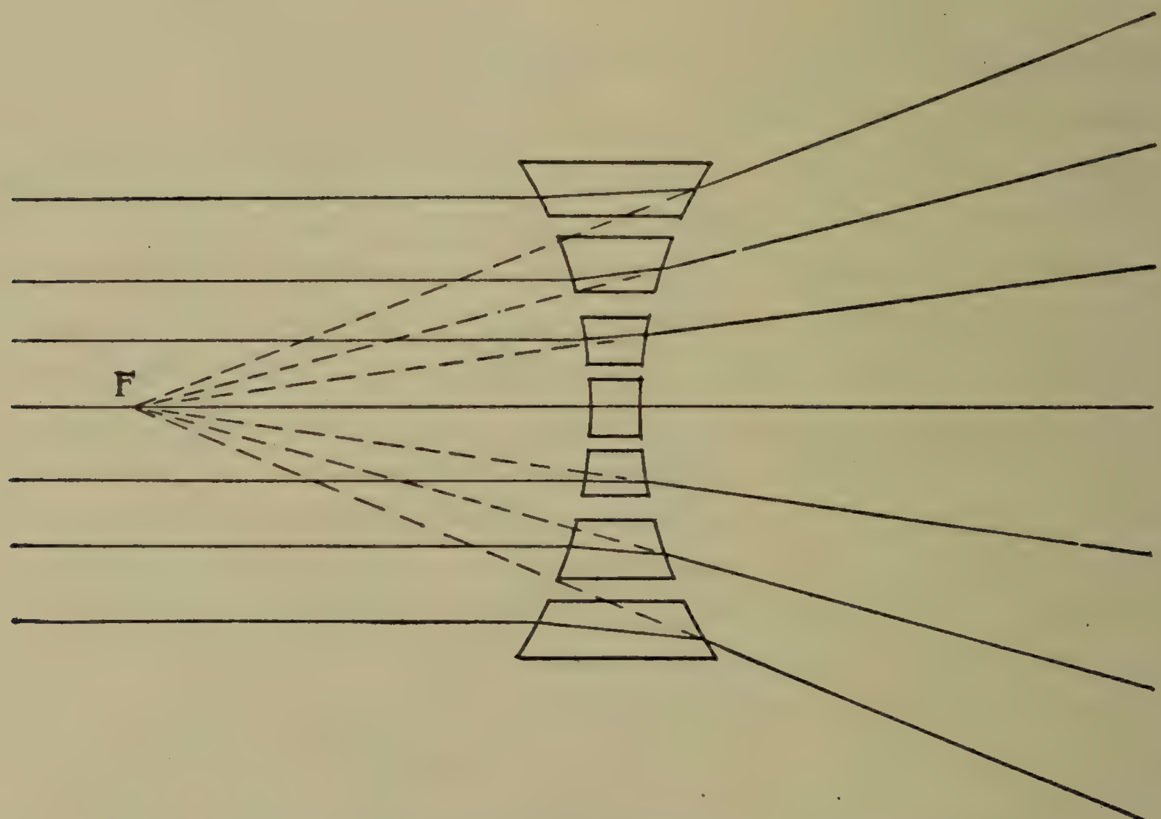
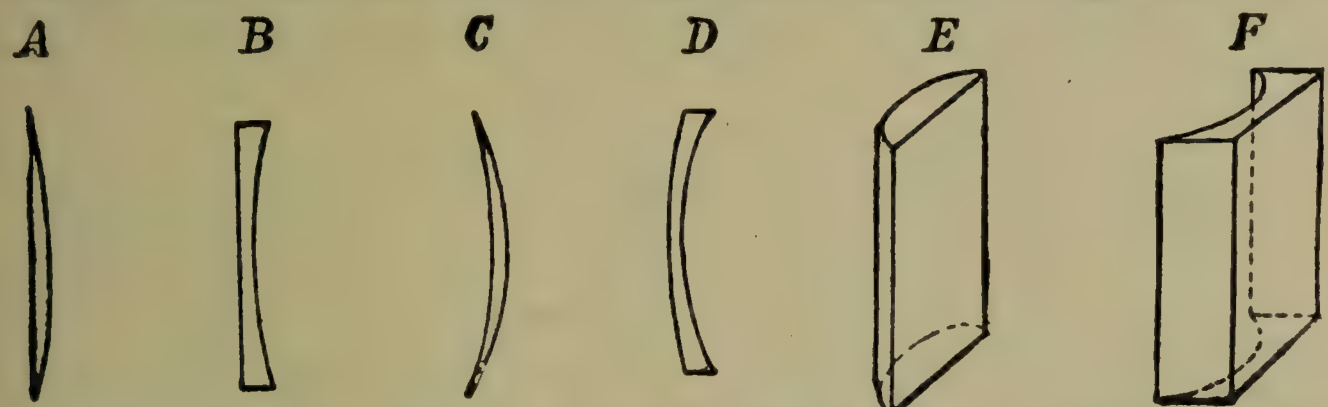


Fig. 60.—Aggregation of Prisms Forming a Biconcave Lens. F, virtual focus.

and plano-concave (Fig. 61 B) lenses are employed, the refracting power of which is one-half of that possessed by doubly curved lenses having the same surface curvature. There are also lenses one surface of which is convex, the other concave. If the radius of curvature of the two surfaces is the same,

o that they are parallel with each other, the glass acts very nearly like a ›lane glass. This is the case in the coquille or curved protective glasses.)n the other hand, the lens acts as a convex lens if the convex bulges more han the concave surface (concavo-convex lens, meniscus, Fig. 61 C) and as , concave lens in the reverse case (convexo-concave lens, Fig. 61 D). Con-avo-convex and convexo-concave lenses have the advantage that we see



IG. 61—A, plano-convex lens; B, plano-concave lens; C, meniscus; D, convexo-concave lens; E, convex cylindrical lens; F, concave cylindrical lens (concavo-convex lens).

s distinctly when looking through the marginal portion of the glasses as 'hen looking through their centre, while ordinary lenses give distorted nages when we look through their marginal portion. Hence they are lso called periscopic [2] glasses.

In most cases the surface bounding the lens has a spherical curvature hence, spherical lens). Some concavo-convex and convexo-concave glasses, owever, are ground with a toric surface.[3]

Convex, including biconvex, plano-convex, and concavo-convex lenses re called positive and are denoted by the + sign; concave lenses are called egative and receive the — sign.

150. Cylindrical Lenses.—A segment cut out of a cylinder (Fig. 62) ›rms a convex cylindrical lens (Fig. 61 E). The cylinder from which such segment is cut may be regarded as consisting of an ıfinite series of circles set one on top of the other. 'he centres of these circles lie in a straight line which called the *axis* of the cylinder. Any plane passing ırough this axis is called an axial plane. Rays im-inging on the lens and lying in an axial plane suffer o deviation. Rays, on the other hand, which lie in plane perpendicular to the axis (corresponding to ›b) undergo the maximum refraction—i. e., that of sphere having the same curvature as *bbb*. The ıme thing is true of concave cylindrical lenses (Fig. 1 F), which may be regarded as representing the ıold of the positive cylinder.

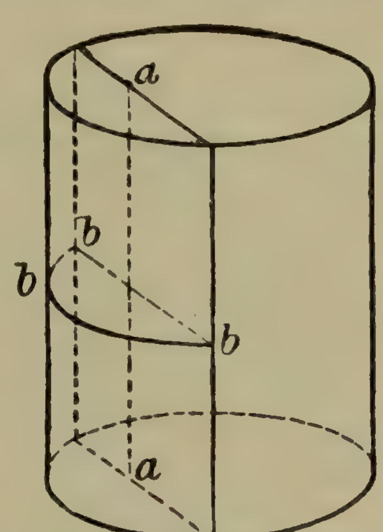


Fig. 62.—The Convex Cylindrical Lens Considered as a Segment of a Cylinder.

Astigmatism of Lenses.—Rays impinging on a cylinder a plane oblique to the axis are refracted by an amount which creases with the angle that the plane makes with the axis. Though thus refracted, ıey are not brought anywhere to a focus; i. e., for all planes that are not axial or trans-ial the lens is *astigmatic*. The like is true of a spherical lens when tilted or when the

[2] From περί, about, and σκοπεῖν, to see.
[3] A toric surface is one generated by rotating a circle about some point other than its centre.

impinging rays strike it quite obliquely or near its periphery. Astigmatism due to the non-focusing of the peripheral rays is called *spherical aberration.* Lenses also have *chromatic aberration* due to the fact that the different colored rays of which white light is composed differ in refractivity, so that they are nowhere focused at the same point. The image formed by such a lens has colored fringes. A lens free from spherical aberration is *aplanatic;* one free from chromatic aberration is *achromatic.*

151. Notation of Axis in Cylinders.—According to the notation prevailing in this country the direction of the axis of a cylinder is indicated by

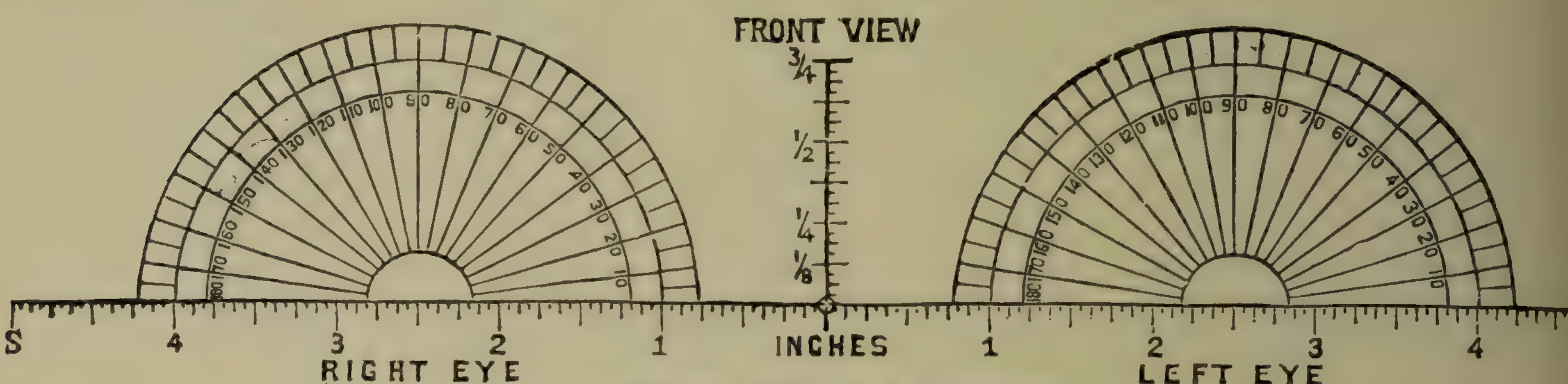


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Fig. 63

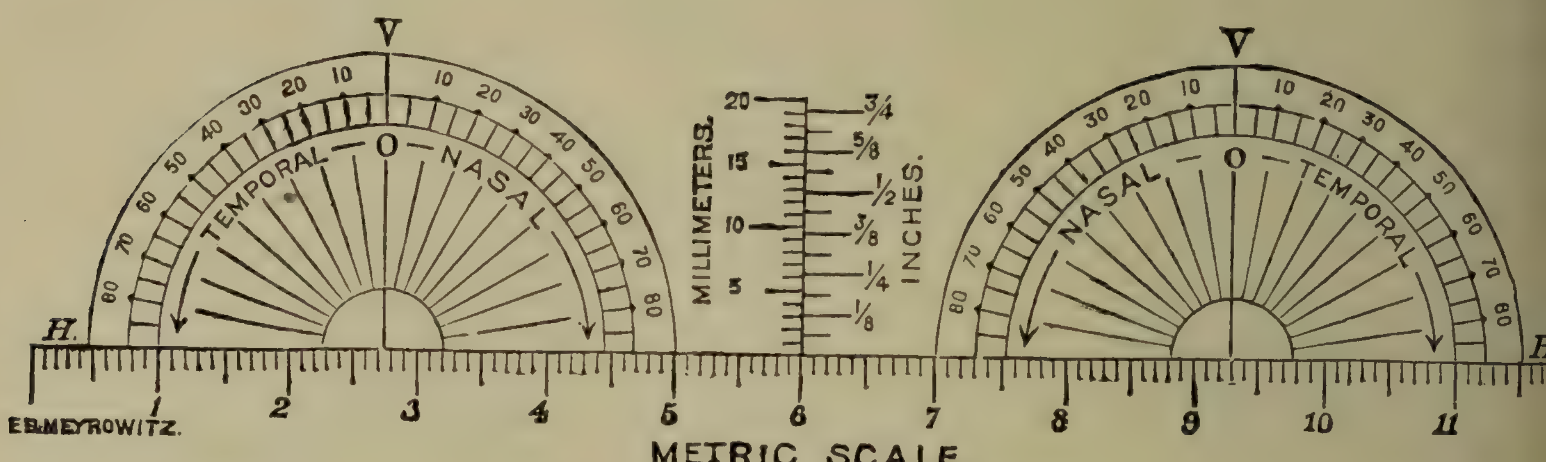


Fig. 64

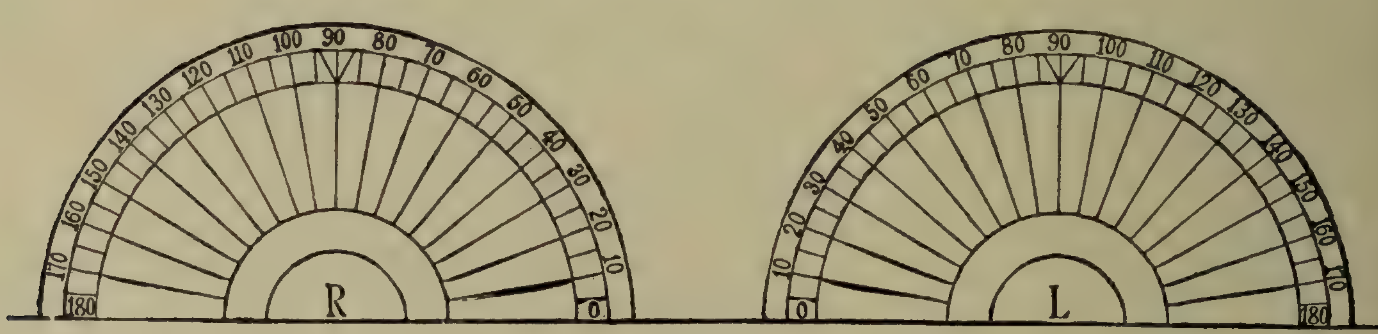


Fig. 65

the angle which it makes with the horizontal, the angles being numbered continuously from 0°, which is situated at the left side of either eye (nasal side of the right eye, temporal side of the left eye), round to 180° at the right side of the eye. This system is shown in Fig. 63.

According to another system the vertical meridian is indicated by 0° or *V*, and from this point the angles are numbered on either side to 90° (or *H*), which is the horizontal, those angles on the temporal side being indicated by *t*, those on the nasal side by *n*. This system is shown in Fig. 64.

A third system recommended by the International Ophthalmological Congress of 1909, is shown in Fig. 65. The angles are numbered in each eye from 0° at the nasal to 180° at the temporal side.

These systems are also employed for indicating the direction of the apex or base of a prism —e. g., prism 1°, apex at 75°, indicates a prism of 1° refracting angle with its apex turned to the left 15° from the vertical.

152. Prismatic Effects of Lenses.—Since a spherical lens is really composed of an aggregation of prisms (see § 148) it causes an object seen through it to appear displaced, the amount of the displacement increasing from the centre of the lens to the periphery.

This property is made use of when, instead of prescribing prisms, we *decentre* lenses, i. e., direct them to be worn so that the wearer looks through some point other than the optical centre. The decentration, required to produce a desired prismatic effect, is given well enough for all practical purposes by the simple formula

$$e = \frac{10c}{L} = \frac{18d}{L}$$

Where c is the prismatic effect in centrads or prism-dioptries, d is the prismatic effect in degrees of actual deviation, L is the strength of the lens in dioptries, and e is the decentration in mm. Thus to produce an effect of 0.5d in a 3D lens, we should have to decentre the latter $18 \times 0.5 \div 3 = 3$ mm. So also a 2.5 D lens decentred 2 mm. would produce an effect of 0.5 centrad.

The prismatic effect produced by decentering a *cylindrical lens*, compared with the effect s of a sphere of the same strength may be stated as 0, 0.5s, or 1s, according as direction of the decentration is 0°-20°, 40°-50°, or 70°-90° from the axis of the cylinder.

153. Foci, Principal Points, and Nodal Points.—In every lens or combination of lenses, in which the centres of curvature of the refracting surfaces are in line with each other (centred system), there are two principal foci and two principal points. The *first principal focus* is a point so situated that rays emanating from it (in the case of a convex lens) or directed toward it (in the case of a concave lens) before refraction become parallel after refraction. (It is often called the anterior focus, because in a convex-lens system it lies in front of the first refractive surface. In a concave lens, however, it lies behind the refractive surface, so that term anterior is inapplicable.) The *second principal focus* of a lens is the point in which rays that were parallel before refraction will intersect after refraction. It is posterior to the first refractive surface in the case of convex lenses, anterior in the case of concave lenses. The *first focal distance* (often, less properly, called the anterior focal distance) is the distance from the first focus to a point of determinate position, situated usually within the lens or near it, called the *first principal point*. Similarly the *second* (or posterior) *focal distance* is the distance from the second focus to a determinate point called the *second principal point*.

A focal distance, whether first or second, is positive if the focus from which it is measured is behind its corresponding principal point; otherwise is negative.

If, as in the case of a lens in air, the medium traversed by the emergent rays has the

same optical density as the medium traversed by the incident rays, the two focal distances are the same. Otherwise, the following relation holds good

$$p : s = n_1 : n_2$$

where p is the first, and s the second focal distance, and n_1 is the index of refraction of the first, and n_2 the index of refraction of the last medium. Thus in the eye, in which the index of refraction of the vitreous is four thirds that of the air, the first focal distance is three fourths that of the second.

The *nodal points* are two points so situated that the first is behind the first principal point, and the second behind the second principal point by an amount equal to the difference between the two focal distances. Hence, when the latter are the same, e. g., in the case of a lens in air, the nodal points coincide with the principal points. The important property of the nodal points is that any ray which before refraction is directed toward the first will after refraction pass through the second and have a direction parallel to its original direction. Hence, if we know the position of the nodal points in the eye and wish to determine the situation of the retinal image, *b*, of an object, *o*, all we have to do is to draw a line from *o* to the first nodal point and then another line from the second nodal point, parallel to the first line. The point where this second line strikes the retina will indicate the site of *b* (cf. § 161). The two nodal points, the two foci and the two principal points together constitute the *cardinal* points of an optical system.

Unlike a spherical lens, which focuses parallel rays in a single point, a cylindrical lens focuses them along a straight line (*focal line*) parallel to the axis of the cylinder.

154. Conjugate Foci.—If we hold a lighted candle at a point *A* some distance in front of the first focus of a convex lens, the rays emanating from the flame will be collected to form an inverted image of the latter at a point *B* some distance back of the second focus of the lens. If now, leaving the lens in place, we put the candle at *B*, an inverted image of the flame will be formed at *A*. In other words, we have simply made the rays of light retrace their course and have substituted image for object.

This is an illustration of a principle of universal applicability, called the *principle of conjugate foci*. This may be stated as follows:

In any suitably constructed refractive system rays intersecting in (either diverging from or converging to) any point *A* before refraction will intersect in some point *B* after refraction. If then the direction of the rays is reversed —i. e., if they are made to intersect in *B* before refraction and go backward —they will intersect in *A* after refraction. *A* and *B* are called conjugate foci (cf. Fig. 183).

A lens, it will be seen, has any number of pairs of conjugate foci, the position of one member of a pair varying, often very greatly, according to the position of the other. Thus in the example cited as the candle *A* is brought closer and closer to the first focus of the lens, the image *B* recedes faster and faster, and when A coincides with the first focus, *B* recedes to infinity, i. e., the refracted rays are parallel. If now the candle *A* is brought closer still to the lens, the refracted rays are no longer even parallel, but are simply rendered rather less divergent, so that they apparently emanate from a point *B* in front

of the lens. B is still the conjugate focus of A, but is now a virtual focus. In every case, however, the principle of conjugate foci holds good. Thus when B becomes virtual the rays travelling backward and directed toward B before refraction will unite in A after refraction. The same principle of reversed action applies to the principal foci, too (cf. § 148).

155. Images.—By refraction through a lens an object situated at any point A will form an image at B, the conjugate focus of A. This image is real or virtual, according as the focus B is real or virtual. Thus, as stated in the preceding section, an object situated some considerable distance from a convex lens will form on the other side of the latter an inverted image of itself, which is produced by the actual coming together of the rays and which we can therefore catch upon a screen (*real image*). This is the case, for example with the image formed by a camera or that formed on the retina. But if, as is the case when we use the convex lens as a magnifying glass, the object is brought up to some point within the first focus of the lens, so that the rays passing through the latter from the object are not brought together at all, but simply rendered somewhat less divergent, we shall if we place ourselves on the other side of the lens, see an enlarged, erect image of the object formed at the point from which the rays have been made to diverge. This *virtual image* is nothing but the object itself seen under a different angle. The same is true of the virtual, erect and diminished image formed in an altogether similar way by a concave lens.

Quite like the virtual image formed by a magnifying glass is the image of the fundus that we see when standing some distance from a patient with a fairly considerable degree of hyperopia we throw light into the eye with the ophthalmoscope. On the other hand the fundus image that we see in a strongly myopic eye when examined in the same way is a real inverted image formed in front of the eye by the eye itself acting like a camera lens (cf. §§ 172, 399).

The image, real or virtual, of a distant object is formed at the principal focus of the refractive system.

156. Strength of Glasses; Numbering of Glasses.—By the strength of a lens we mean its refractive power, i. e., its ability to bend the rays of light. The more it does this, the closer the point of union of the rays comes to the lens, and the shorter, therefore, is the focal distance. The latter accordingly is in inverse proportion to the refracting power of the lens, and can hence be utilized as a measure for it. A principal focal distance of one metre is assumed as unity, and the lens which has this principal focal distance is called a metre lens, and its refractive power a *dioptry* (D). A lens of double this strength (2D) will have half the focal distance, or 50 cm., one of 4D a focal distance of 25 cm. ($=100$ cm. $\div 4$). On the other hand, a lens of 0.5 D refractive power has a focal length of 100 cm. $\div 0.5 = 200$ cm. The focal distance of a lens of nD is thus 100 cm. $\div n$.

The indicated strength of a *cylinder* is the refractive power of the strongest refracting meridian, i. e., that which is at right angles to the axis. Thus a cylinder of $+2$D is one in which this meridian has the same refractive power as a $+2$D spherical lens. The axis, on the contrary, has no refractive power; i. e., in the plane of its axis the cylinder acts like plane glass.

10

The following table will be convenient not only in determining the dioptric strength (D) of a lens of a focal distance of *f* centimetre and vice versa, but also as giving the number of dioptries of accommodation (D) used in focusing on an object *f* centimetres from the eye (see § 180).

D	*f*	*D*	*f*	*D*	*f*
0.12	800			6.75	14.8
0.25	400			7.00	14.3
0.37	267			7.25	13.8
0.50	200			7.50	13.3
0.62	160	3.12	32.	7.75	12.9
0.75	133	3.25	30.8	8.00	12.5
0.87	114	3.37	29.6	8.50	11.8
1.00	100	3.50	28.6	9.00	11.1
1.12	88.8	3.62	27.6	9.50	10.5
1.25	80.0	3.75	26.7	10.	10
1.37	72.7	3.87	25.8	10.50	9.5
1.50	66.7	4.00	25.	11	9.1
1.62	61.5	4.12	24.2	12	8.3
1.75	57.1	4.25	23.5	13	7.7
1.87	53.3	4.37	22.9	14	7.1
2.00	50.	4.50	22.2	15	6.7
2.12	47.1	4.75	21.	16	6.3
2.25	44.4	5.00	20.	17	5.9
2.37	42.1	5.21	19.1	18	5.6
2.50	40.	5.50	18.2	19	5.3
2.62	38.1	5.75	17.4	20	5
2.75	36.4	6.00	16.7		
2.87	34.8	6.25	16.		
3.00	33.3	6.50	15.4		

The refractive power of a lens depends on the optical density (index of refraction) of the material composing it and on its curvature. Furthermore when two or more lenses are placed in front of one another, their combined refractive power will depend a good deal on their *degree of separation.* Thus when a convex and concave lens of equal strength are placed in absolute contact, the combination acts like plane glass; when they are separated a little distance it produces an appreciable refractive effect. And in applying glasses to the eye (by which we combine the refractive power of the latter with the refractive power of the glass) the same thing obtains. Generally speaking, the effect of concave glasses is weakened and of convex glasses is strengthened the farther they are held from the apex of the cornea (see § 173).

157. Combination of Glasses.—1. Two spheres of the same sign make a sphere equal to their sum; e. g., −3.00 sph. ○ −2.00 sph. = −5.00 sph.

2. Two spheres of opposite signs make one sphere equal to the difference of the two; e. g., −3.00 sph. ○ +4.00 sph. = +1.00 sph.

3. The above rules apply to cylinders when both have the same axis; e. g., + 1.00 cyl. axis 90° ○ +2.00 cyl. axis 90° = +3.00 cyl. axis 90°; and +1.00 cyl. axis 90° − ○ 2.50 cyl. axis 90° = −1.50 cyl. axis 90°.

4. Two cylinders of the same sign and same maximum strength placed at right angles to each other make a sphere of the same sign and same strength; i. e., +2.00 cyl. axis 90° ⊂ +2.00 cyl. axis 180° = +2.00 sph.

5. A cylinder by the addition of a sphere of equal strength and opposite sign is converted into a cylinder of opposite sign and with its axis at right angles; i. e., +1.00 cyl. axis 90° ⊂ −1.00 sph. = −1.00 cyl. axis 180°.

6. Two cylinders, on which one (a) is plus, the other (b) is minus, when crossed at right angles, make either a plus sphere equal in strength to a and combined with a minus cylinder equal in strength to $a+b$ and having the axis of b; or they make a minus sphere equal in strength to b and combined with a plus cylinder equal in strength to $a+b$ and having the axis of a. Thus +2.00 cyl. axis 90° ⊂ −3.00 cyl. axis 180° = either +2.00 sph. ⊂ −5.00 cyl. axis 180° or = −3.00 sph. ⊂ +5.00 cyl. axis 90°.

These rules are not strictly applicable unless the glasses that are combined are in absolute contact.

158. Analysis of Glasses.—We are very often called on to determine the precise strength and character of the glass a patient is wearing. To do this we first ascertain whether it is convex or concave. This we do by means of the parallactic displacement. This is the displacement produced in the apparent position of an object when we look at it through a lens and move the lens to and fro. This takes place as follows. An object that we look at through the centre of a convex lens appears in its natural place, but if we move the lens down so as to see the object through the upper part of the lens, the object will appear displaced upward (§ 152), i. e., will move in a contrary direction to the lens. Per contra, an object that we see through a concave lens will appear to move in the same direction that we move the latter. If in either case we place in contact with the lens another of equal strength and opposite sign, we shall neutralize the convexity of the one with the concavity of the other, so that the combination has the effect of plane glass and now an object seen through it no longer moves when it moves.

A *cylinder* when moved in a direction at right angles to its axis causes a parallactic movement like a convex or concave spherical glass. When moved in the direction of its axis it produces no movement, because along its axis it acts like plane glass. A cylinder also distorts objects seen through it. Hence a cross seen through a cylinder will look skewed (Fig. 66 I). If now the cylinder is rotated, the cross will become straight whenever either arm of it coincides with the axis of the cylinder (Fig. 66 II and III).

The presence of a *prism* is shown by a displacement of the arms of the cross, which, however, always remain parallel to their original position (Fig. 67 I). By rotating the glass we can ascertain the direction of the prism and the amount of prismatic displacement (Fig. 67 II and III).

Accordingly, to *analyze a glass*, we look through it at a cross, rotate the glass until the cross seems no longer skewed (Fig. 66 II), and add a sphere of opposite sign till all movement in the direction AB is abolished. Leaving this neutralizing glass in place, we move the lens in the direction CD, and, according to the direction of the parallactic movement, neutralize this also with a convex or concave cylinder having its axis placed in line with AB.

The combined addition with reversed signs will equal the sphero-cylinder in the glass. If in addition there is a prism as shown by a displacement of the cross arms like that in Fig. 67, we determine its strength by neutralizing it with a prism placed in the opposite direction and of such strength that it will abolish the displacement.

It is often important to determine the *optical centre* of a glass. If on looking through the glass we see the cross arms skewed (Fig. 66 I) we

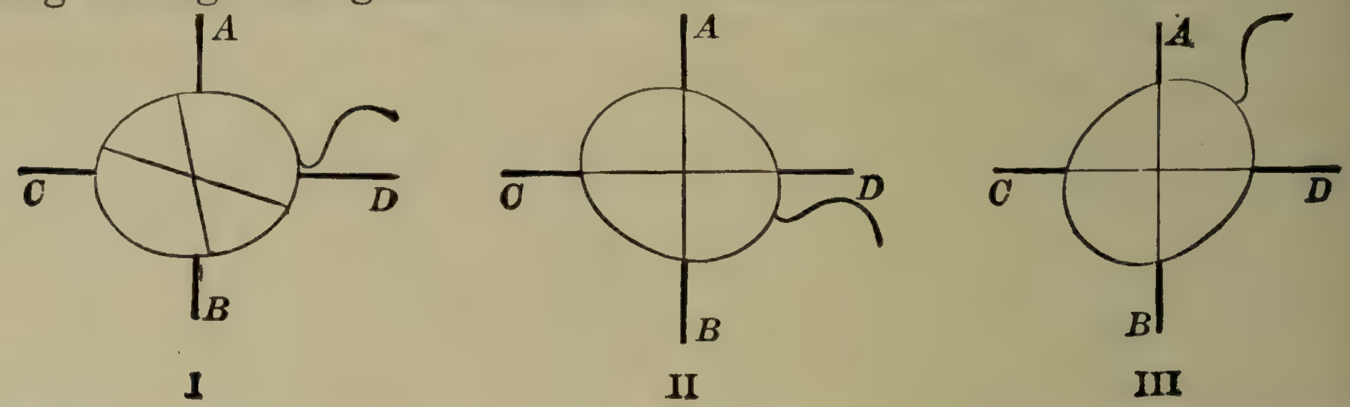


FIG. 66.—DISTORTION PRODUCED BY A CYLINDER. DETERMINATION OF AXIS AND OPTICAL CENTRE. (From Posey and Wright.)

A right-angled cross, *AB-CD*, is seen through a glass containing a cylinder. If (I) the axis of the cylinder does not coincide with either, *AB* or *CD*, the cross appears skewed, so that the arms no longer make a right angle. The cross, however, is not displaced as a whole either to one side or the other (cf. FIG. 67). If now the glass is rotated until the axis of the cylinder coincides with one arm *AB* of the cross, the cross will appear right-angled and unbroken (II). The same thing will happen if the glass is rotated 90° further (III), so that the axis of the cylinder coincides with *CD*. In both II and III the point on the glass where the two arms *AB* and *CD*, being undisplaced, appear to intersect is the optical centre.

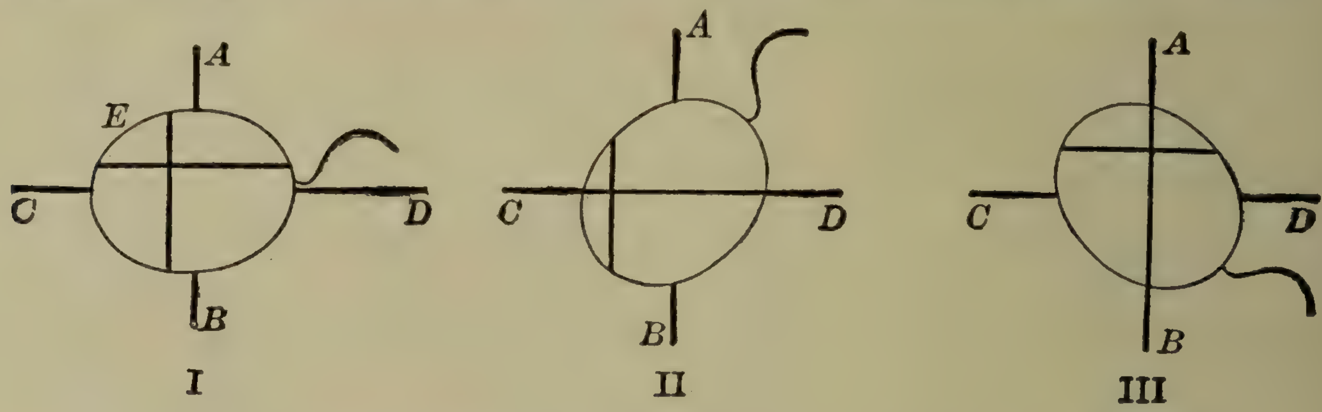


FIG. 67.—DISPLACEMENT PRODUCED BY PRISM. (From Posey and Wright.)

A right-angled cross, *AB-CD*, is seen through a glass containing a prism. (I) The apex of the prism does not lie in line with either arm of the cross, being, in fact, directed toward *E*. Both arms of the cross appear displaced bodily, but neither is skewed. The amount of displacement of *AB* indicates the lateral effect, and the displacement of *CD* the vertical effect of the prism when in this position. If now the glass is rotated, both arms will appear to shift, but each will always remain parallel to its original position. (II) The glass has been rotated until *CD* appears unbroken, while *AB* is displaced toward *C*. The amount of its displacement now represents the total strength of the prism, and the apex of the prism points toward *C*. (III) The glass is rotated 90°. Now the line *AB* appears unbroken, and *CD* is deflected toward *A*. The apex of the prism points toward *A*, and the amount of deflection, of *CD* corresponds to the total strength of the prism. A prism placed with its base at *A* and neutralizing the displacement *CD* measures the strength and gives the direction of the prism in the glass.

rotate the glass till they are so no longer, and then if the arms are displaced, as in Fig. 67 I, we slide the glass up and down or in and out until the cross is unbroken (Fig. 66 II), and mark on the glass the point where the two arms seem to intersect. This will be the optical centre.

159. Bifocal Glasses.—It is frequently the case that a person needs glasses both for distant and near vision, but of a different strength. This is especially true of presbyopes. The glass for near work in those who wear concave glasses must be less concave or actually be convex, in those who wear convex glasses must be more convex than the distance glass. Hence, in order to avoid changing the glasses constantly, spectacles have been con-

structed which have a stronger refractive power in the lower part than in the upper. The first person to make such a glass was Franklin who constructed it for his own use by joining together two half lenses in such a way that they were in contact along a horizontal line (Fig. 68 A). Since the horizontal dividing line causes a good deal of confusion when the gaze is directed straight forward, such glasses are now constructed so that the frame contains the glass designed for distance, and upon the lower part of this is cemented a paster, or small semilunar convex lens (bifocal glass; see Fig. 68 B and C). In near vision, in which the visual plane is depressed, the eye looks through the paster, while for distant vision the upper part of the glass is used. Instead of cementing the segment upon the distance glass, it may be fused or ground into the latter (fused bifocal, kryptok). Trifocal glasses have also been prescribed.

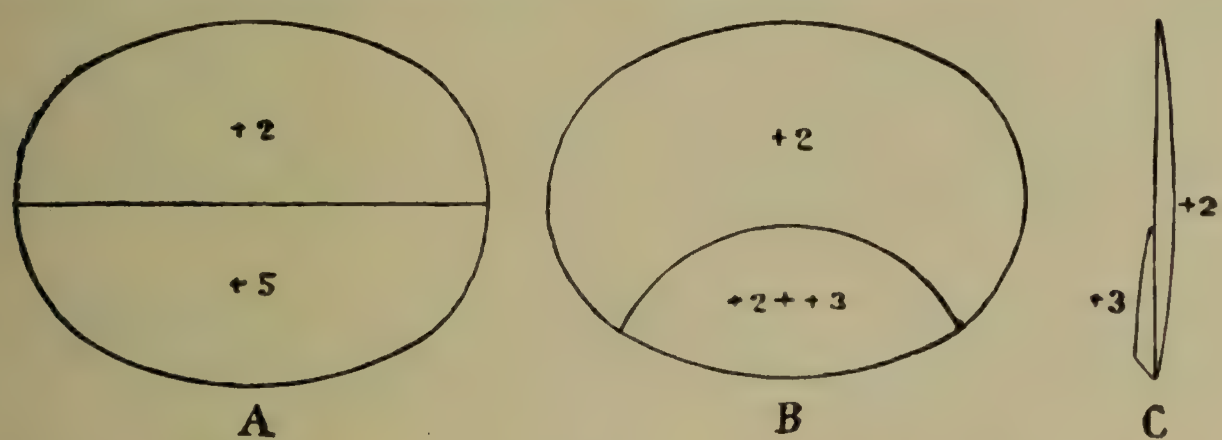


Fig. 68.
A, Franklin glass, B and C, bifocal glass for an old hypermetrope, who no longer has any accommodation and hence needs for near work a glass 3 D stronger than for distance.

160. Stenopæic Spectacles.—Stenopæic[4] spectacles consist of a blackened disc of metal, in which is placed a small round hole or a narrow slit. They are sometimes employed with advantage in cases of corneal opacities. They are adapted for those cases in which a part of the pupillary area of the cornea is clear, while another part is occupied by a semitransparent spot of cloudiness, which by diffusing the light causes confused vision. If the disc is held before the eye in such a way that the aperture lies in front of the transparent portion of the cornea, the latter alone is used for seeing, and the opacity which causes the confusion is excluded. Since a man has only a very small field of vision in looking through such an aperture, and since, moreover, he can not move his eye about behind it, stenopæic spectacles are not suited for going about, but they often enable the patient (who can best manage them by holding them in his hand) to read when it would be impossible to do so in any other way. (Cf. also § 168.)

For *protective glasses* and *eye-guards* see §§ 114, 115.

[4] From στενός, narrow, and ὀπή, peep-hole.

CHAPTER XIII

GENERAL OPTICAL PROPERTIES OF THE EYE

I. The Normal Eye

161. Optical System of the Eye.—By the refraction of the eye we mean its optical adjustment when in the state of rest—i. e., in the absence of any accommodative effort. The refraction depends on the situation of the retina, on the one hand, and the character of the refracting media, on the other.

The refractive media of the eye comprise the cornea, which may be regarded as a convexo-concave lens, the aqueous, which has nearly but not quite the same optical density as the cornea, the crystalline lens, which presents a series of refracting layers increasing in general in optical density from the periphery to the centre of the lens, and the vitreous, which has the same optical density as the aqueous.

The system formed by the combination of these various refractive surfaces and media has like other systems six cardinal points (see § 153). An average situation of these in an eye with normal refractive media may be stated as follows:

First principal focus	14.02 mm. in front of cornea.
First principal point	1.78 mm. behind anterior surface of cornea.
Second principal point	2.13 mm. behind anterior surface of cornea.
First nodal point	7.12 mm. behind anterior surface of cornea.
Second nodal point	7.47 mm. behind anterior surface of cornea.
Second principal focus	23.27 mm. behind anterior surface of cornea.

The first focal distance (distance of first principal focus from first principal point) is thus —15.80 mm., and the second focal distance (distance of second focus from second principal point) is 21.14 mm.

Since the two nodal points are so close together, they may in most cases without sensible error be regarded as one, called simply the nodal point of the eye and situated 15.80 mm. in front of the retina, i. e., just about at the posterior pole of the crystalline lens. In that case the principle enunciated in § 153 may be thus stated: To find the position of the retinal image b of a point o, we draw a line from o through the nodal point, k, of the eye. The point where this line strikes the retina will be the site of b, (see Fig. 69). This principle enables us to solve all sorts of optical problems concerning the eye. In particular it enables us to calculate the size of the retinal image of a given object. To do this the size of the object and its distance from the eye must be known. We find the retinal image of an object by drawing from the terminal points, o, o_1 (Fig. 69), of the latter lines through the nodal point, k, to the retina, which they meet in b and b_1. Accordingly, $b\,b_1$ is the retinal image of the object $o\,o_1$. The triangles $o\,o_1 k$ and $b\,b_1 k$ are similar; hence, $b\,b_1 : o\,o_1 = b\,k : ok$, and so $bb_1 = \dfrac{o\,o_1 \times b\,k}{o\,k}$. If we call the size of the images (bb_1) B, that of the object ($o\,o_1$) O, and the distance of the latter ($o\,k$) from the eye E, then $B = \dfrac{O \times 15.8 \text{ mm}}{E}$. The size of the retinal image is therefore directly proportional to the size of the object and inversely proportional to the distance of the object from the eye, e. g., a rod 1 metre high placed at a distance of 15 metres from the eye would give a retinal image whose altitude $B = \dfrac{1{,}000 \text{ mm.} \times 15.8 \text{ mm.}}{15{,}000 \text{ mm.}} = 1.05$ mm. This rod, if

approximated to one-third of the distance—i. e., 5 metres—would give a retinal image of $\frac{1,000 \text{ mm.} \times 15.8 \text{ mm.}}{5,000 \text{ mm.}} = 3.16$ mm. or three times as great as before. This sort of calculation is often applied in order to discover the size of a diseased area of the retina corresponding to which is a scotoma in the visual field, whose size can be determined, by examination. The scotoma is then regarded as the object for which the size of the retinal image is calculated.

The relation above given holds good whether we regard the nodal points as fused or as separate. Furthermore, it is obvious from the figure that if k approaches the retina ($o\,o_1$ remaining the same in size and position) $b\,b_1$ will become smaller. In other words, the size of the retinal image and hence the *apparent size of the object* that corresponds to it diminishes in proportion as the second nodal point approaches the retina.

162. Part Taken by the Different Media in Refraction.—A large part of the refraction of the eye is performed by the cornea and aqueous. The lens serves to supplement to a considerable degree the action of the cornea and also helps to correct the astigmatism and aberration due to the latter.

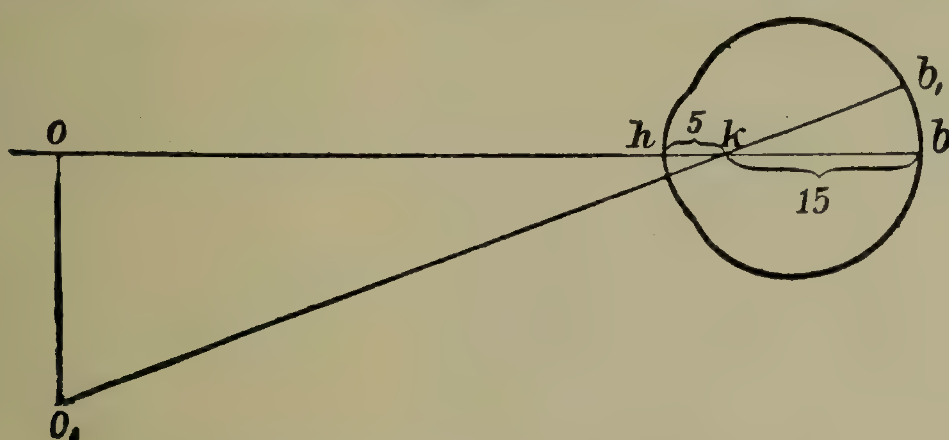


FIG. 69.—NODAL POINTS OF EYE REDUCED TO ONE.

The figure represents what is called the reduced eye of Donders, which is supposed to consist of a single refracting substance with an index of refraction of 1.33 and bounded by a refracting surface, whose centre of curvature (nodal point of system) is 5 mm. posterior to the surface and 15 mm. in front of the retina.

Furthermore by its ability to change its shape it effects the accommodation of the eye (see § 177)[1]

As an optical instrument the eye may be said to consist of a nearly rigid, spherical capsule with a bull's-eye lens (cornea) in front, the capsule being filled with a liquid of uniform optical density (aqueous and vitreous), in which is suspended a lens of adjustable focus. Between the bull's-eye lens in front and the suspended lens behind is stretched a diaphragm (iris) which acts as a stop to cut off the peripheral, irregularly refracted rays. This stop works automatically, its aperture (pupil) contracting whenever the illumination gets too intense and dilating when the illumination becomes feeble (see § 365).

The lens consists of strata varying in optical density from depth to depth. If in such a structure we connect all the points whose index of refraction is equal we get what is called an *isoindicial surface.* Gullstrand has determined the situation of the isoindicial surfaces of the lens (see Figs. 70, 71). From his researches confirming those made years ago by Young it appears that the refractivity of the lens increases from the periphery to the centre, the change taking place rapidly at first, more slowly afterward.

163. Visual and Fixation Lines.—The optic axis (centric line) of the eye is the line connecting the centres of curvature of its refracting surfaces[2]. It is perpendicular to the cornea at the point where it pierces the latter and passes through the two nodal points. It pierces the retina usually some

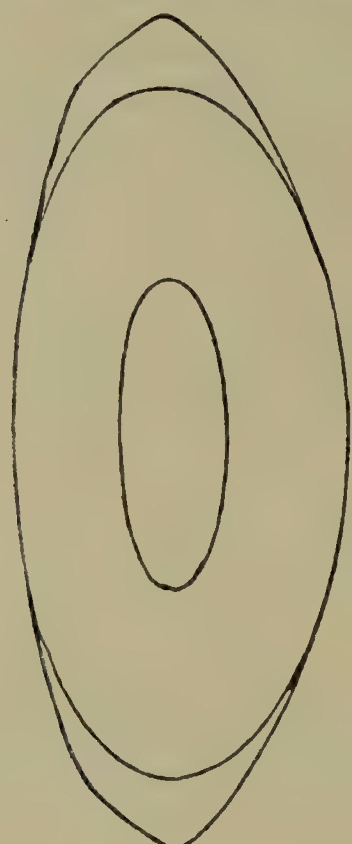
[2] The refracting surfaces of the eye probably are not quite centred (see § 153), but yet are so nearly so that without essential error we may say that the eye has a true centric line or optic axis.

distance to the inner side of the macula. Roughly speaking, it passes through the centre of the pupil. See Fig. 72, $ACK'K''$.

The *visual line* is the line connecting the object of fixation O (Fig. 72) with the fovea F, or, more strictly speaking, it is the two, nearly coincident parallel lines, OK', $K''F$, one drawn from the object of fixation to the first nodal point K', the other from the second nodal point K'' to the fovea.

The *fixation line* is the line OM connecting the object of fixation with M, the centre of rotation of the eye.

The angles formed by these lines have been variously denoted (Percival). Thus the angle α of Donders (angle β of Brubaker) is the angle $AK'O$ between the visual line and



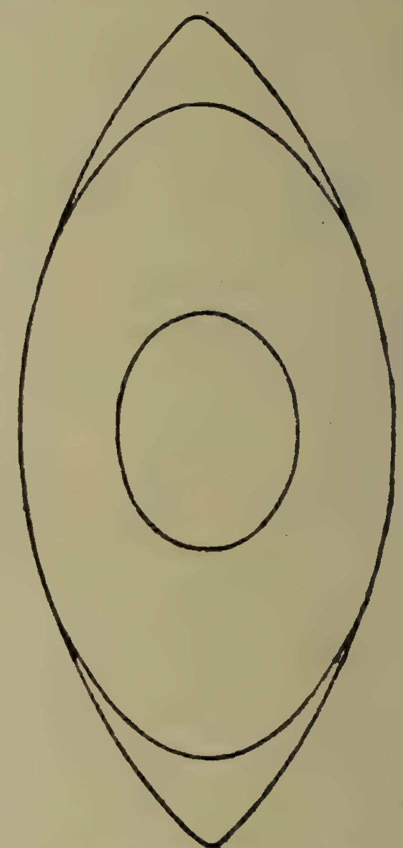


FIG. 70. FIG. 71.
ISOINDICIAL SURFACES OF THE LENS (after Gullstrand).

FIG. 70.—LENS AT REST.—The two regular ovals represent the limits of the isoindicial surfaces, the outer one connecting points having an index of refraction of 1.386, the inner connecting points having an index or refraction of 1.404. The irregular figure above and below shows in an approximate way the outline of the portion of the lens not concerned in refraction. The rounded projection on the front of this portion shows the point of attachment of the suspensory ligament.

FIG. 71.—SAME SURFACES IN THE ACCOMODATING LENS.

centric line (optic axis). (The angle a of Landolt is the angle OXE between the visual line and the major axis EL of the cornea regarded as an ellipsoid. As the cornea is now known not to be an ellipsoid, Landolt's angle has lost its significance). The angle γ is the angle AMO between the fixation line and the optic axis. Nearly, but not quite identical is the angle between the fixation line and the perpendicular to the cornea passing through the centre of the pupil (angle κ)

164. Emmetropia.—In an eye having the cardinal points given in § 161, a sharp inverted image of a distant object will be formed at the second focus of the system, i. e., at a point 23.14 mm. behind the cornea. If the percipient elements of the retina are situated here, these will receive a distinct image and hence a precise representation of the object. The eye is then said to be emmetropic. Emmetropia[3] (E, Donders) then, is the condition in which the curvature and density of the various refractive media

[3] From ἔμμετρος, in due measure, and ὤψ, sight.

are such that rays coming from a distant object can be focused sharply on the retina. The image in that case will be clearly perceived, provided the refractive media are transparent and the retina itself has good sensory power.

II. Ametropia

165. Varieties.—All refractive conditions other than emmetropia are grouped under the name ametropia.[4] Ametropia, in other words, is the condition in which rays coming from a distant object cannot be accurately focused upon the retina. Its varieties are as follows:

Myopia[5] (*My*). This is the condition in which the percipient screen (the retina) is beyond the principal (second) focus of the eye, so that the refracted rays emanating from a distant object (parallel rays) come together before they reach the retina. (See Fig. 78.)

Hypermetropia[6] or *Hyperopia*[7]. This is the condition in which the retina lies in front of the principal focus, so that parallel rays are brought to a focus behind it (at *f*, Fig. 79).

Astigmatism or *Astigmia*[8] (*As*). This is the refractive condition of the eye in which parallel rays falling upon the latter are not at any spot brought to a common focus.

Anisometropia[9] is the condition in which the refractive state of the two eyes is different.

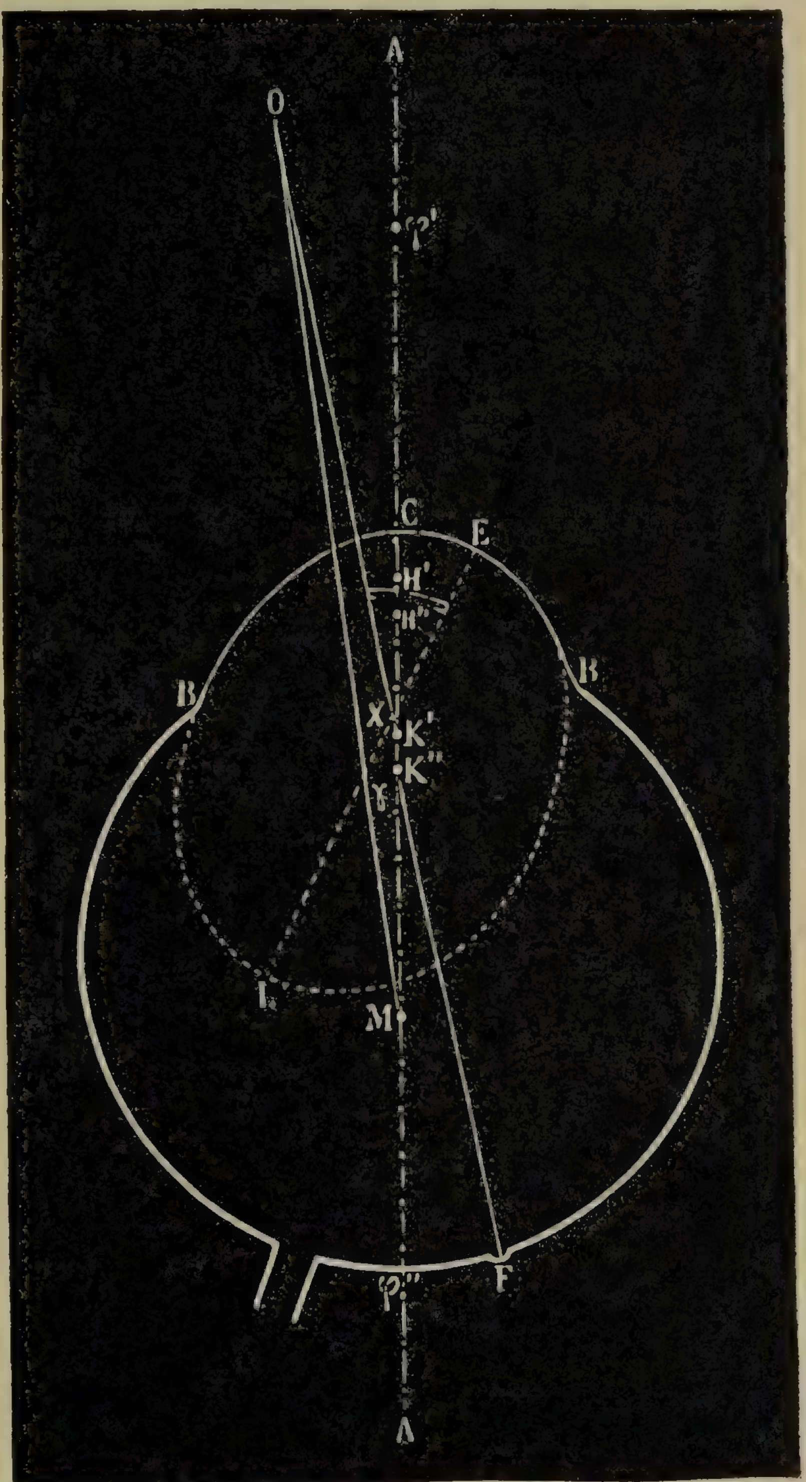


Fig. 72.—Visual and Fixation Lines. (After Landolt.) *O*, object of fixation. *K'*, first and *K''* second nodal point *M*, centre of rotation of eye. *F*, fovea. *ACK'K''*, optic axis. *OK'K''F*, visual line. *OM*, fixation line.

Eccentricity.—The distance between the retina and the second focus of the eye may be called the eccentricity (*e*). The greater it is, the greater the ametropia. Indeed, as we shall see, its linear amount bears a direct and simple relation to the strength of the glass correcting the ametropia. The eccentricity may be regarded as positive in myopia, negative in hyperopia.

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4 From ἀ, privative, μέτρον, a limit, and ὤψ, sight.

5 From μύειν, to shut, to blink, and ὤψ, sight, on account of the habit that myopes have of closing the lids together so as to increase the distinctness of the images (see § 168).

6 From ὑπέρ, in excess of, μέτρον, measure, and ὤψ sight.

7 From ὑπερ and ὤψ, sight.

8 From ἀ privative and στίγμα, point.

9 From ἀ, privative, ἴσος, equal, and μέτρον, measure, and ὤψ, sight.

166. Varieties of Astigmatism.—Astigmatism comprises two main varieties:

1. *Latitudinal Astigmatism* (aberration) in which rays passing through the centre and the periphery of the cornea in any one meridian of the latter are focused at different points. This sort of astigmatism is remedied by the contraction of the pupil, which cuts off the peripheral rays, and also by the compensatory action of the crystalline lens.

2. *Longitudinal astigmatism* (astigmatism in the ordinary sense of the word), in which each successive meridian of the eye varies in refractive

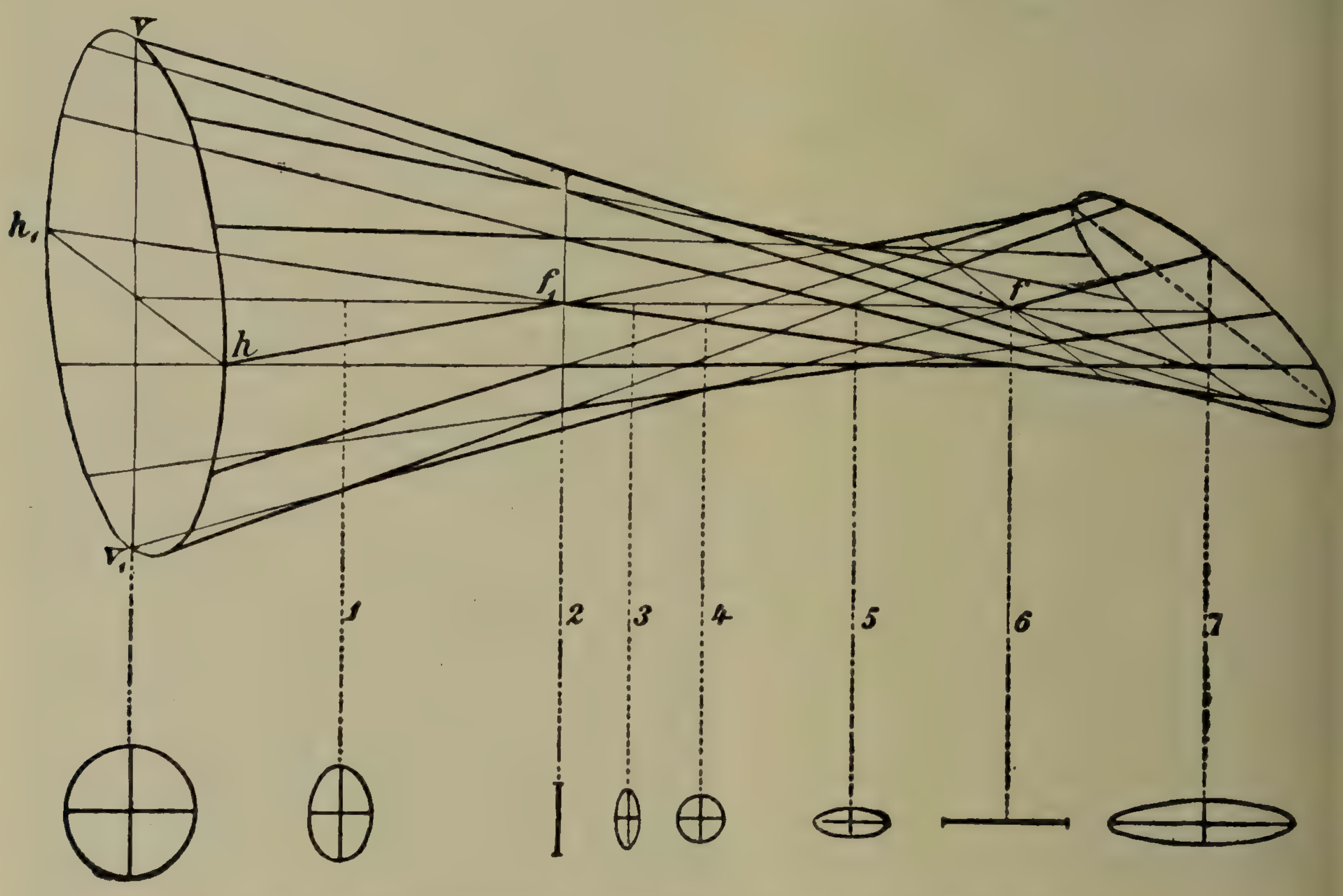

FIG. 73.—REFRACTION OF THE RAYS IN REGULAR ASTIGMATISM.

power. Astigmatism of this sort is called *regular* or *irregular* according as the change in refraction from one meridian to another takes place by uniform increments or not. In the former case there is usually one meridian, called a principal meridian, in which the rays are focused fairly well at a point, f_1, (Fig. 73), and another principal meridian, at right angles to the first, in which the rays are focused at a point, f, further back. Rays passing through the other meridians are not focused sharply at all. Such an eye, in fact, refracts the rays very much as a cylindrical lens does (see § 150).

The relation indicated in Fig. 73, in which the horizontal meridian is the most refractive and the vertical meridian the least so (*inverse* astigmatism, astigmatism *against the rule*) is less common than the reverse state (*direct* astigmatism, astigmatism *with the rule*). It may also happen that the principal meridians are neither vertical nor horizontal, in which case the astigmatism is called *oblique*.

Of regular astigmatism there are the following varieties, produced by variations in the situation of the retina:

1. *Compound hyperopic* astigmatism. Retina situated in front of f_1, (e. g. at *1*). All meridians are hyperopic, but one (in this case the horizontal one) is least so.

2. *Simple hyperopic* astigmatism. Retina situated at f_1. One meridian (the horizontal) is emmetropic; the rest are hyperopic.

3. *Mixed* astigmatism. Retina situated between f_1, and f (e. g. at *3*, *4*, or *5*). One principal meridian (the horizontal) is myopic; the other (the vertical) is hyperopic.

4. *Simple myopic* astigmatism. Retina at f. One meridian (the vertical) is emmetropic; all the others are myopic.

5. *Compound myopic* astigmatism. Retina back of f (e. g. at *7*). All meridians myopic, but one (in this case the vertical) least so.

The essence of astigmatism consists in the fact that the eccentricity of the eye is different in the two principal meridians. If the retina is at *1*, the eccentricity for the horizontal meridian is represented by the distance *1-2*, that of the vertical meridian by *1-6;* if the retina is at f_1, the eccentricity of the horizontal meridian is zero, that of the vertical meridian is *2-6*, etc. In the case shown, however, no matter where the retina lies, the difference between the eccentricity of one principal meridian and the other—which difference is the measure of the astigmatism—is the same, being equal to the distance between f_1 and f. That is, we have not changed the amount of the astigmatism by shifting the retina from *1* to *7*, but have simply changed its character. This sort of transformation occurs very frequently in the course of development. The elongation of the eye, which regularly takes place (see § 191), causes a gradual recession of the retina, which converts a hyperopic into a mixed, and finally into a myopic astigmatism—the amount of the astigmatism remaining unchanged during the process.

The space $f_1 f$ is called the focal interval. Its linear amount bears the same relation to the strength of the glass correcting the astigmatism that the eccentricity bears to the strength of the glass correcting a hyperopia or myopia (see § 173).

167. Axial, Curvature, Index and Positional Ametropia.—The eye has been aptly compared to a camera, and the ametropic eye is like a camera that is badly focused. As in the case of a camera, bad focusing may be due either to the fact that the receptive screen (in this case the retina) is out of place, or to the fact that the refractive media (corresponding to the camera lens) are improperly constructed. We hence distinguish four varieties of ametropia.

1. *Axial* ametropia. In this the refractive system of the eye is normal i. e., its cardinal points are essentially as given in § 161, but the retina is too far forward or back. This form comprises the great bulk of cases of ordinary hyperopia and myopia.

2. *Curvature* ametropia. In this the retina is in the proper place, but the refractive surfaces of the eye (surfaces of cornea and lens) are too much or too little curved, so that the cardinal points of the eye differ essentially from those given in § 161. Some kinds of acquired hyperopia and myopia and nearly all kinds of regular as well as most kinds of irregular astigmatism are instances of curvature ametropia.

3. *Index* ametropia. In this the eye refracts too strongly or too little because the refractive media, although having the proper surface curvature,

are too dense or not dense enough. Index ametropia comprises some kinds of acquired hyperopia and myopia and some forms of astigmatism (especially lenticular astigmatism).

4. *Position* ametropia. In this ametropia is due to the fact that the lens is not in its normal situation, being either too near the cornea or too far from it.

If in an eye otherwise normal the lens is carried bodily forward, the second focus of the eye is carried forward somewhat too, so as to be now in front of the retina. That is the eye has been rendered somewhat myopic. The second nodal point of the eye is also carried forward slightly. Its distance from the retina is thus increased, thereby making the size of the retinal image and hence the apparent size of the object seen, somewhat greater (see § 161). If the lens is carried backward, a certain amount of hyperopia is produced and the apparent size of the objects is slightly diminished.

Actual dislocation of the lens is usually associated with considerable refractive changes, but these are due either to actual disappearance of the lens from the pupillary area (aphakia—see § 193) or to changes of lens curvature (see § 662).

168. Vision in Ametropia; Diffusion Images.—Bad focusing in the eye —ametropia—acts just like bad focusing by a lens. It produces *diffusion images*, which in the case of the eye cause the blurred vision that is the mark of ametropia. The way diffusion images are formed is as follows:

The eye may be regarded as a lens with a screen (retina) and an aperture (pupil). Usually this aperture is circular. In the case of a non-astigmatic eye, parallel rays coming from a distant point and impinging on the eye are converged by the cornea and, passing through the pupil, are converged still more by the lens so as to be brought to a sharp focus at the second focal point of the eye (regularly at a point 23.27 mm. behind the cornea). These refracted rays thus form a conical sheaf, the base of the cone being the pupil, the apex the second focus of the eye. If the eye is hyperopic, the retina is interposed as a screen between the base and apex of the cone (Fig. 79), thus cutting off a circular section of the latter, which represents the retinal image of the distant object. The rays, then, instead of being concentrated into a sharp punctate image on the retina, form a circle there (diffusion circle). The larger the circle the dimmer it is, because all the light that in the case of a sharp image would be concentrated into a single point is now spread out over a wide area.

Precisely the same thing happens in myopia. The conical sheaf of rays emanating from a distant object ($\infty \infty$, Fig. 78) come to a sharp focus in the interior of the eye, and then begin to diverge again so as to form another conical sheaf which is intercepted by the retina (at aa_1) forming thus a circular diffusion image.

Why vision should be rendered indistinct by diffusion circles is easy to understand. Suppose that there are two points so far distant from each other that when the eye is accurately focused they appear as two separate punctate images upon the retina (Fig. 74 A); the points are then readily recognized as two. But if, in consequence of the eye's being incorrectly focused for the position of either point, a diffusion circle is formed upon the retina, the two circles, provided they are but a short distance from each

ther partly overlap (Fig. 74 B), and the eye imagines that it has before it nly one elongated point. A line (Fig. 74 C), when seen in diffusion circles, loes not look distinct, but appears broadened and hazy; for we may coneive a line to be composed of an infinite number of points placed side by ide, and if each one of these is seen as a diffusion circle, and the circles to a reat extent overlap (D), the narrow line is converted into a broad band (E).

Vision, then, is always in diffusion images when the eye is not properly ocused for the object of fixation. In fact, whatever indistinct vision is aused by an anomaly of refraction or accommodation is caused by difusion images. The larger the diffusion mages are, the more indistinct is the vision. We must therefore inquire upon what the *size* of the diffusion images depends:

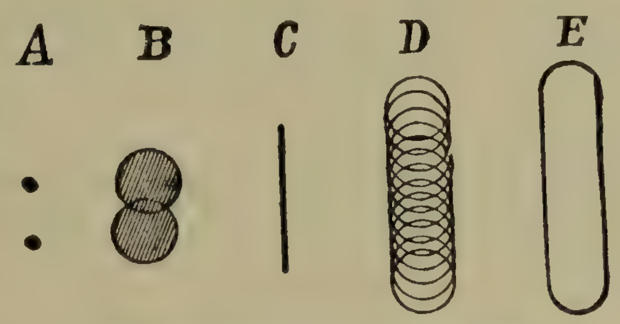

Fig. 74.—Diffusion Circles.

1. The diffusion images get larger in proportion as the focus of the rays emanating from the object gets further from the retina. For, the further from the apex of the cone of rays the intersection of this cone by the retina is, the greater will be the size of this intersection—namely, the diffusion image. It is easy to see from Fig. 79 that the shorter the hyperopic eye, i. e., the greater its excentricity, the larger and hence dimmer the retinal image will be.

The other factor which influences the size of the diffusion image is—

2. The width of the pupil. The pupil forms the base of the cone of rays; the smaller it is the smaller will be the section of the cone, supposing the distance of this section from the apex to remain the same. When one of two equally near-sighted persons see better at a distance with the naked eye than the other does, it is owing to the fact that the former has narrower pupils. Short-sighted persons often believe that they become less near-sighted with increasing years because they see better at a distance; but this is often simply due to the circumstance that their pupils diminish in size with age. Far-sighted persons who are compelled to read close to them without convex glasses try to get as brilliant an illumination as possible, so that their pupils may become very greatly contracted and thus diminish the size of the diffusion images. The same object is secured to a still greater extent by placing a fine stenopæic aperture before the eye. This allows only a very narrow beam of rays to pass, and reduces the diffusion images so greatly that they no longer exert a disturbing effect. By means of a stenopæic aperture myopic persons can see distinctly at a distance even without concave glasses.

The pupil, being the base of the cone of light, determines not only the size but also the *shape* of the diffusion image, which accurately mirrors the shape of the pupil. In this way it happens that persons with irregular pupils (owing, for example, to posterior synechiæ) are very well able themselves to perceive these irregularities entoptically.

Diffusion images due to refractive errors cause not only blurred vision but often also *monocular diplopia*, the letters on the test-card, for example, appearing double (see § 171).

The statements above made serve to explain in a general way the behavior of diffusion images in simple myopia and hyperopia. In strictness, however, they apply only to rays passing quite close to the axis of the eye. But the eye receives many rays which are remote from the axis and which therefore, even in simple myopia or hyperopia, are affected by aberration (latitudinal astigmatism). Apart from this, nearly all eyes have some longitudinal astigmatism too. Hence for the full consideration of diffusion images in ametropia we must refer to the next section.

169. Diffusion Images in Astigmatism.—Rays that pass through a convex lens near its periphery are brought to a focus sooner than those that are near the centre. When owing to this cause rays fail to focus in one point, the condition is called aberration (latitudinal astigmatism). It produces a blurring of the image, which is greater in proportion to the number of the peripheral rays that are allowed to pass. Such a condition occurs in all eyes, even in simple myopia and hyperopia. Its effect increases with the width of the pupil. When the pupil contracts to about 3 mm., so as to include only the centric or visual area about the visual axis, there is very little aberration. When the pupil is widely dilated, the refraction near its periphery, as skiascopy shows, usually differs appreciably from that of the centric area so that the aberration is well marked.

Longitudinal astigmatism (astigmatism in the ordinary sense) is due to the fact that successive meridians of the eye differ in refractive power. Astigmatism of this sort is seated both in the cornea and lens, but as a rule mainly in the former (see § 206). As showing how diffusion images are formed in this sort of astigmatism we may take the case in which the cornea is its seat. In Fig. 73, let $v\ h\ v_1\ h_1$ represent the circumference of the cornea, and $v\ v_1$ its vertical meridian, which has a curvature such that the rays passing through it come to a focus at f. In the meridian immediately adjoining, the curvature becomes a little greater, and in the succeeding meridians it increases still more, so that it reaches its maximum value in the horizontal meridian, $h\ h_1$. The rays passing through the latter may be supposed to intersect at a point as near as f_1. In this case we would have one meridian (the horizontal one) which refracts rays most strongly, and one perpendicular to it (the vertical meridian) which refracts most feebly; and corresponding to these are the most anterior and posterior foci, f_1 and f. These two meridians thus distinguished from the others are called principal meridians; those meridians lying between them represent all intermediate stages of curvature and refractive power[10]. We see that when the refracting surface is of this character there is no point at all at which all the rays passing through the surface will unite. The image of a point cast by such a surface upon a receptive screen is therefore not a point, but a diffusion image, the shape of which depends upon the spot where the retina is situated and cuts the beam of converging rays. Let us assume that the retina is at the

[10] This statement is not strictly correct. In no meridians except the two principal ones do the rays come to a focus. Astigmatic refraction, indeed, as Gullstrand has shown, is a very much more complicated process than is here depicted. Nevertheless, the figure does represent fairly well the character of the diffusion images formed by an astigmatic eye and the sort of vision that the latter possesses.

oint, marked 1. Here the rays passing through the horizontal meridian re already brought closer together than those incident upon the vertical ıeridian; hence the section of the sheaf of rays is an erect ellipse. At 2, here the rays of the horizontal meridian come exactly to a focus, the image f the point is a vertical line. In the same way the shape of the cross section f the beam—i.e., the shape of the diffusion image of the point—can be scertained for the more posteriorly situated points from 3 to 7. This cross ection is sometimes an erect or horizontal ellipse, sometimes a vertical or orizontal line, according as it is more or less distant from the refracting urface. It is only at 4 that there is really a diffusion circle, because here he rays passing through the horizontal meridian diverge to the same extent hat those of the vertical meridian converge.

The statements just made in this and the preceding sections give only a rough idea f how diffusion images are formed in the eye. We have given these statements in detail

Fig. 75.—Retinal Images in Regular Astigmatism.
, two lines placed perpendicular to each other; B, their image upon the retina of an astigmatic person.

ecause by and large they do correspond to the clinical facts. But the researches of ullstrand have shown that diffusion images are really more numerous and formed in a ıuch more complex way than here described. Even the emmetropic eye is flooded with ıem, and if they had all the effect ascribed to them clear vision would be impossible. he truth is that a great many of the rays intersect to form a caustic surface, so that the ght in a diffusion image is not uniformly scattered but is concentrated in a peculiar way nd this sort of distribution of light within the diffusion image is much more significant r vision than is the size or shape of the image itself.

170. Vision in Astigmatism.—The vision of an astigmatic person is not imply indistinct, like that of one who is near-sighted or far-sighted, but resents special peculiarities on account of the elongated form of the diffuion images. Circular surfaces—e. g., the full moon—appear elliptical. traight lines sometimes look distinct, sometimes indistinct, according to he direction that they take. Let us assume that we have before us an stigmatic who sees the diffusion image of a point under the form of a ertical line (2, Fig. 73). If this man looks at two lines standing perpendiclar to each other (Fig. 75 A) the horizontal line appears broadened and ıdistinct but the vertical line seems sharply defined. For, we may imagine ıese two lines to be composed of an infinite number of points. Each one f these points appears upon the retina of the astigmatic patient under the rm of a short vertical stroke, and the horizontal line therefore appears nder the form of a series of such vertical strokes, which coalesce and contitute a band of a certain degree of breadth (Fig. 78 B). In the vertical ne the vertical strokes are superimposed and cover each other, so that the

line appears sharply defined. Only the uppermost and lowermost diffusion lines extend beyond the terminal points of the vertical line and make it seem somewhat longer than it is. Thus, for every astigmatic person there is one direction in which straight lines appear most distinct, and one, perpendicular to it, in which they appear most confused.

As the example above given shows, vertical lines appear distinct when the horizontal meridian is emmetropic. At the same time horizontal lines appear indistinct. These lines, on the other hand, would be distinct if the vertical meridian were emmetropic. But the vertical meridian can be rendered emmetropic by adding the glass that corrects its refractive error. In other words, the glass with which a given set of lines is seen distinctly measures the refraction of the meridian at right angles. This fact finds its application in ophthalmoscopy by the direct method. If we look into a highly astigmatic eye with the ophthalmoscope, the vessels at a right angle to the emmetropic meridian of the eye look distinct, those parallel to it look blurred—the whole fundus being drawn out into streaky lines. (For the application of the same fact in the correction of astigmatism see § 214).

The above answers for a general statement of astigmatic vision as clinically observed. It is not, however, strictly accurate. A point of light seen by an astigmatic eye (or refracted by a sphero-cylinder) is not merely drawn out into a line or band, but is converted into a star-shaped figure (Gullstrand).

171. Monocular Diplopia or Polyopia in Refractive Errors.—A person may see double either because the two eyes are not directed properly (*binocular diplopia*, see § 288) or because there is a refractive trouble in one eye by which rays from a simple object are concentrated more or less sharply at two spots on the same retina (*monocular diplopia*). It may even happen that with one eye a person gets multiple images of the same object on one retina (*polyopia*). Binocular is distinguished from monocular diplopia by the fact that it disappears at once if either eye is shut.

Monocular diplopia occurs regularly in patients affected with a comparatively low refractive error, especially astigmatism. Such patients often fail to notice their double vision until examined with the test chart when they see two images of each letter, one faint, the other sharp. In the higher degrees of astigmatism and especially in irregular astigmatism, corneal or lenticular monocular diplopia may be marked and annoying. It occurs particularly in subluxation of the lens and in incipient cataract, being then due to the unequal refractivity of different parts of the lens. In this case, however monocular polyopia is more common (see § 638).

In ametropia of any kind or if the eye is not accommodating for the object of fixation monocular diplopia will occur if there is a *double pupillary opening* (e. g., in iridodialysis). If the eye is properly focused for the object of fixation a double pupillary aperture will not produce monocular diplopia. This fact is accounted for by the well-known experiment of Scheiner. Two holes are made in a piece of cardboard (*D*, Fig. 76) with a needle, the distance between them being somewhat less than the diameter of the pupil, so that, when looked through, both lie at the same time in front of the pupil. Through this apparatus we look at an object—e. g., at a tightly stretched thread, *o*, at a distance of 25 cm. If the eye is focused for this distance, all the rays emanating from the object, *o*, are united upon the retina at the point, o_1. If, now, out of the whole cone of rays only those are transmitted which pass through the two holes, these rays still unite to form an image at o_1; the only change which this image undergoes by having the dia-

hragm placed before it is an enfeeblement of its luminosity due to the cutting off of nany rays. But if the eye is not focused for the distance of the object (Fig. 77), the pex of the cone of rays does not fall upon the retina, but (in this case) behind it at o_1. The cone of rays is cut off in front of its apex by the retina, so that the image of the point , is a disc (diffusion circle), *a*, and the point looks completely blurred. But if now only wo bundles of rays out of the entire cone are admitted to the eye through the diaphragm ach one casts its own smaller diffusion circle (b and b_1); the point, *o*, is now seen more listinctly, it is true, but is seen double.

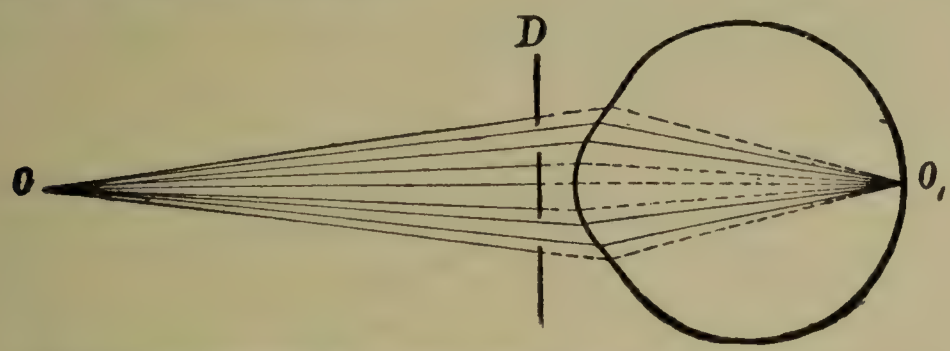

FIG. 76.—SCHEINER'S EXPERIMENT. (The eye is adjusted for the point *o*.)

172. Far Point of the Eye.—In emmetropia rays coming from a disant object—parallel rays—are brought to a sharp focus upon the retina. n other words, the eye is naturally adjusted for far distance.

The point for which an eye is naturally adjusted, i. e., adjusted without ny effort of accommodation or help from glasses, is called its *far point* *R*, punctum remotum, Donders). In an emmetropic eye the far point is at nfinite distance. To such an eye the fixed stars, which, practically speaking, re infinitely far away, are distinctly visible.

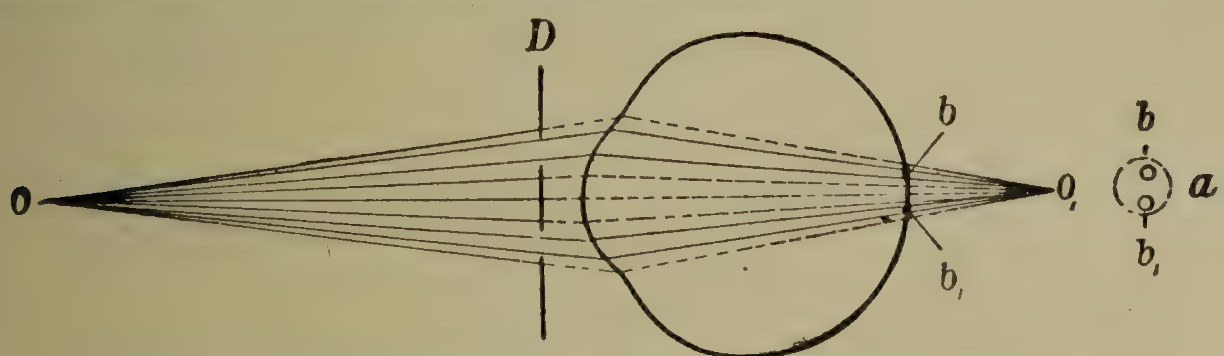

FIG. 77.—SCHEINER'S EXPERIMENT. (The eye is not adjusted for the point *o*.)

A myopic eye is not adjusted for far distance. Relatively speaking, it s too refractive, bringing parallel rays together too quickly, so that they ocus in front of the retina. The rays which it will focus upon the retina nust have a certain degree of divergence as they arrive at the eye, which is he case when they emanate from a point *R* situated close by (Fig. 78). According to definition, this point is the far point. Accordingly, the far oint of the myopic eye lies at a finite distance. The greater the myopia, he farther in front of the retina is the point of intersection of parallel rays nd hence the greater is the divergence that rays must have in order to nite upon the retina, and hence, too, the nearer to the eye must the far oint (*R*) lie. Consequently the degree of myopia is determined by the listance of *R*.

The far point of a hyperopic eye is evidently not at far distance either —for, without exercise of the accommodation, such an eye cannot bring parallel rays to a focus upon the retina (rays ∞ ∞ Fig. 79). Its refractive

power being not strong enough even for this, it is evident that still less will it be able to focus rays that already have a certain amount of divergence, i. e., those which emanate from a comparatively near object (O, Fig. 79).

The only rays that it can focus without the exercise of accommodation are those which are already converging somewhat (*c. c.*, Fig. 80), i. e., which if prolonged would meet at a point R behind the eye. The distance of this point from the eye gives us the measure for the degree of convergence that the rays must have for them to meet upon the retina. This point is,

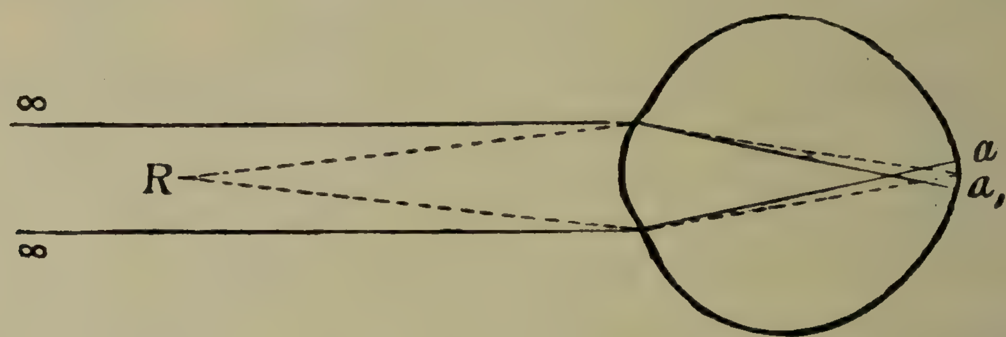

FIG. 78.—PATH OF THE RAYS IN A MYOPIC EYE.

accordingly, the far point, R—i. e., the point for which the hyperopic eye is adjusted when in a state of accommodative repose.

It is at a finite distance, just as is the far point in myopia, but is behind the eye and not in front of it, as in myopia. The difference between the two consists in this, that in myopia the rays that are brought to a focus by the eye emanate from the far point, while in hyperopia they converge to the far point. The far point in hyperopia, therefore is not a point from which the rays actually start or at which they actually unite, but is

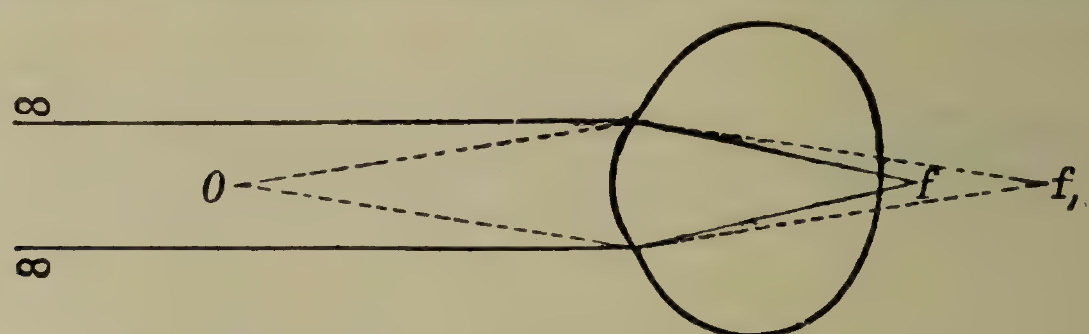

FIG. 79.

an imaginary point which is simply adopted to indicate the direction of the rays. We say, therefore, that the hyperopic eye has only a virtual far point, and we designate it by the negative sign:—R.

In astigmatism the two principal meridians have each a different far point. Thus in Fig. 73, if the retina is at f, the far point for the horizontal meridian will be at infinity, that for the vertical meridian at some finite distance in front of the eye.

Another and better definition of the far point is that it is the *conjugate focus of the retina*. Thus if rays emanating from the far point, R (Fig. 78), of a myopic eye come to a focus upon the retina, it is evident from the principle of conjugate foci (§ 154) that rays emanating from the retina and emerging from the eye will come to a focus at R. And, in fact, if we throw light into a myopic eye with an ophthalmoscope, so that a portion of the retina being illuminated sends out rays, these rays emerging from the eye will come to a focus at the far point of the latter and form there an inverted

nage of the fundus. If the myopia is so great that the patient's far point ; between us and him, we can see this image distinctly just as we see the ke image formed by an ordinary convex lens (see § 155). So also in hyperpia rays emanating from an illuminated point in the fundus and emerging rom the eye will diverge as though they emanated from the far point R Fig. 80) and will form an image of the point there. If R is sufficiently lose to the eye, i. e., if the hyperopia amounts to several dioptries, the image husformed by the eye of its own fundus will be clearly visible to the observer ith the ophthalmoscope. The image in this case is a virtual, erect image,

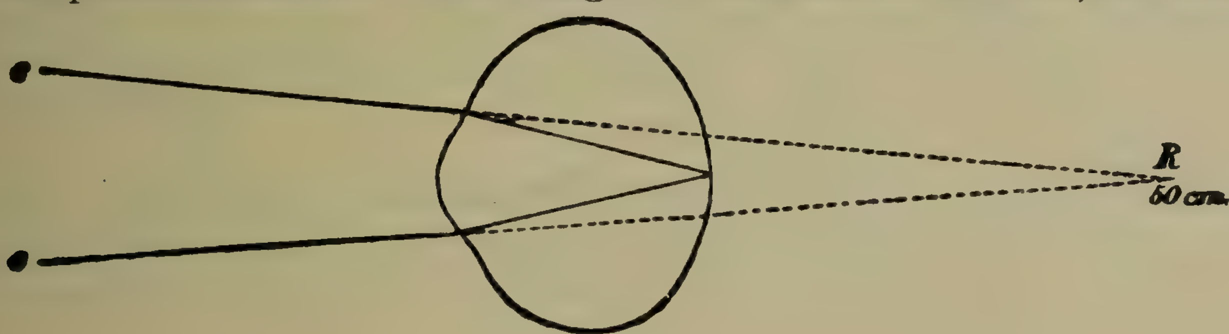

Fig. 80.

he eye acting like a convex lens when it is used as a magnifying glass (see 155). In an emmetropic eye, according to the principle of conjugate foci, ays emanating from an illuminated portion of the fundus will emerge arallel (see § 154).

173. Correction of Ametropia.—The correction of ametropia consists 1 placing such a glass before the eye that parallel rays will be brought to a ocus upon the retina. In other words we are to render the eye emmetropic.

Since the eye is already so adjusted that it will focus rays that inter-

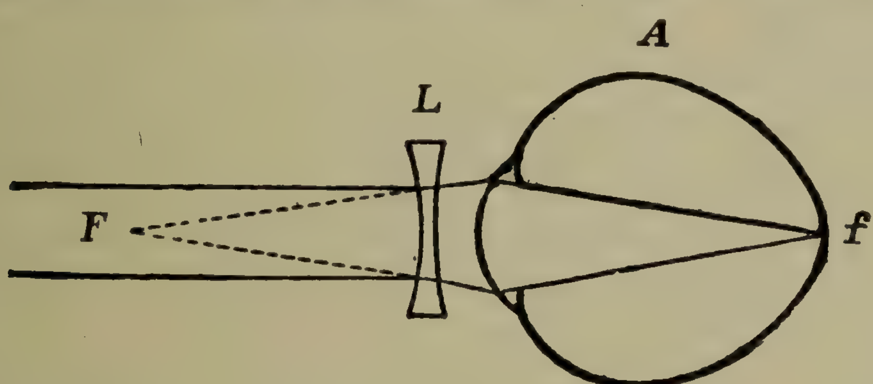

Fig. 81.—Correction of Myopia by a Concave Glass.

ect in its far point (R, Figs. 79 and 80), it is evident that to adjust it for arallel rays we must place before it a lens that will give parallel rays a lirection such that they will intersect in R. Thus in a myopic eye with a ar point F (Fig. 81) we must in order for it to focus parallel rays place before t a lens which will make parallel rays diverge as if from F. Such a lens vill be a concave lens with its second focus at F (see § 148). So too, in yperopia (Fig. 82) in order to make parallel rays meet upon the retina, we nust give them such a convergence that they will be directed toward R the far point of the eye), for it is rays that are so directed that the eye is ble to focus. But a lens which will make parallel rays converge to R is a onvex lens with its second focus at R.

Accordingly, both in myopia and in hyperopia the *correcting glass is that lens which placed before the eye will have its second focus at the far point of the eye.* The focal distance of the correcting lens, then, is equal to the distance from the second principal point of the lens, or, practically from the lens itself, to the far point.

It will be seen from the above that the strength of the correcting lens must vary *with its position* with regard to the eye. For suppose that the concave lens correcting a myopia (*L*, Fig. 81) is moved away from the eye or toward the far point, *F*. Since its focus must still be at *F*, it must now have a shorter focal distance, i.e., must be stronger

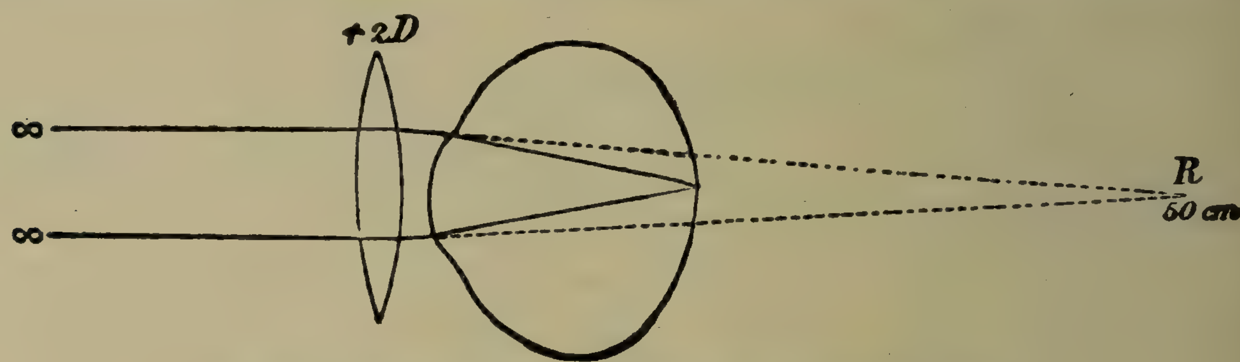

Fig. 82.—Correction of Hyperopia by a Convex Lens.

lens, to do its work properly. Thus a myopia corrected by a −12D lens (lens of 83 mm. focal distance) placed 10 mm. from the eye, would require a lens of 73 mm. focal distance i.e., one of nearly 14 D, if placed 20 mm. from the eye. In other words the −12 D lens, if carried 10 mm. further out, would become nearly 2 D too weak. Just the reverse would be the case with a convex glass (cf. Fig. 82). Thus we see that in the correction of distant vision the effect of a convex glass is increased and that of a concave glass is diminished by withdrawing it from the eyes. This effect must be borne in mind in examining the eye. Unless the correcting glasses applied are placed at the proper distance from the eyes, i.e., at the distance at which they will subsequently be worn, our tests both subjective and by skiascopy are likely to give erroneous results. Similarly it is important that in estimating the refraction by the direct method (§ 219) the ophthalmoscope be held at the standard distance (about half an inch in front of the cornea). Care also should be taken that reading glasses are in the proper place, since their effect, too, is altered if the glasses are shifted. But in the case of convex reading glasses it is not necessarily true that the effect is increased by withdrawing them from the eye. Whether this is so or not depends partly on the strength of the glass, partly on the distance of the object looked at.

The stronger the glass the greater the influence which its distance from the eye exerts. For this reason the intervals between the separate numbers are not required to be so numerous in the case of the stronger glasses as in the weaker ones, since the effect of the former can be readily increased or weakened by slight alterations in the distance. This change of strength produced by changing the distance is mainly of advantage in those that have been operated on for cataract, who possess no power of accommodation, and hence with their strong convex glasses could never have the sight adjusted except for one definite distance. By slightly displacing the glass they so far help matters that with the same glass they can see sometimes a little closer to them, sometimes a little further off.

For various reasons the best place for a glass is at the first focus of the eye, i. e., 14 mm. in front of the cornea. If the glass is so placed and the eye has its cardinal points in about the usual position, the following simple relation holds good: The strength of the correcting glass in dioptries is three times the eccentricity of the retina in millimetres. That is, if the eye is 2 mm. too long, it is myopic 6 D; if a portion of the retina projects 3 mm.

above the rest it will require a +9D glass to make it distinct in the direct method, etc. This relation finds constant application in ophthalmoscopy.

It must be remembered that it does not hold if the refractive media of the eye have an unusual curvature or composition so that the cardinal points of the eye occupy positions far from the ordinary. Thus in infants, who have very short but highly refractive eyes, the ratio between glass and eccentricity, instead of being 3: 1 is about 5: 1, so that 2 mm. of elongation would correspond to a difference of 10 D in refraction, instead of 6 D as in the adult eye. In aphakia, on the other hand, the relation is only 4: 3, so that an elongation of 3 mm. represents a change of about 4 D instead of 9 as in the lens-containing eye. Moreover in aphakia the change in the strength of the correcting glass is not strictly in proportion to the degree of elongation unless the correcting glass is placed 24 mm. in front of the cornea.

174. Correction of Astigmatism.—Astigmatism is corrected by a cylindrical lens. Thus, if in the case represented in Fig. 73 the retina is at f_1, the horizontal meridian being emmetropic will require no correction, the vertical meridian being hyperopic will require a convex glass. The correction here is effected by a convex cylinder with its axis horizontal. For along its axis the cylinder acts like plane glass, so that if we place the axis horizontal the rays passing through this meridian, being unaffected, will still come to a focus at f_1. At right angles to its axis the cylinder acts like a convex spherical lens, and if this is sufficiently strong it will cause the rays that pass through the vertical meridian and normally focus at f to gain such an added degree of convergence as to focus at f_1. If, on the other hand the retina is at f, the astigmatism will be corrected by a concave cylinder with its axis vertical. For now the vertical meridian is emmetropic and requires no correction, so that we must place in line with it the axis of the cylinder, which does not affect the rays passing through it, and place in line with the myopic horizontal meridian that meridian of the cylinder which is at right angles to the axis and which acts like a concave lens. If the concave cylinder is of the proper strength, the rays in the horizontal meridian, which would naturally focus at f_1, will thus be made to focus at f like the rays in the vertical meridian. In either case the strength of the correcting cylinder in dioptries will be found by multiplying the length of the focal interval $f_1 f$ measured in mm. by 3.

We thus see that a simple hyperopic astigmatism is corrected by a convex cylinder, and a simple myopic astigmatism by a concave cylinder; the axis of the cylinder in either case being placed in line with the emmetropic meridian of the eye. In other varieties of astigmatism we must add a spherical lens to the cylinder. Thus if the focal interval $f_1 f$ is 2 mm. and the retina is at 1, (Fig. 73) one mm. in front of f_1, we shall first put on a +3 D spherical lens, which will make the rays in the horizontal meridian focus at f_1 and those in the vertical meridian focus at f, (i. e., reduce the compound hyperopic astigmatism to a simple hyperopic astigmatism like the case first cited), and then add a +6 D cylinder, axis horizontal, which will correct the astigmatism. So also, if the retina is at 7, 2 mm behind f, we shall correct this compound myopic astigmatism, by putting on first, a −6 D spherical lens, which will convert the error into a simple myopic

astigmatism, as in the second case cited, and then a -6 D cylinder, axis vertical. If the retina is at *3*, two-thirds of a millimetre behind f_1, we may either put a -2 D spherical lens, thus converting the mixed astigmatism into a simple hyperopic astigmatism as in case 1, and then add a $+6$ D cylinder, axis horizontal; or we may put on a $+4$ D spherical lens, thus making a simple myopic astigmatism as in case 2, and add a -6 D cylinder, axis vertical. We may also in this case use the equivalent combination -2 D cylinder 90° $\bigcirc$ $+4$ D cylinder 180°. By the concave cylinder, which leaves the rays in the vertical meridian unaffected, the rays in the horizontal meridian would be made to focus at *3* instead of at f_1; and by the convex cylinder, which leaves the rays in the horizontal meridian unaffected, the rays in the vertical meridian would be made to focus at *3* instead of at f.

Correction of Irregular Astigmatism.—Astigmatism due to irregular curvature of the cornea (keratoconus, facet of cornea) may be relieved by any device which substitutes for this irregularity a regular curved surface. In Lohnstein's *hydrodiascope* a chamber filled with water and containing a strong plano-convex lens in front is placed in contact with the cornea; in the *contact glasses*, devised by Sulzer and Fick and manufactured by Müller, a thin glass plate having the curvature of the cornea is placed directly on the cornea of the patient and held there by air pressure, the capillary space between the two being filled with water. These glasses can be worn for several hours.

175. **Ametropia as a Variant of Emmetropia.**—In an eye myopic 3 D, parallel rays come to a focus 1 mm. in front of the retina. This usually occurs because the eye is 1 mm. longer than the emmetropic eye. But we may conceive this myopia as being due not to an increase in length of the eye, but to an increase in refracting power such as would be produced by the addition of a convex lens (Straub). That is, we may regard an eye which is myopic 3 D as an emmetropic eye with a $+3$ D lens before it. So too, an eye which is hyperopic 2 D may be regarded as an emmetropic eye with a -2 D lens before it, and an astigmatic eye as an emmetropic eye which is wearing a convex or concave cylinder. This conception is useful in making clear to ourselves and to patients the symptoms of ametropia. Thus to make an emmetrope realize how a myope of 5 D sees, we have simply to put before his eye a $+5$ D lens. He will then have the blurred vision for distance, the sharp and enlarged vision for near, and the close near point of one having a myopia of this amount. And like such a myope he will be reduced to an emmetrope again by adding a -5 D lens to the lens he already has. A myope of 5 D, an emme rope with a $+5$ D before his eye, and an emmetrope using 5 D of his accommodation have for the time being the same sort of vision (cf. page 172). So, too, to demonstrate how a hypermetrope of 2 D sees we have simply to put over the emmetrope's eye a -2 D lens. Such a one will have blurred vision, speedily rendered clear by an often painful and strained effort of accommodation, and a recession of his near point, just as the hypermetrope has. We can in like manner produce any desired variety and amount of *artificial ametropia*.

III. Accommodation.

176. **Adjustment of the Eye for Different Distances by Accommodation.**—Unless its refractive state is changed in some way, an eye is adjusted only for rays intersecting in its far point. Thus the emmetropic eye is naturally adjusted for rays coming from a distance, but not for rays emanating from an object near by. Such rays would be focused behind its retina. Still less would the hyperopic eye focus rays coming from a near point, since it cannot even make parallel rays converge enough to meet upon the retina. It is only the myopic eye that naturally and without

effort can focus rays diverging from a near object, and even in this case the eye is adapted for only one distance, namely its far point. In all these cases objects that are not at the far point of the eye appear indistinct because they are seen in diffusion images, which are formed in just the same way as the diffusion images in myopia and hyperopia. Thus suppose one of our eyes is myopic with its far point at B (Fig. 83) and that we place an open book at B and a pencil at some nearer point S. Then rays emanating from the book are without any effort of the eye united upon the retina at

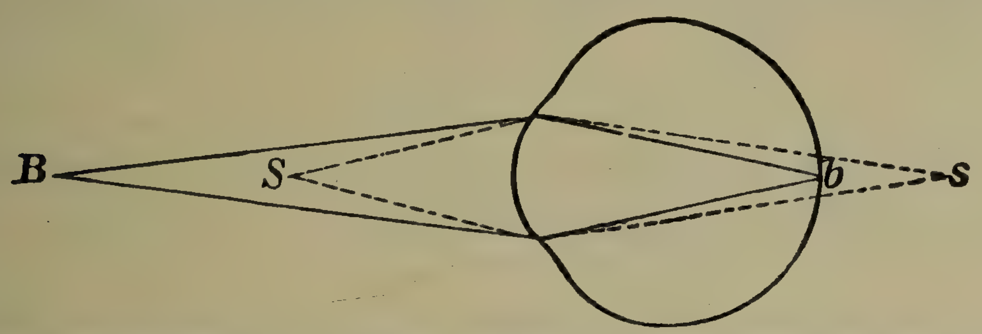

Fig. 83.—The eye being focused for a certain distance, B, rays emanating from a nearer point, S, form a diffusion circle upon the retina.

b to form a sharp image. The rays coming from the point of the pencil (S), which is nearer, have a greater divergence, and hence if the condition of the refracting media remains the same, are simply rendered somewhat less convergent by the latter; they would therefore unite at s—that is, behind the retina.

The condition here is therefore the same as that of a hyperopic eye when looking at a distant object (Fig. 79). A diffusion image is formed on the retina, which becomes larger and dimmer in proportion as s recedes behind the retina, i. e., the closer S approaches to the eye. The book therefore is seen distinctly, the pencil indistinctly. But just as the camera can be focused for different distances, so can we make the eye change adjustment from print to pencil point and vice versa, getting a distinct image of either alternately but never simultaneously. In doing this we are conscious that it takes some time to "focus" from one object to the other, and a change is felt to take place in the eye. This change is the accommodation, by which the optical adjustment of the eye is altered.

177. Nature and Mechanism of Accommodation.—In making the above experiment with the pencil and book, we feel that the eye is called upon to make an active effort when it is directed by a process of adjustment from the more distant book to the less distant pencil. In the same way, although not quite so distinctly, we feel a relaxation of this effort when adjustment is made for the book again. It may be concluded from this that the change of adjustment from a more distant to a less distant point is an active process—i. e., a muscular effort, which we call accommodation. On the other hand, the relaxation of the accommodation by which the eye is again adjusted for a greater distance consists in a relaxation of the contracted muscle. When in a state of perfect rest, the eye is adjusted for its far point. This condition of adjustment we find existing when the ciliary muscle is completely relaxed, either naturally from paralysis of the oculomotor nerve, or artificially from the use of atropine.

The *mechanism of accommodation* was determined mainly by the investigations of Helmholtz. It depends upon the elasticity of the lens, owing to which the latter always tends to approximate to the shape of a sphere. In the living eye the lens is inclosed in a capsule which is attached to the ciliary body by the fibres of the zonula of Zinn. These fibres are tightly stretched, and hence exert a uniform traction from all sides upon the capsule, so that the latter and the lens as well are flattened. The elasticity of the latter can make itself apparent only when the tension of the fibres of the zonula, and hence, too, of the capsule of the lens, is relaxed. This takes place most completely after division of the fibres of the zonula. When we remove the lens from the eye of a young person, we see it assume a spherical shape immediately upon the division of its connections. The same thing is observed in traumatic luxation of the lens into the anterior chamber. In the act of accommodation, the relaxation of the zonula is effected by the contraction of the ciliary muscle. It is the annular layer of fibres of the latter (Müller's portion; see *Mu*, Fig. 20) that is mainly of account in accomplishing this. When this fibre layer contracts, it lessens the size of the circle formed by the ciliary processes by approximating their apices to the border of the lens (shown by the black line in Fig. 84). In this way the space between the ciliary body and lens, that is bridged over by the fibres of the zonula, is contracted and the zonular fibres themselves are relaxed. It is the task of longitudinal fibres of the ciliary muscle (Brücke's portion; *M*, Fig. 20) to re-enforce the action of the circular fibres. The former have their anterior, fixed insertion in the corneo-scleral margin, while their posterior extremity loses itself in the movable chorioid. By the contraction of these fibres the flat portion of the ciliary body and the most anterior portion of the chorioid are drawn forward, and thus the relaxation of the fibres of the zonula which lie upon the surface of these structures is facilitated; but the main part of the work of accommodation always falls upon the annular fibres of the ciliary muscle, for which reason we find these fibres particularly well developed in eyes which have to accommodate a good deal—e. g., those of hypermetropes (see Fig. 100).

By the relaxation of the zonula the tension of the lens capsule is diminished, so that the lens is enabled by its elasticity to assume a more curved shape. At the same time there is necessarily produced a corresponding decrease in the equatorial diameter of the lens. The equator of the lens, accordingly, recedes inward toward the axis of the eye, and is thus kept from coming into contact with the ciliary processes as they advance.

The increase in curvature affects both the anterior and the posterior surface of the lens, but the former to a much higher degree (Figs. 71 and 84). The posterior surface of the lens does not change its place in the fossa patelliformis of the vitreous, the increase in thickness of the lens being effected simply by the advance of its anterior surface. Hence, the anterior chamber becomes correspondingly shallower; at the periphery alone is there a deepening of the chamber, inasmuch, as here the iris recedes a little. The sphincter pupillæ and, if vision is performed with both eyes, the two

internal recti also, contract in conjunction with the ciliary muscle. The act of accommodation, accordingly, is regularly accompanied by a contraction of the pupils and a movement of convergence.

According to Gullstrand's researches in maximum accommodation the thickness of the lens increases about 11 per cent, the curvature of the anterior surface 87 per cent and the curvature of the posterior surface 12.5 per cent. At the same time, the isoindicial surfaces (§ 162) particularly those near the core of the lens, become more nearly spherical (cf. Figs. 70 and 71). The result of this change in shape of its stratified refractive surfaces is that the lens gains in refractive power even more than the change in its external shape would account for.

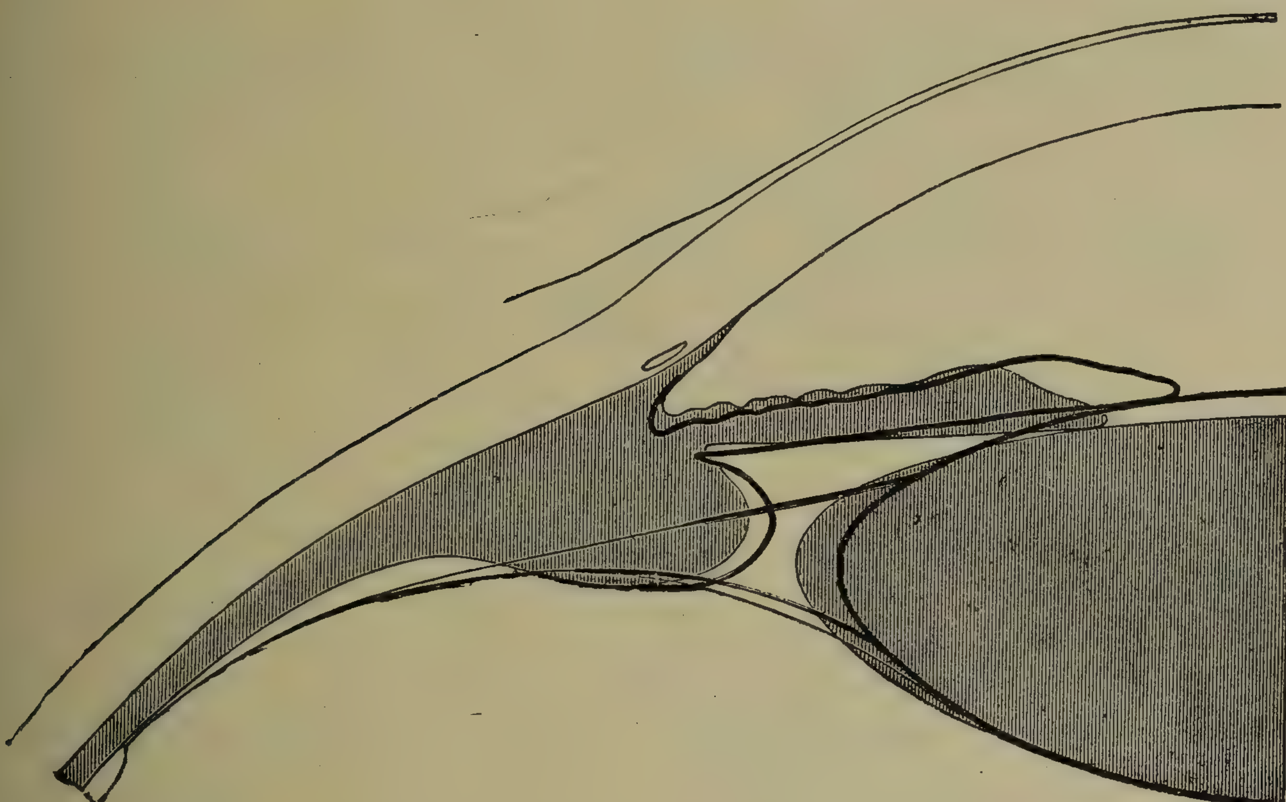

FIG. 84.—SCHEMATIC REPRESENTATION OF THE PROCESS OF ACCOMODATION.

The relation of the parts when the accommodation is at rest is designated by the shaded portions, and the relation when there is an effort of accommodation, by the thick black lines. In the drawing it looks as if the zonula fibres were running through the ciliary process. In reality the fibres of the zonula for the most part lie in the depressions between the ciliary processes, and hence run to the lateral slopes of the ciliary process that is represented in the drawing. With these slopes they are connected by numerous fibres and hence, when the ciliary processes advance toward the sagittal axis of the eye, the zonula fibres are taken along with them and are likewise pushed inwards; and to a corresponding amount the equator of the lens recedes toward the sagittal axis of the eye. Both surfaces of the lens become more curved and the anterior surface is advanced. The iris is broader and at its pupillary border is displaced forward; at its ciliary border backward. (Cf. also FIG. 71).

Tscherning believes, in opposition to Helmholtz, that contraction of the ciliary muscle causes a tightening, not a relaxation, of the zonula and that, consequently, the bulging of the lens in accommodation is produced by an active compression instead of a passive dilatation. But experiments seem to show conclusively that the explanation given by Helmholtz and described in the text is the true one (Hess, Gullstrand).

The Helmholtz theory, however, does not explain all the phenomena of presbyopia (see § 182).

178. Effect of Accommodation on Ametropia.—The effect of the relaxation of the zonula is to increase both the curvature of the lens and also its optical density (Gullstrand). The result is that the eye becomes more refractive. It acts, in fact, like an eye which has had a convex lens placed in front of it. If emmetropic before, it now becomes for the time being myopic, and is adjusted for a comparatively near point. A myopic eye

becomes still more myopic, so as to be adjusted for a distance even closer than its far point. A hyperopic eye becomes, first, emmetropic, so that it can focus parallel rays and thus see clearly at a distance, then becomes myopic so as to see near objects also. Since the hyperope thus uses up part of his accommodation in seeing at distance, it is clear that he has less left to use at near points; and it is easy to understand how the constant effort he has to put forth in order to see even at a distance may cause asthenopia or other troubles. The myope, on the other hand, necessarily uses less accommodation than the emmetrope and may not have to use any at all (see for further § 201).

Accommodation not only enables a person naturally hyperopic to overcome his error and thus see distinctly at a distance without glasses, but may also correct an *artificial hyperopia* or simulate an *artificial myopia.* That is, it enables one to see clearly through a concave glass, even if, being an emmetrope, he does not require such a glass at all or, being myopic, really requires a glass of less strength. The tendency thus to overcome a natural or an artificial hyperopia is very strong, and the effort is often obstinately maintained, so that the eyes refuse to relax even when the proper glasses are supplied that would render the eye emmetropic. See also § 195.

While a person can thus by accommodation render the eye apparently more refractive than it really is, he cannot reverse this process, i. e., make himself appear more hyperopic or less myopic than is actually the case. This is obvious since his actual total hyperopia or myopia represents the condition when the accommodation is relaxed to the utmost. A person therefore with normal vision may be hyperopic, having then corrected his error by forcing his accommodation; but he cannot be myopic to any extent, since even moderate degrees of myopia cause blurring of sight and there is no way in which he can correct this myopia by accommodation

179. Measurement of the Accommodation.—To measure the amount of the accommodation we must determine its limits, which are defined by the far point and the near point. The far point (*R*, punctum remotum[11]) is that point for which the eye is focused when the accommodation is completely relaxed. The near point (*P*, punctum proximum) is that point for which the eye is focused when the accommodation is strained to its utmost.

In emmetropia *R* lies at an infinite distance, since the emmetropic eye when in the state of rest is focused for parallel rays. While the position of *R* is thus the same for all emmetropic eyes, that of *P* varies greatly. It may, for example, be at 10 cm. ($P = 10$ cm.). The space lying between *R* and *P*—i. e., in the example selected the space between ∞ and 10 cm.—is called the *region of accommodation.* But the extent of this space affords

[11] The far point receives its name because for the emmetropic and myopic eye it is the farthest point at which the eye can see distinctly. This definition, however is not applicable in hyperopia, in which the far point lies behind the eye. If the accommodation is put into play then in proportion as the hyperopia is corrected by it, the point for which the eye is adjusted recedes from the eye until it has got out to infinite distance. If the accommodation is not strong enough to adjust the eye for infinite distance (absolute hyperopia), the near point, too, lies behind the eye and in fact at a greater distance from the latter than is the far point. As an example of this may serve an eye with *H* of 4 D, whose region of accommodation is represented in Fig. 96 A. If this eye should have an accommodation of only 2 D, its near point would be 50 cm. behind the eye, and with a range of accomodation of 4 D, it would be at infinite distance; i.e., in either case would be farther than the far point, which lies but 25 cm. behind the eye. For this case, therefore, the expression near point is not very sensibly chosen.

no measure for the amount of work done by accommodation; this, in fact, being measured by the increase of refractive power which the eye undergoes in passing from the state in which the accommodation is at rest (R) to the state in which the utmost effort of accommodation is made (P). The amount of increase of refractive power is called *amplitude or range of accommodation* (A), and is accordingly the difference between the refractive power of the eye when the accommodation is exerted to its utmost and when it is at rest—i. e., $A = P—R$. For R and P in this equation should be substituted not their linear values, but the corresponding number of dioptries.

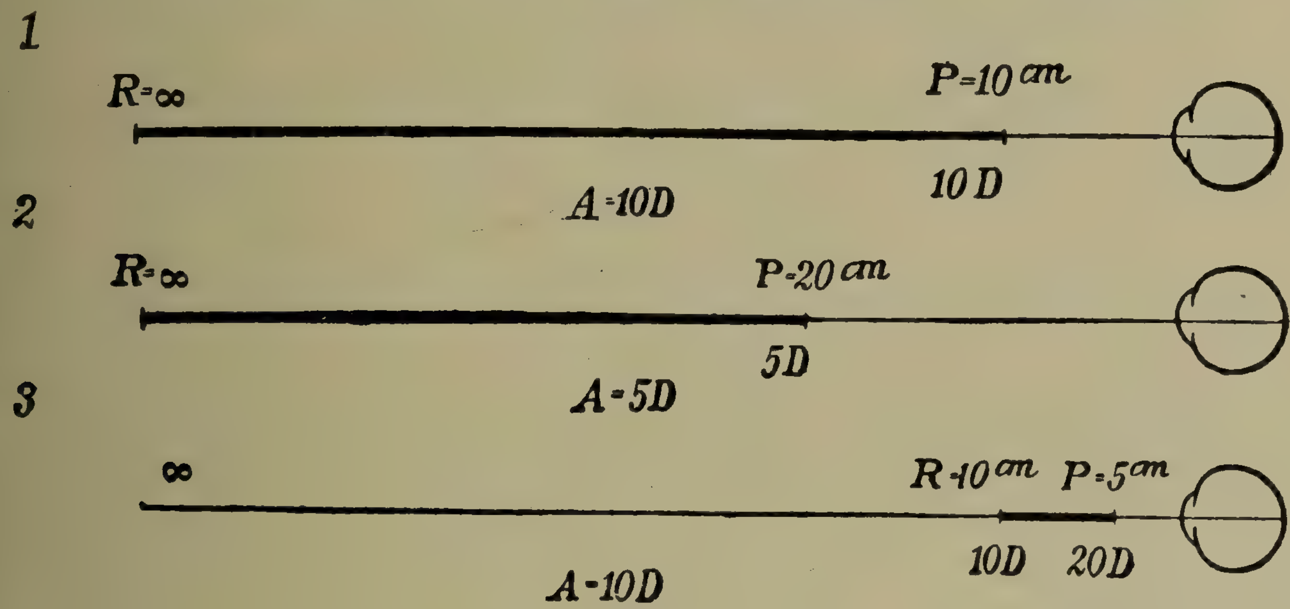

FIG. 85.—REGION OF ACCOMMODATION.
1, In a young emmetrope; 2, an older emmetrope; 3, a young myope.

The method of determining the amplitude of accommodation may be illustrated by the following examples represented graphically in Fig. 85. 1. A young emmetrope whose far point lies at an infinite distance and near point at 10 cm. from the eye; 2. An older emmetrope whose $R = \infty$ and $P = 20$ cm.; and lastly, 3. A young myope, whose $R = 10$ cm. and $P = 5$ cm. The region of accommodation—i.e., the space lying between R and P—is of a very different extent in these three cases. In cases one and two it is infinitely large, since it extends to an infinite distance, while in case three it amounts to only 5 cm. If, therefore, we were to reckon the work done in accommodation by the extent of the region of accommodation, we would arrive at the erroneous view that, with regard to the former as well as the latter, there is an enormous difference between the first two cases on the one hand and the third case upon the other.

But, as a matter of fact, the case is quite different, as can be gathered from an experimental test of the accommodation. Suppose that while we close one eye we hold before the other a book at a distance of 20 cm. and a pencil point midway between the book and the eye—i.e., at a distance of 10 cm. from the latter. We then first look out into space over the book, so that the accommodation is completely relaxed, and afterward fix our gaze upon the print of the book. In so doing, we have a sense of accommodative effort in the eye. We now turn our glance from the book to the pencil point, and endeavor to see the latter distinctly. If this is possible at all, it costs us a very considerable effort which will tax the energies of most persons more than does the effort required to adjust the eye from infinite distance to the book. Hence, the act of changing the accommodation from 20 cm. to 10 cm. costs us at least as much effort as the change from infinity to 20 cm. From this it is clear that we are not justified in taking the linear distance between the points of fixation as a measure of the accommodative effort, and that, therefore, the region of accommodation can not serve as an expression of the work done in accommodation.

We get a correct idea of the amount of accommodation that is called into play if we take into consideration the increase produced in the refractivity of the eye by the accom-

modation. This increase of refractivity is effected by an increase in the curvature and density of the lens, a thing which we can also conceive of as accomplished by the addition to the unchanged lens of a second, weaker convex lens. This "supplementary" crystalline lens (z, Fig. 86) represents the increase in refractivity, and would form the best measure of the accommodation. Instead of determining the refractive power of this supplementary lens directly, we can determine its equivalent, i.e., the glass which placed before the cornea of the eye would produce the same increase of refractive power. Take, for example, case 1 (Fig. 85). In this, when the accommodation is making its utmost effort the refractivity of the eye is increased by the supplementary lens to such an extent that ray

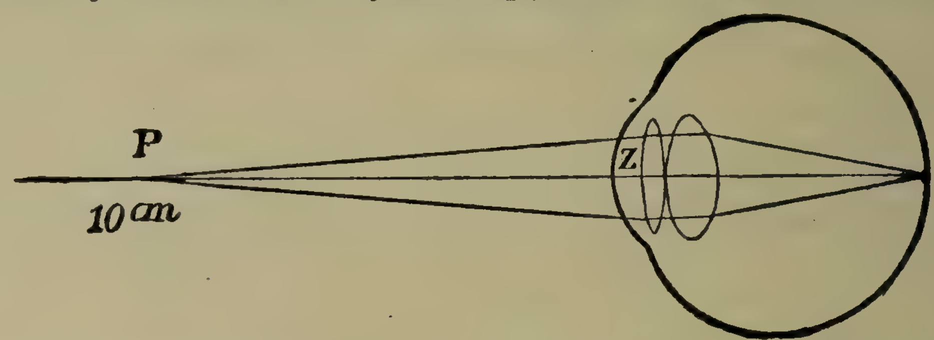

Fig. 86.—Accommodation represented schematically by a "Supplemental" Crystalline Lens, z.

emanating from P—that is, from a distance of 10 cm. from the eye—are united upon the retina (Fig. 86). We now paralyze the accommodation in this eye with atropine, so that the eye remains steadily focused for infinite distance, and try to find the convex glass with which the eye is enabled to see the point, P, distinctly. We find that for this purpose a glass, L, of a focal distance of 10 cm. = one of 10 D is necessary (Fig. 87). If this glass is placed in front of the eye—i.e., 10 cm. behind the point P—the latter will lie just in the principal focus of the glass. The rays (p p, Fig. 87) emanating from P are hence made parallel by the glass, and being so, are united by the non-accommodating eye upon its retina. The lens L, therefore, does the same work as the natural accommodation (repre-

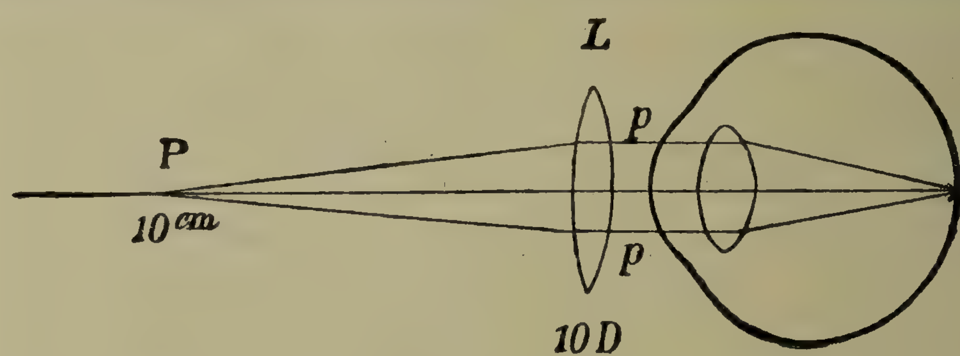

Fig. 87.—The Accommodations replaced by a Lens of Glass, L, set before the Eye.

sented by the supplementary crystalline lens, z), and can accordingly be taken as the measure of the latter. Accommodation measured in this way we call *amplitude* or *range* of accommodation. This, therefore, would be in the first case $A = 10$ D. In the second case (Fig. 85, 2) as may be shown in the same way, $A = 5$ D. Hence, in the emmetropic eye, A is expressed by the lens whose focal distance equals the distance of the near point of the eye; or $A = P$, when P is expressed in dioptries.

What relation does A bear in the third case (Fig. 85, 3)? Here P is situated at 5 cm. and hence $P = 20$ D. But this value can not be regarded as the expression of the work done in accommodation, since the eye in question being short-sighted, is, even when the accommodation is at rest, adjusted for a distance less than infinity—i.e., for 10 cm. This eye, when its accommodation is at rest, acts like a non-accommodating emmetropic eye before which has been placed a lens of +10 D (Fig. 87). For this eye, therefore, we can say $R+10$ D. But when making its utmost accommodative effort, this eye acts like a non-accommodating emmetropic eye before which has been placed a glass of +20 D. Obviously, in order to determine the work done in accommodation—i.e., the increase in refractivity in passing from R to P—we must subtract from the value corresponding to P

the value of R, or $A = 20\ D - 10\ D = 10\ D$. Hence, we may enunciate the following formula as representing the general law:

$$A = P - R,$$

in which P and R are expressed in dioptries. This formula holds good for all conditions of the refraction. For the emmetropic eye it is simplified by the fact that the latter, when the accommodation is at rest, is adjusted for infinite distance, so that $R = 0\ D$, and consequently $A = P$, as we found above.

Let us take another survey of the three cases, and compare the *region of accommodation* with the range of accommodation. We find that the former is infinitely large in the first two cases, and only 5 cm. in the third case. Now, A in the first case is 10 D, in the second only half as great—i.e., 5 D—and in the third case again is as great as in the first. Accordingly, from the range of accommodation we get an entirely different and in fact a more correct conception of the work done in the accommodative act than we do from the region of accommodation. This is owing to the fact that different portions of the region of accommodation represent entirely different values. It takes as much accommodative effort to bring the accommodation from 10 cm. to 5 cm. (case three) as to accommodate from infinity to 10 cm. (case one); the value in both cases being 10 D. This fact is in harmony with the sensations that we experience in our eyes, when in the experiment previously adduced, we look successively at infinite distance, at the book, and finally at the pencil point. Displacement of the accommodation through 1 centimetre of the region of accommodation is significant of an effort which is the greater, the nearer this centimetre is situated to the eye.

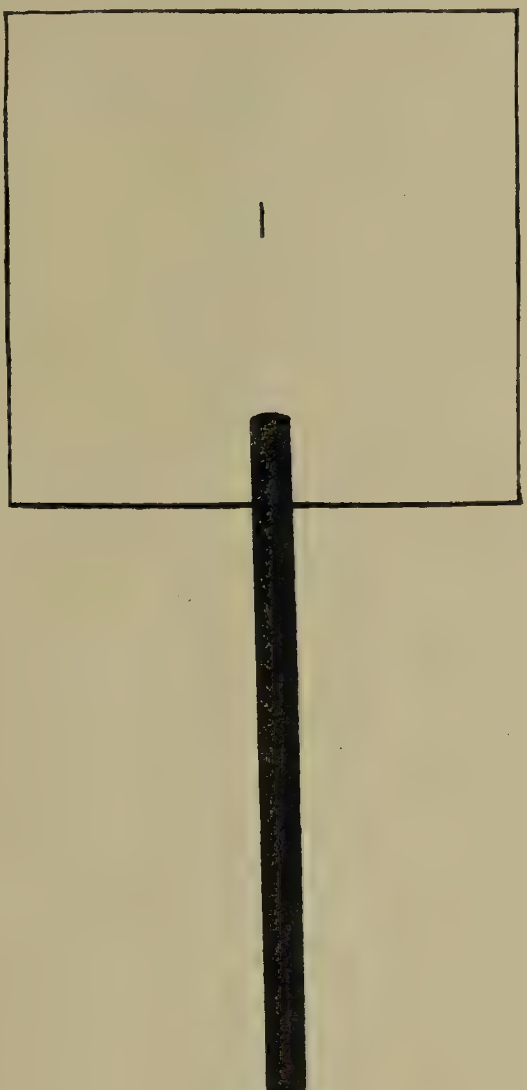

FIG. 88.—ACCOMMODATION CARD. Natural size.

The region of accommodation, however, gives us a good idea of the *availability of accommodation*. In case one the region of accommodation is so situated that the eye sees clearly at all distances which can be considered to exist in practical life. But in case three the region of accommodation lies so close to the eye that practically it has no value whatever; this eye would be no worse off without accommodation. (This, of course, holds good only upon the supposition that correcting glasses are not worn, as by these the location of the region of accommodation is shifted.)

180. Determination of the Far and Near Points.—We determine the *far point* when we determine the refraction, for the latter is, in fact, the refractive state of the eye when focused for the far point. If we know the refraction R is determined to. (See §§ 173, 174, and Chapter XV.)

The *near point* may be determined by making the patient look at fine print, which is brought up to the eye until it ceases to appear distinct. A better test-object is the accommodation card (Fig. 88). The card is approximated to the eye until the black line on it which is 0.2 mm. in diameter begins to grow faint or doubles. The distance at which this occurs is measured with a rule graduated in cm. and D (Prince's rule Fig. 89). The measurement is preferably taken not from the eye itself but from its anterior focus (14 mm. in front of the cornea), as this is the point where

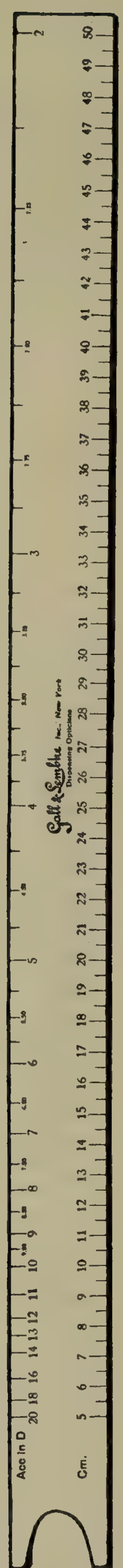

FIG. 89.—MODIFIED PRINCE'S RULE.

The rule 50 cm. long is graduated in cm. and dioptries. The notched end is fitted against the nose in such a way that the ends of the prongs on either side are 14 mm. in front of the cornea. The accommodation card is then carried in along the top of the rule in testing the binocular accommodation and along either side in testing the accommodation of either eye separately.

glasses that are designed to replace and hence to measure the accommodation are placed. Except in the case noted in the next paragraph, the eye is provided with the full correction, i. e., is rendered emmetropic, so that R = 0 D and *P* (read off in D) gives at once *A* or the total range of accommodation. It is well to test first the accommodation of each eye separately and then the accommodation of the two eyes together.

When *P* has receded to such a distance from the eye that small objects like the accommodation line appear under too minute an angle, and hence cannot be seen distinctly at all, the following expedient is adopted: We place before the eye a convex glass—e. g., one of 3 D, by which near vision is rendered distinct—and then determine the near point. This is found to be, say, 25 cm. The refraction corresponding to this distance is 4 D, from which, in order to learn the actual near point, we must subtract the 3 D supplied by the lens. Thus $P = 4\text{ D} - 3\text{ D} = 1\text{ D} = 100$ cm. If with such a convex addition the test object (accommodation line) is carried away from the eyes until it blurs, we can likewise determine *R*. Suppose, for example, that with a +4 D before the eye the line just begins to blur at the point on the Prince's rule marked 4.50. Then $R = 4.50 - 4.00 = +0.50$; i. e., there is 0.50 D myopia. If the line had begun to blur at the point marked 3.25, *R* would have been 3.25—4.00 = −0.75; i.e., there would be 0.75 D of hyperopia. If *R* and *P* are both taken in this way the range is given directly. Thus if with +3.00 added, *R* is at 3.50 and *P* is at 5.00, $A = 1.50$. This method is particularly valuable for determining the residual range when the eye is wholly or partly under the influence of a cycloplegic (see § 217). In computing the number of dioptries used in focusing for a certain distance, or, vice versa, the distance at which an object must be if a certain amount of accommodation is used the table in § 156 will be found useful.

181. Relative Accommodation.—In considering the accommodation hitherto, we have started from the assumption that vision is performed with only one eye. When the two eyes are employed simultaneously the convergence as well as the accommodation must be taken into account. These two functions go hand in hand. When our gaze is fixed upon the distance, $A = 0$, and the visual axes are parallel—i.e., the convergence, too, is in a state of rest. When we look at a near point—e. g., one situated at 29 cm.—we are compelled both to accommodate and to converge for this distance. Hence, through constant practice an intimate connection is effected between accommodation and convergence, so that with any given accommodation the corresponding effort of convergence is always made, and vice versa.

This connection, however, is not one that is rigid and insusceptible of change. On the contrary, we have the ability of emancipating ourselves from it within certain limits—that is, while in the act of converging for a

certain distance, of making a little more or a little less accommodation than corresponds to this distance. A man is made to fix his gaze upon fine print at a distance of 33 cm. Now, let us suppose that the subject is emmetropic and thus has his far point (R) at infinity, while P is situated at 10 cm., which corresponds to a range of accommodation (A) of 10 D (Fig. 90). Of this range of accommodation 3 D ($100 \div 33 = 3$) will be employed if the subject is converging for a distance of 33 cm. Now, a concave glass of 1 D is placed before each eye. The subject will for the first moment have obscuration of vision, but will soon see distinctly. He has compensated for the diminution of the refractive power of this eye, caused by the -1 D glass, by exerting 1 D more of his accommodation. But the convergence meanwhile remains unchanged—i. e., it is still adjusted for 33 cm. The same phenomenon occurs when, instead of -1 D, a glass of $+1$ D is placed before the eye. The refractive power of the eye is made too great by the convex glass, and this is neutralized by the eye's relaxing its accommodation through 1 D. In this way we can bring stronger and stronger convex and concave glasses successively before the eye until finally we come to those with which distinct vision is no longer possible. We thus find the limits within which the accommodation may be augmented or relaxed, the convergence remaining the same (*relative accommodation*).

In the example selected, suppose that the subject is able to see distinctly at a distance of 33 cm. with a convex glass of 2 D. This corresponds to a relaxation of his accommodation from 3 D to 1 D —that is, his relative far point, R_1, lies at a distance equivalent to 1 D, or 1 metre, from the eye. Suppose on the other hand, that the subject with the same degree of convergence overcomes concave glasses of 3 D, a thing which is effected by an augmentation of the accommodation from 3 to 6 D; his relative near point, P_1, then is at a distance equivalent to 6 D = 17 cm. The relative range of accommodation $A_1 = P_1 - R_1 = 6D - 1D = 5D$. This is the relative range of accommodation for a convergence of 33 cm.; for a different convergence the relative near point, far point, and range would be different still. On the other hand, there is only one absolute far point, near point and range of accommodation.

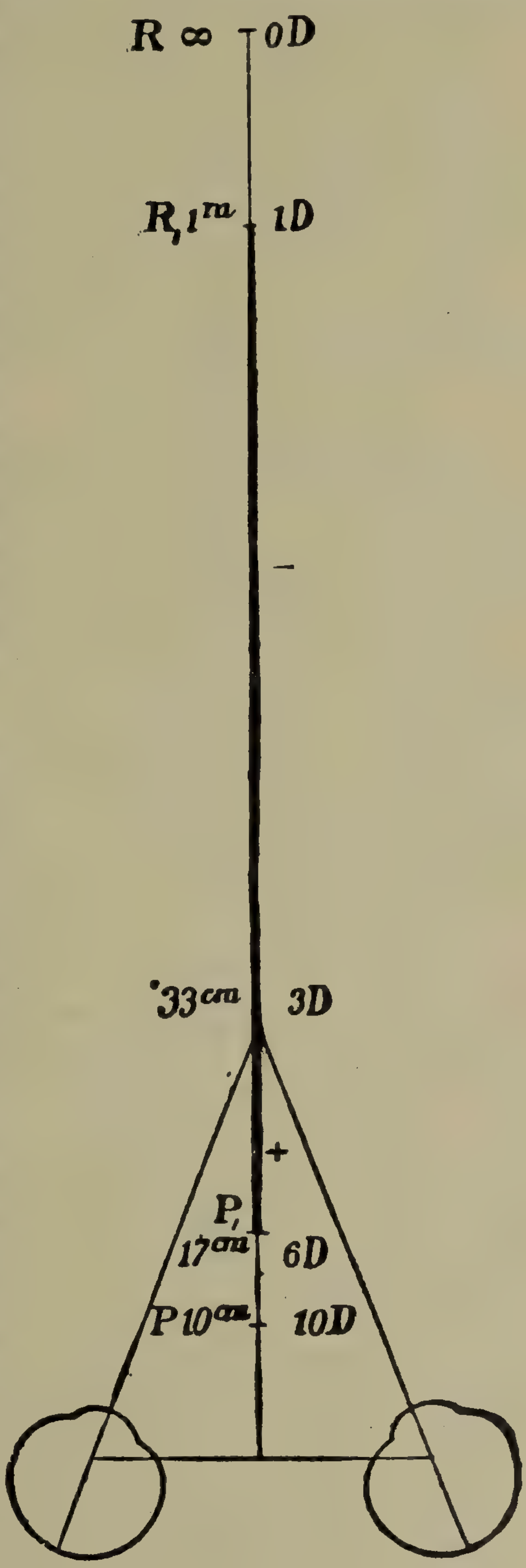

Fig. 90.—Schematic Representation of Relative Accommodation.

The range of relative accommodation is divided by the point upon which convergence is made into two segments. One lies upon the proximal side of the point of fixation, and thus in the example selected extends from 3 to 6 D. It represents the amount of accommodation which, if necessary, one can still press into service while keeping the convergence the same—i.e., it represents the amount of accommodation which one may be said to have in reserve. It is hence denoted as the *positive* portion (+, Fig. 90) of the relative range of accommodation. The other segment lies on the distal side of the point of fixation, and in our case extends from 3 to 1 D. It is the *negative* portion of the relative accommodation (—, Fig. 90). Hence, with a convergence of 33 cm. the positive portion of the relative accommodation amounts to 3 D, the negative portion only to 2 D.

182. Changes of the Accommodation with Age.—The accommodation diminishes with age, and this diminution is manifested by a continuous recession of the near point. The diminution in the accommodation can not be referred to the decrease in the power of the muscles in general and the ciliary muscle in particular, occurring in old age, for it begins in childhood—that is, at the time when the muscles are still gaining in strength. In fact, the cause of the diminution of the accommodation lies in the gradual decrease of the elasticity of the lens. This, again, stands in causal connection with the condensation of the lens due to loss of water and leading to a process of sclerosis that begins in the centre of the lens (formation of a nucleus). The harder the lens becomes in virtue of this process the more its elasticity is impaired, so that even after the zonula is relaxed the lens becomes less and less able to change its shape.

A man of 40 whose near point is at 17 cm. has probably as much real accommodative power (actual contractile power of the ciliary muscle) as the boy of 10 whose near point is at 7 cm. That is, if the lens in each was equally fluid, both would be able to focus for an object, extremely near, say at 6 cm. This would be their *physiological near point* (Fuchs), which would then be the same for each, while the point to which, owing to the differing rigidity of their lenses, each can actually focus (*physical near point*) is very different in the two cases. Moreover, under certain conditions it seems likely that the adjustment (contraction) of the ciliary (muscle *physiological accommodation*) may not by any means necessarily correspond with the adjustment thereby effected in the lens (*physical accommodation*). For example a presbyope with a natural accommodation of only 1 D is enabled by his glass to see objects at 33 cm. In so doing he need contract his ciliary muscle only to the extent required to produce an effect of 1 D in the lens refraction. Yet it is probable that he really exerts an amount of ciliary contraction that would produce an effect of 3 D if the lens could respond. In other words, he contracts his ciliary muscle to accord with the stimulus set up by the convergence rather than to accord with the effect on the lens that the convergence is designed to produce (Fuchs). The *physical accommodation diminishes progressively* with age, while the physiological accommodation is supposed to remain practically unchanged. If the latter view is correct the sum-total of ciliary energy is about equal in the child, the youth, and the middle-aged. This sum-total of energy we may divide into two parts—one producing an effect noticeable in accommodative effort (manifest energy) the other producing no effect (latent energy). Obviously, the less able the lens is to expand,—i.e., the older the subject, the less manifest ciliary energy will be required and the greater will be the amount that is latent. Thus suppose we have three persons, aged respectively 15, 25 and 40, having all three, an amount of ciliary muscle energy, which if unrestricted would produce an accommodative effect of 20 D. Owing now to the differing states of their lenses, the first has an accommodation of 15 D, the second of 10 D, and the third of 5 D, so that the amount of latent or unused energy is 5 D, in the first, 10 D in the second and 15 D in the third. Suppose further that we paralyze the ciliary muscle in all three by a gradually acting poison like homatropine This will have to abolish all the latent ciliary energy before it produces any effect on the accommodation that we can appreciate. It should then have a very different effect on the three persons. In the first it should show its effect early since there are only 5 D of latent ciliary energy to be abolished. In the second, it should occur much later, and in the third later still or perhaps hardly be noticeable at all. Now as a matter of fact, repeated experiments show that the paralysis of accommodation will begin to be apparent at about the same time and will advance at the same rate in all three. Hence it seems likely that the latent ciliary energy is about the same in the three, so that the total energy (latent plus manifest), i.e. the *physiological accommodation too diminishes* progressively with age.

The accommodation recedes equally fast in men and in women.

In the same subject the *binocular accommodation* is regularly greater—sometimes several dioptries greater—than the monocular. Quite commonly

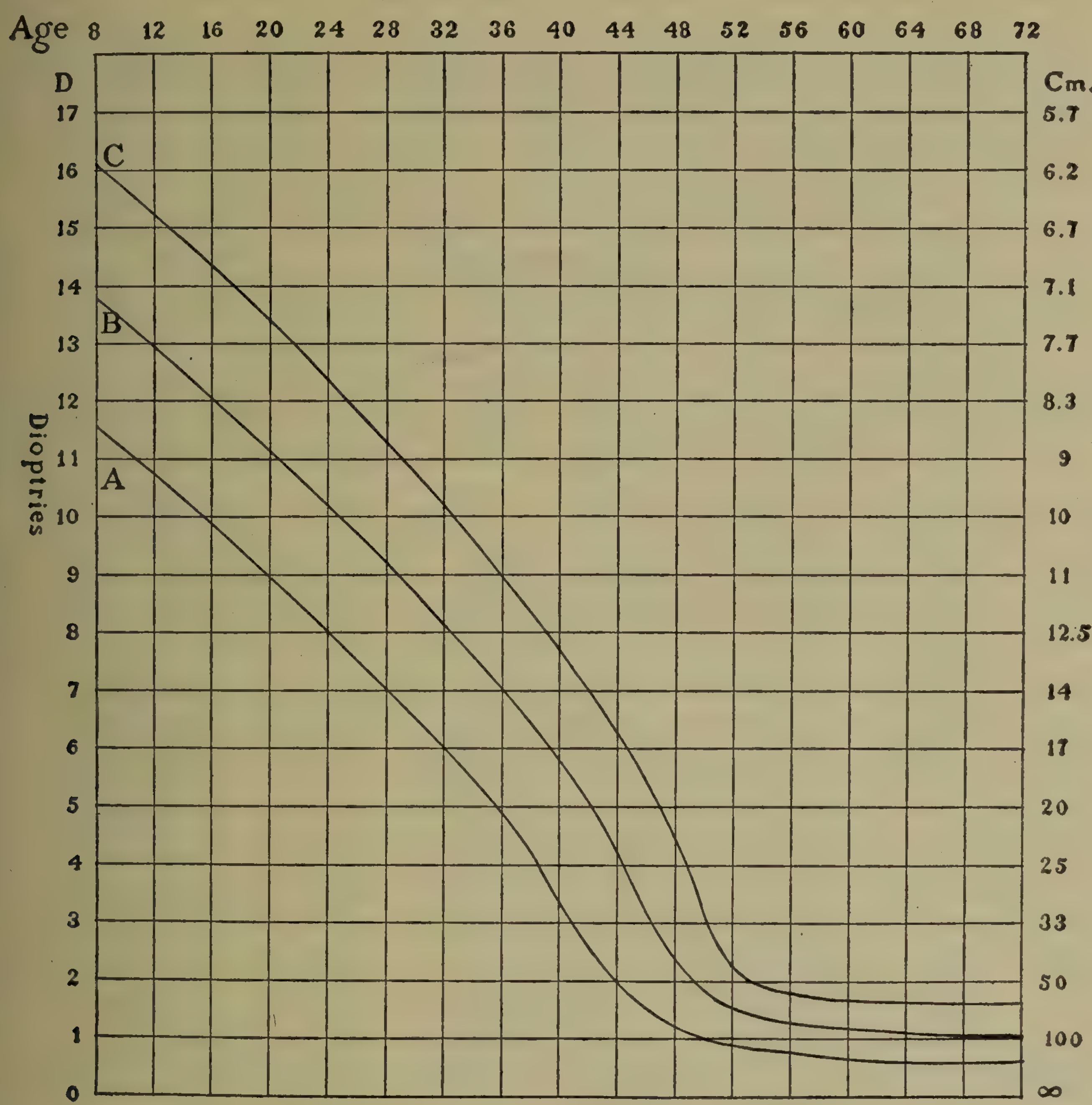

FIG. 91.—RANGE OF MONOCULAR ACCOMMODATION AT DIFFERENT AGES. (Donders, amended by Duane.)

The numbers on the left hand give the strength of the lens, which placed before the emmetropic eye, at a distance of 14 mm. from the apex of the cornea (i.e., placed at the anterior focus of the eye), can replace the accommodation of the eye at the given age, and hence is equivalent to the accommodation, so far as regards the increase of refractivity in the eye which the latter produces. These numbers, therefore, give the range of accommodation of the eye. The right-hand numbers give the focal distances of these lenses or the distance of the near point from the anterior focus of the eye in cm. Obviously no single observer can follow the progress of the range of accommodation from youth to age, in one and the same individual. The progress of accommodation can, therefore, be found only by determining it in a large number of persons with normal eyes, at different ages and taking the mean of the observations. This is shown by the line B, which indicates the mean position of the near point from the anterior focus of the eye and also the range of accommodation in D, of an emmetropic eye at different ages. The line A gives the least values, the line C the maximum values of the accommodation that have been found in the individual cases. They accordingly show the limits within which the position of the near point and hence too the accommodation can still be regarded as normal. For binocular accommodation the limits are on an average 0.2—0.6 D above those indicated, the difference growing less as the age increases. An accommodation which in any given subject persistently falls below the lower limit indicated by A for his age, must be considered as pathologically weak.

it is greater even when the vision of one eye is very different from that of the other or when there is no real binocular fixation. Probably the effort to converge, even when this effort is not fully successful, is mainly responsible for the greater amount of accommodation in binocular vision.

12

The superiority of binocular over monocular accommodation is of special importance in persons over 48, in whom slight variations in the maximum power of accommodation have a considerable bearing on the range of near vision and the strength of reading glass accepted.

The state of the accommodation at different ages for each eye taken separately is shown in Fig. 91 and the accompanying table.

TABLE OF ACCOMMODATION

AT VARIOUS AGES.

In D and tenths. Near point reckoned from anterior focus of eye (14 mm. in front of cornea)

	Accommodation				Accommodation		
Age	Minimum	Mean	Maximum	Age	Minimum	Mean	Maximum
8	11.6	13.8	16.1	37	4.5	6.7	8.8
9	11.4	13.6	15.9	38	4.1	6.4	8.5
10	11.1	13.4	15.7	39	3.7	6.1	8.2
11	10.9	13.2	15.5	40	3.4	5.8	7.9
12	10.7	12.9	15.2	41	3	5.4	7.5
13	10.5	12.7	15	42	2.7	5	7.1
14	10.3	12.5	14.8	43	2.3	4.5	6.7
15	10.1	12.3	14.5	44	2.1	4	6.3
16	9.8	12	14.3	45	1.9	3.6	5.9
17	9.6	11.8	14.1	46	1.7	3.1	5.5
18	9.4	11.6	13.9	47	1.4	2.7	5
19	9.2	11.4	13.6	48	1.2	2.3	4.5
20	8.9	11.1	13.4	49	1.1	2.1	4
21	8.7	10.9	13.1	50	1	1.9	3.2
22	8.5	10.7	12.9	51	0.9	1.7	2.6
23	8.3	10.5	12.6	52	0.9	1.6	2.2
24	8	10.2	12.4	53	0.9	1.5	2.1
25	7.8	9.9	12.2	54	0.8	1.4	2
26	7.5	9.7	11.9	55	0.8	1.3	1.9
27	7.2	9.5	11.6	56	0.8	1.3	1.8
28	7	9.2	11.3	57	0.8	1.3	1.8
29	6.8	9	11	58	0.7	1.3	1.8
30	6.5	8.7	10.8	59	0.7	1.2	1.7
31	6.2	8.4	10.5	60	0.7	1.2	1.7
32	6	8.1	10.2	61	0.6	1.2	1.7
33	5.8	7.9	9.8	62	0.6	1.2	1.6
34	5.5	7.6	9.5	63	0.6	1.1	1.6
35	5.2	7.3	9.3	64 to 72	0.6	1.1 to 1	1.6
36	4.9	7	9				

The values given above, especially for the lower ages, differ materially from those given by Donders and others. The reason for this is that these investigators measured the distance of the near point from either the cornea or from the nodal point of the eye, while in the table the measurement is supposed to be made from a point 14 mm. in front of the eye. This latter measurement gives, indeed, a truer idea of the dioptric value of the accommodation, since the glass that we use to replace the accommodation is, as a matter of fact, situated here and not in contact with the cornea. If, for example, a person has a near point situated 10 cm. from the cornea, he would according to the old method of reckoning have an accommodation of 10 D. But if paralyzed with atropine his accommodation would be replaced by a lens, not of 10 D but of nearly 12 D, since this lens being placed 14 mm. from the cornea would be only 8.6 cm. from the near point and must therefore have a focal distance of that amount. The same principle applies where we are correcting with a glass a partial loss of accommodation, as in presbyopia.

A further reason for the discrepancy is that some of the middle-aged subjects examined by Donders probably had a latent hyperopia of a dioptry or more. This being unallowed for would make the estimate of the range just so much too low.

According to Roy the accommodation in the natives of Africa (who are very largely emmetropic) is from 1 to 3 D higher than in Europeans of the same age.

The diminution in the range of accommodation goes on steadily from youth to age. It begins to be troublesome when the near point has receded so far from the eye that the finer kinds of work, and especially the reading of fine print, become difficult or impossible, so that there develops a desire for glasses. This is the case when the near point recedes beyond one-third of a metre ($A = 3$ D), i. e., between the ages of forty-five and fifty. This is the period, therefore, to which we assign the beginning of *presbyopia.*[12]

In age, owing to lenticular changes (and particularly the fact that the lens becomes more homogeneous) the eye often becomes more hyperopic or less myopic—i.e., the far point as well as the near point recedes (Donders). Cf. § 192.

Presbyopia is not a disease, but a physiological process which every eye undergoes. Persons who are presbyopic push the book farther away from them, like to avoid fine print, and pass over the footnotes. Reading at night gives them special trouble, because the pupils dilate owing to the enfeebled illumination, and hence the diffusion circles are larger. They then try the expedient of bringing the light between the book and their eyes so as to make their pupils contract by having a large amount of light fall upon them. In the subsequent progress of presbyopia reading or fine work at last becomes absolutely impossible without glasses. Pain, however, or asthenopia usually does not occur, as it does in hypermetropes.

When an eye is not emmetropic, but has an error of refraction, this error shifts the range of accommodation, and hence also the time when presbyopia begins. (See §§ 197 and 201).

183. Correction of Presbyopia.—Ordinarily an emmetrope or one who has been rendered emmetropic by glasses sees distinctly at near points by using his accommodation. In doing this he has in effect added a convex glass to the glass he is using for distance (see § 179). If his accommodation is insufficient he must supplement it by an actual convex glass. The strength of this will depend on a member of considerations which will receive detailed discussion later (see § 215).

When we add a convex glass for this purpose, we in effect make the patient temporarily myopic, and the position of his far and near points is shifted just as if he were veritably myopic. Take, for example, the case of an emmetrope with an accommodation of 3 D. His far point is at infinity, his near point at 33 cm. If we add a $+2$ D he in effect becomes myopic 2 D and his near point is now shifted to 20 cm. (corresponding to the total of $3+2=5$ D of refractive power which he now has at his disposal for use at near points). Theoretically his far point should be at 50 cm, but as he is not in the habit of relaxing his accommodation when looking at near objects he still keeps using it with his convex glass on, so that his far point is usually not beyond 35 or at most 40 cm.; accordingly his region of accommodation is only 15 or 20 cm. instead of 30 cm. as would be the case with the myope.

[12] From πρέσβυς, old man, and ὤψ, sight.

IV. Visual Acuity.

184. Visual Angle.—Visual acuity is dependent on three factors, first, the transparency of the refractive media; second, the perfection of the refractive optical adjustment, i. e., the refraction of the eye; third, the sensitivity of the retina and the conductivity of the optic nerve. While, however, visual acuity is influenced by all of these factors, the tests for determining it are essentially tests of the sensory apparatus and as such should logically receive consideration along with the other tests of the latter in Chapter XVI. But these tests form such an integral part of the examination of the optical functions of the eye and their derangements that they are best described here.

Tests of visual acuity have to do partly with the form sense, partly with the light sense (see § 236 for definition of these terms). The ordinary

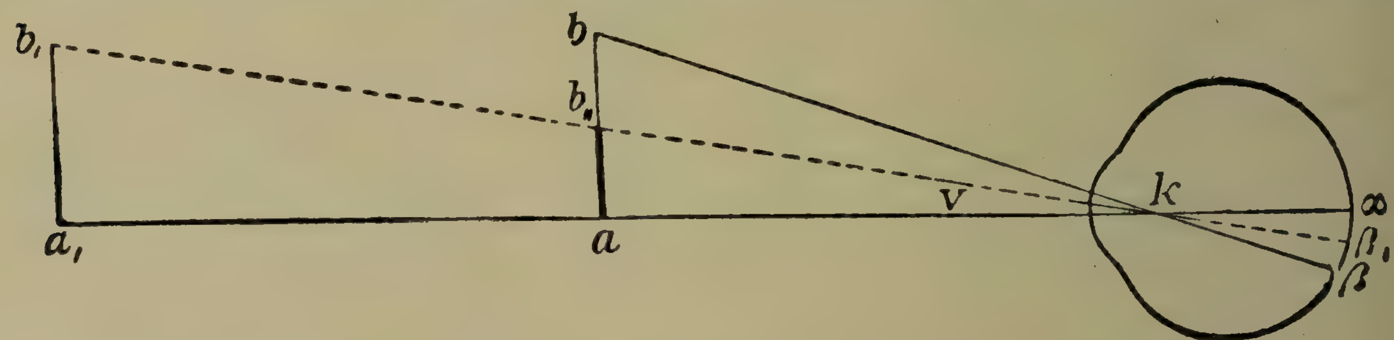

Fig. 92.—Behavior of the Visual Angle when Objects vary in Size and Distance.

visual tests are based on the former. That is, they concern the ability of the eye to distinguish minute differences of form. From this viewpoint the smaller the objects that an eye can distinguish or the greater the distance at which it can distinguish an object of given size, the greater is the acuity of vision that it possesses. Suppose, for instance, that the eye is able just to distinguish the object ab (Fig. 92) at the distance ak. Another better eye still distinguishes the object when it is carried twice as far off, to the distance $a_1 k$. The size of the retinal image $a \beta_1$ is in this case reduced to half of that in the former ($a \beta$)—that is, the visual acuity of the second eye is twice as great as that of the first. Instead of carrying the object $a\ b$ twice as far off, we may leave it at the same spot but make it half as small ($a\ b_{11}$). In this case, too, the size of the retinal image would be reduced one-half. In either case, therefore, a retinal image of the same size is obtained, and that, moreover, because the angle v remains the same. This angle is subtended by the rays which pass from the terminal points of the object through the nodal point of the eye to the retina. It is called the *visual angle* (angulus visorius), and is the true measure of the visual acuity. For estimating the visual acuity—i. e., the minimum visual angle—there are from the above example two ways open to us, both of which are made use of. We may take an object of given size and carry it off with us from the eye until the farthest point at which it can be recognized is reached. This, for instance, is done when we try to find at what distance an eye is able to count the fingers when extended. The second way consists in conducting the test at a constant distance, objects of different size being presented to the eye and the

attempt being made to find the minimum size which the object can have and still be recognized. This method is employed when we test the visual acuity with test types.

In estimating the vision, just as in estimating the refraction, the influence of the accommodation must be excluded for which reason the object used must be placed at a distance at which no accommodation of any notable amount is required, i. e., at a distance of 5 to 6 metres.

185. Tests of Visual Acuity.—What objects are best adapted for testing the visual acuity? A single dot, the distance of which from the eye is altered, is unsuitable because the visibility of a dot depends less upon the visual angle which it subtends than upon its luminosity. The fixed stars, radiant as they are, are nothing but mathematical points even when seen with the most powerful telescope; they have, therefore, a visual angle equal to zero, and yet they are seen very clearly. Every one can recall that a cross upon the top of a church spire, when sparkling in the lustre of the setting sun, was seen at distances at which the church spire itself was scarcely distinguishable. Hence we select for the test not one but two dots (or two parallel lines) and then determine the greatest distance from the eye at which they can still be perceived as separate objects. From this can be readily calculated the minimum visual angle, which for a normal eye amounts to about 1′. On the basis of this determination Snellen has constructed his test types. Snellen's *test types* are placed at a distance of 6 metres. They consist of letters of varying size arranged in rows. Each row contains letters of the same size, and has a number inscribed over it. This number gives the distance in metres at which the separate letters of the row appear to a normal eye under a visual angle of 5′. For example, this is the case with the letter *F* of the row with the superscription 12, when placed at a distance of 12 metres. Like all the other letters of the test card, it is inscribed within a square, whose sides are divided by partition lines into 5 parts each (Fig. 93). If, therefore, at 12 metres the whole square appears under an angle of 5′, the angle for each partial square is 1′. This is the minimum visual angle for the normal eye, and since the partial squares correspond to the details of the letter, these details consequently will still be made out by a normal eye. Hence the numbers standing over the letters give the distance at which each entire letter appears under an angle of 5′, and its details appear under an angle of 1′, and is thus the distance at which each row of letters can be made out if the vision is normal. If the distance at which the letter can be seen is greater or less than that indicated, the vision is greater or less than normal. The vision, therefore, may be expressed by the ratio between the distance *d*, at which the letter is actually recognized, and the distance *D*, at which it ought to be recognized, and which is inscribed over the letter—i. e., $S=\frac{d}{D}$ (where *S* stands for sight—denoted also by V = visus, or vision). Hence, an eye that sees the letter *F*

No. 12.

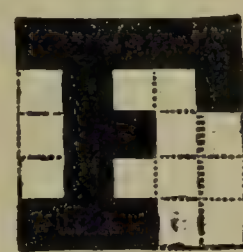

Fig. 93.—A Letter from Snellen's Test Card.

(Fig. 93) at 12 metres has $S=\frac{12}{12}=1$. As a general thing, however, we make the person under examination stand always at the same distance from the card—namely, 6 metres. If at this distance he distinguishes the lowermost row which has 6 written over it, he has $S=\frac{6}{6}=1$; but if, for instance he distinguishes only the uppermost letters, No. 60, $S=\frac{6}{60}$.[13] This is equal to $\frac{1}{10}$ of the normal visual acuity, but we always write the fraction in full without reducing.

To secure uniformity in notation the International Ophthalmological Congress in Naples in 1909 adopted the test types constructed by Hess for the purpose and have pronounced them international.

These *international test types* are designed for a distance of 5 metres because a space of six metres as demanded by the Snellen types is often not at the physician's command. The visual acuity found is expressed in decimals. These are placed alongside the several lines and denote the visual acuity which must be present if the two figures on the line are read at a distance of 5 metres. This means that, for example, the figures on the line marked 1.0 are seen under an angle of 5′ at 5 metres and their details appear under an angle of 1′, which just represents the degree of visual acuity that is regarded as normal. For number 0.5 this would be the case at 10 metres. If, however, one must bring the card up to 5 metres in order to recognize the figures on this line, he would need for this purpose twice as large a visual angle, and would therefore have only 0.5 of the normal visual acuity. This way of denoting the visual acuity is not as good as that of Snellen since it does not indicate the distance at which the test is made. The plate annexed (Fig. 94) is a reproduction of the international test types reduced one-third. Beside figures it also contains the rings proposed by Landolt. Analogous to the latter are the old E-shaped figures which are used for illiterates and children (see Fig. 95). These have the advantage that they are more readily understood, especially by children, than those of Landolt and that by turning the card upside down or horizontally entirely new combinations of symbols are produced.

When the vision has become so reduced that the largest letters of the test card can no longer be recognized at 6 or 5 metres, the patient must go up nearer to it, or we select, instead of letters or numbers, objects that are easier to distinguish— e. g., the outspread fingers—and try to find at what distance they can be counted. When the visual acuity is still worse, nothing can be distinguished but the movements of the hand before the eye. When even this is no longer the case, so that the eye simply distinguishes light from darkness, we say that qualitative vision is lost and that only quantitative vision—i. e., mere perception of light—is present.

Of particular importance are the tests of the vision in case of obstruction in the refracting media (corneal opacity, cataract). The vision should correspond approximately to the amount of dioptric obstruction that is visible. When the opacity is so dense that only quantitative vision is present, the latter is to be tested with a candle flame. We darken the room and station ourselves with a lighted candle opposite the patient. Now, by alternately holding the hand in front of the light and then withdrawing it, we test whether the patient can tell aright the change from light to darkness. We first make this test near by, and then withdraw farther and

[13] In this country and in England the distances are generally expressed in feet—i. e., $V=\frac{20}{20}$, or $\frac{20}{20}$ means that a patient at 20 feet saw the line marked 20.

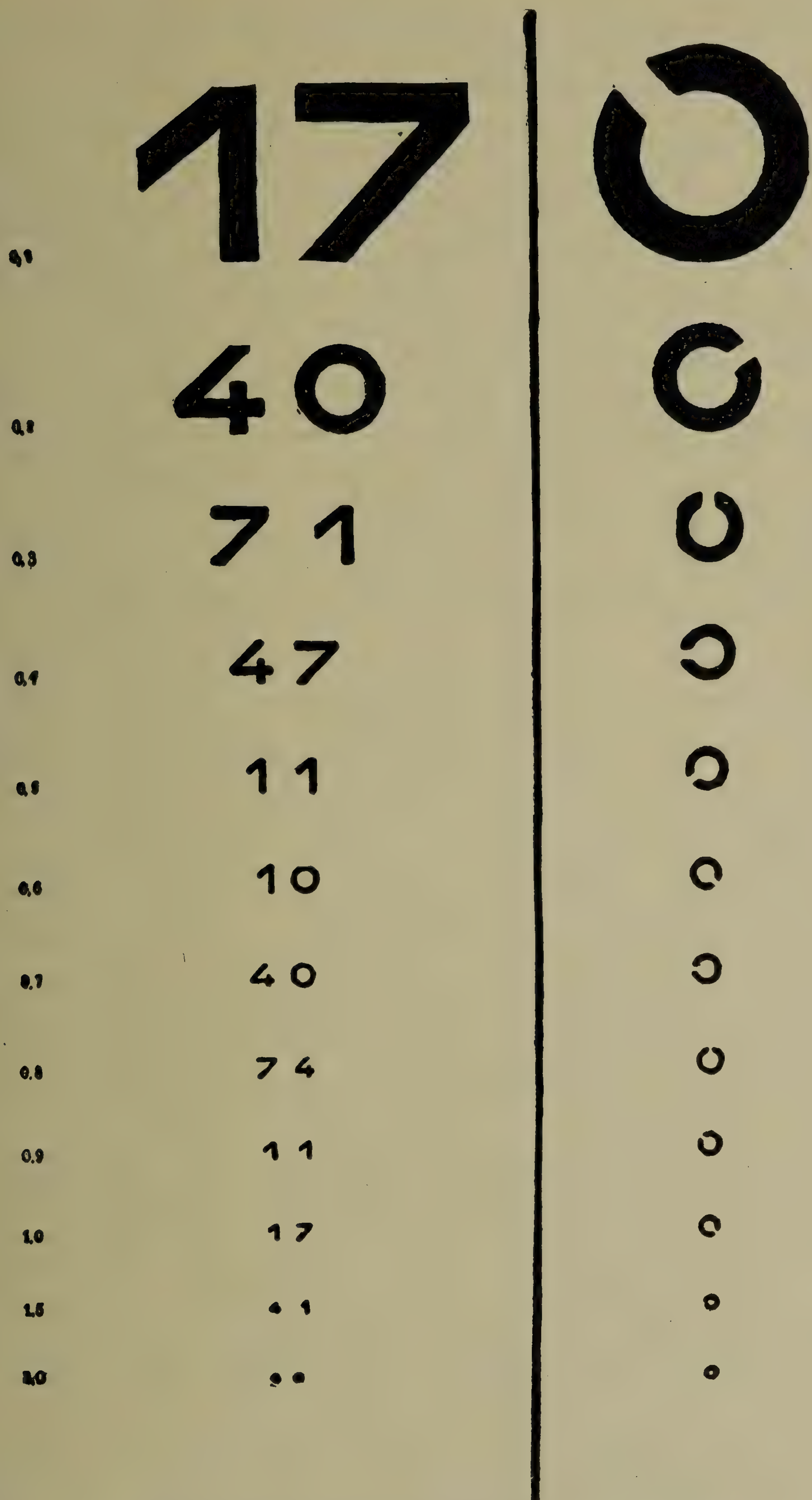

FIG. 94.—INTERNATIONAL TEST TYPES, REDUCED ONE-THIRD.

farther from the patient, so as to find the greatest distance at which he is still able to distinguish between the alternation of light and darkness. The degree of direct perception of light is thus determined. With regard to it we may say that perception of light in the centre and at the periphery is not abrogated by even the densest opacity. If the retina and optic nerve are sound, the glimmer of the candle must be recognized in a darkened room at at least six metres, and it should also be seen by the patient on all sides of him and its place be correctly given (see § 265). If this is not the case, the percipient portions of the eye are not normal.

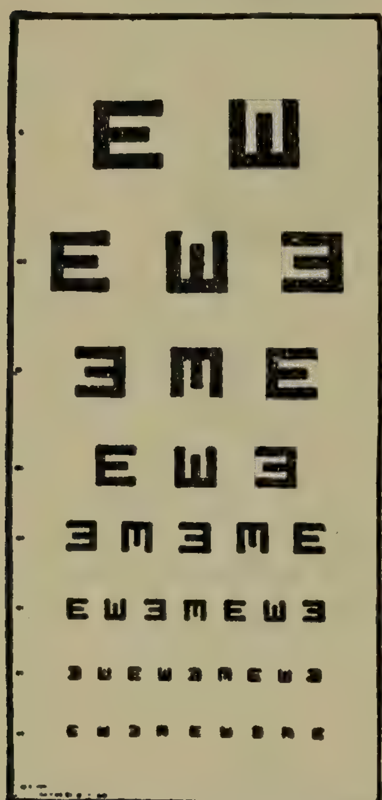

FIG. 95.—TEST TYPES FOR ILLITERATES, REDUCED ONE-SIXTH.

Küchler, in 1843, and Arlt, in 1844, were the first to introduce letters of different size (measured in lines) as a standard for determining the visual acuity. Ten years later Jäger published his scale of types, which soon acquired general acceptance, and which even at the present time is frequently employed.

The test types most extensively employed are those of Snellen. Snellen based them upon the assumption that the minimum visual angle for a sound eye amounts to 1′, so that No. 6 of the test types, the details of which appear under an angle of 1′ at 6 metres, can still just be read at this distance. He therefore assumed $S=\frac{6}{6}$ as the *normal visual acuity.* But we are not to suppose that this is absolutely the greatest visual acuity that there is. Most eyes in young persons see No. 6 at a greater distance, as far as 12 metres or even farther, so that they may possibly have $S=\frac{12}{6}=2$, or more. $S=\frac{6}{6}$ is accordingly to be looked upon simply as the minimum to be required of a normal eye; if the visual acuity sinks below this minimum, the eye is no longer to be considered as perfectly normal. The eyes of very aged persons are an exception, as such people even without any disease show a visual acuity less than $\frac{6}{6}$. Between 60 and 70 the normal vision in non-cataractous eyes may be taken as $\frac{5.5}{6}-\frac{5}{6}$; after 70 as $\frac{4.5}{6}-\frac{5}{6}$. The cause of this lies mainly in the lessened transparency of the refracting media and more particularly in the darkness of the nucleus and unequal refractivity of the lens in old age.

Some experiments seem to show that the *minimum visual angle* is much less than 1′. Thus a wire seen against the sky can be seen at such a distance that it subtends an angle of 1″ or less. But visibility here is due to the light sense rather than the form sense—the wire being seen, not because it subtends an appreciable angle, but because it affords a contrast with the sky just as a fixed star does. Nor even in the discrimination of test letters and similar objects is it solely the minimum visual angle, or the degree of separation of the parts of the letter, that is concerned. The *direction* of the parts is also determining and may be used as a test in the case of a single line (Roelofs).

In order that persons who have a vision $S>\frac{6}{6}$ may not have to go beyond a distance of 6 metres, Snellen's cards are provided with still smaller letters—namely, with the numbers from 5 to 2. These, moreover, can be used for those cases in which the physician does not have a room 6 metres in length but has at his command one of, say, only 5 or 4 metres. For persons who cannot read, there are cards with figures and with hooks (see Fig. 95). The international test types to indicate a visual acuity > 1 have numbers 1.5 and 2.0.

If the examining room is only 9 or 10 feet long, the distance can be doubled by placing the test card back of the patient and making him look at its reflection in a mirror at the opposite end of the room. In this case the card must have no symbols but those whose legibility is unimpaired by mirror reflection (T, X, 8, etc., or those shown in Fig. 95).

186. Effect of Illumination on Visual Acuity.—In tests made with test types and similar objects both the form sense and the light sense are involved. Variations in the latter cause considerable variations in the ability to distinguish the test letters, so that two persons having the same visual acuity under one lighting condition will show quite a different visual acuity under another (cf. § 258.) For this reason it is important that a definitely measurable standard illumination should be adopted for test cards, so that the results obtained by different observers shall be comparable. Furthermore the illumination should be alterable in a way definitely measurable, so that we can test the patient's ability to see under a known degree of reduced illumination (see § 168). This, of course, is possible only with artificial light. When daylight must be used this should be bright and diffuse, so as to illuminate the card well and uniformly and without glare.

If we have to make tests by daylight on a dark day we must correct the result obtained from the patient by our own visual power. If a physician, who with good illumination has $S = \frac{6}{6}$ (International 1.0), sees on a cloudy day only $\frac{6}{12}$ (International 0.5), the visual acuity of the patient found upon the same day must also be doubled. But, in view of the difference in light sense shown by different patients, such an estimate can be regarded as approximate only, and in cases of hemeralopia, for example, it would not answer at all.

As Ferree and Rand have pointed out, even minor errors of refraction are apparently associated with reduction of the light sensitivity, so that persons with a slight astigmatism, for example, fail to make minute discriminations when the illumination is reduced. Thus an astigmatic corrected by a +0.25 cylinder will when looking at a very poorly illuminated test card with his glass properly adjusted discern letters which he cannot make out if the cylinder is rotated so as to be somewhat off axis (cf. § 214).

187. Binocular Visual Acuity.—If we test the vision with both eyes together, the visual acuity so obtained is usually better than that gotten with either eye singly. This is the case even in anisometropia, where the visual character of one eye differs markedly from that of the other, and is usually the case even when the vision of one eye is quite poor. When this is not the case i. e., when the binocular visual acuity is less than that of the better eye tested by itself, we are led to suspect the presence of a muscular error, especially one of small amount which, when the eyes are used together, causes diplopia with overlapping and, therefore, confused images.

In more marked squint with either wide diplopia or with suppression, the binocular acuity is regularly equal to that of the fixing eye when examined singly. A very considerable difference between binocular and monocular acuity is found in those rare cases in which nystagmus is set up only when one eye is covered (latent nystagmus). Here the acuity with both eyes together is evidently very much greater than that with either eye alone.

188. Absolute and Relative Visual Acuity; Visual Requirements.—To know the *absolute visual acuity* of an eye we must test it in a condition of emmetropic refraction and with the accommodation completely relaxed. If the eye under examination is not emmetropic but has an error of refraction, it must first be corrected by glasses up to the point of emmetropia (E). The visual acuity, which an ametropic eye shows without glasses is its *relative visual acuity*, and furnishes no measure whatever of the general usefulness of the eye for vision.

This distinction must be borne in mind when we are testing the *sight in a case of eye disease*. As De Schweinitz well says, we shall often be misled as to the progress of disease and its effect on the sight, if, as is too often done, we test the visual acuity without attempting to correct as far as we can any existing error or of refraction. In iritis, for example, in which a temporary myopia of 1 or 2 D may develop, we shall get the erroneous impression that the vision has deteriorated greatly if we test the case without applying concave glasses.

Although the relative visual acuity (i. e., the acuity of the uncorrected eye) is not ordinarily an index of the usefulness of the eye, since in most occupations glasses can be used, yet there are certain conditions in which a low relative acuity is a handicap. Thus the exigencies of certain occupations require that there shall be good vision without glasses. This is particularly true of the *military and naval service*. In other occupations, too, e.g., the railway service and manufacturing of various kinds, in which glasses are constantly obscured by dust, grease, and moisture, they are a handicap, and those who do not see well without them are at a disadvantage. In the army, navy, merchant marine, and railway service, it is very important that a man have a good *light sense*—more so, in fact, than that he have a good visual acuity as determined by the test cards in the ordinary way. Good *color vision* is also often imperatively required. For the tests of both see Chapter XVI.

189. Visual Acuity at Near Points.—In determining the visual acuity for near the accommodation comes into play and must receive consideration accordingly. The testing of vision for near is made with fine print, especially with the lower numbers of Jäger's or Snellen's test types. In doing this, we try to determine two distinct things—namely, what is the smallest-sized print that can be read, and what is the least and the greatest distance at which it is legible.

Thus we say that a patient can read Jäger No. 2 as close as 25 cm. and as far as 50 cm. from the eye.

If this test is made with reading glasses, we can determine by it the region and approximately the range of accommodation of the eye when thus corrected. The range, however, is determined more accurately, in the way described in § 180.

It is evident from Fig. 92 that, the farther an object is from the eye the smaller is its retinal image, and hence the greater will be the visual power requisite for its recognition. Conversely, the retinal images enlarge as the object is approximated to the eyes. Hence, the smaller the objects are, the nearer we hold them to the eye. Very myopic eyes often have defective visual acuity, and yet pass as being excellent because they can distinguish extremely minute objects. But the only reason for this is that myopes are able to hold objects extremely close. Again, persons with diminished visual acuity like to bring objects up extremely close, so as to get quite large retinal images from them, and in this way make up for what the images lack in distinctness or the retina lacks in sensitiveness. Such persons are often wrongly regarded as very myopic.

The most scientific way of reading the visual acuity at near points is to indicate it just as for distance by a fraction, in which the denominator denotes the size of the print on the Snellen scale and the numerator the distance at which it is just legible.

Thus just as $V = \frac{5}{5}$ denotes that type which should be seen at 5 metres is read at 5 metres, so $V^1 = \frac{0.3}{0.5}$ denotes that near vision (V^1) is such that a type which should be seen at 0.5 metres is legible only when brought as close as 0.3 metres.

CHAPTER XIV

ETIOLOGY AND SYMPTOMS OF REFRACTIVE ERRORS

I. Evolution of Ametropia

190. Congenital Ametropia.—The eye of the new-born infant is very short. If it had the same refractivity, or in other words the same cardinal points as the adult eye it would because of its shortness have a hyperopia of 24 D. As a matter of fact, however, although nearly all infants' eyes are hyperopic, their hyperopia rarely exceeds 4 D. The optical system of the eye, therefore, must be much more refractive than in the adult. This excess of refractive power cannot be seated in the cornea, since this has about the same curvature as in later life (De Vries). It must then be in the lens, and, in fact, measurements show that in infants this has twice as great a curvature as in the adult (Holth, Stadfeldt). The infantile eye, accordingly, is one in which two opposing refractive states are combined, namely a very considerable curvature myopia seated in the lens and a still more considerable axial hyperopia due to the excessive shortness of the eye.

About 2.5 or, according to more reliable observations, 5 per cent of new-born infants are myopic, 15 per cent emmetropic[1], and nearly all the others have hyperopia of from 1 to 4 D (Randall, De Vries).

191. Postnatal Changes in Ametropia.—As the child grows the eye elongates and evidently at the same time the lens becomes less refractive—the former process diminishing the hyperopia, the latter the myopia. The effect of the elongation is usually predominant, so that there is a progressive tendency of the growing eye to lose its hyperopia and become emmetropic. Actual emmetropia, however, results in only a small number of the cases, the great majority of eyes either remaining hyperopic, although generally less so than at birth, or overshooting the mark and becoming myopic. This tendency of the eye to increase its refractive power usually keeps on during the whole period of growth. It seems to be most active between the ages of 6 and 16, during which time the percentage of cases of myopia steadily increases. After 16 it is generally but little marked, and in the great majority of cases it ceases altogether by 20. If it keeps on after 21 it must be regarded as an abnormal process and, if it affects an eye already highly myopic, is a dangerous condition since these eyes because of their excessive elongation suffer serious internal changes which may destroy the sight altogether. (See § 199.)

By the age of 16, according to the statistics made by Tenner from the very cosmopolitan population of New York City, 48 per cent of the school children have become myopic, 4 emmetropic, and 48 per cent remain hyperopic. In Germany, the proportion of myopes in those attending school is even greater than this. In other countries, on the

[1] As a contrast to this it may be noted that Tenner found no cases of emmetropia in a large number of school children of 5 and 6 years of age, examined under atropine.

other hand, it is less. Among the general population it is considerably lower than th figures would indicate.

In Europe and America the proportion of emmetropes in the adult population i comparatively small, although statistics vary. According to Roy, among the nativ Africans, emmetropia is the rule and myopia the rare exception.

Little is known about the development of astigmatism. It is founc early in life and probably, in many cases at least, is congenital. It usuall shows no very great tendency to change during the growing period, remaining about the same from early childhood till adult life.

Between 20 and 50 the refraction of the eye undergoes, as a rule, comparatively little change. Moderate changes in the astigmatism and especially moderate increase in even a slight myopia may occur. These change may be due to alterations in the structure, curvature, or position of the len or to altered curvature of the cornea, caused, e. g., by pressure of the lids. Between 40 and 50 the accommodation becomes so inefficient that usually a reading glass has to be employed, but there is no special tendency to a change in the refraction at this time.

192. Changes in Advanced Life.—Beginning at about the age of 50 it commonly, but by no means invariably happens that a decrease in the refractive power of the eye takes place, causing an increase in hyperopia or a diminution in myopia. The reduction in refractive power amounts, as a rule, to 1 or 1.5 D between the ages of 45 and 60 and, according to Straub, there is a further loss of 1 D between 60 and 70. The changes here are in all probability lenticular, being due either to a general increase in density of the lens, which renders it more homogeneous and hence less refractive as a whole, or to flattening of the cornea and lens.

In some cases the reverse process takes place, a moderate amount of *myopia* developing with often a rather considerable amount of astigmatism. This, again, is due to lenticular changes—particularly nuclear sclerosis—which in many cases are the precursors of cataract or accompany its development. After 50 the astigmatism also often changes in kind and amount. The change usually found is such as would result from an increase in the relative refractive strength of the horizontal meridian of the eye; i. e., a direct astigmatism tends to diminish and an inverse astigmatism increases, in some cases the latter actually replacing the former. These changes in astigmatism are rarely considerable except in the cases in which myopia develops.

II. Hyperopia.

193. Normal and Pathological Hyperopia.—The ordinary hyperopia of young persons and adults is an axial hyperopia due to the fact that the eye, which was primarily too short has not in the process of growth elongated to the point of producing emmetropia (see § 191). A very large proportion of adults have a hyperopia of this sort, which may therefore be regarded as a *normal* condition.

Other forms of hyperopia which are due, not to shortness of the eye, but to changes in the curvature, density or position of the lens or in the

curvature of the cornea, are regularly the result of pathological changes or of abnormalities of development. The conditions causing this *pathological* variety of hyperopia include facets, scars, or a congenitally low curvature of the cornea, causing the latter to be flatter than normal; recession of the lens (see § 167); and changes in composition or density of the lens occurring especially in incipient cataract. A high degree of hyperopia is produced when the lens disappears from the pupillary area either from being luxated (§ 662) or because it has been removed from the eye altogether (aphakia) [2]. In some way not well understood hyperopia also at times develops in diabetes.

Refraction in Aphakia.—In an emmetropic eye of normal optical construction, theory shows that removal of the lens causes a hyperopia of slightly over 10 D. And, in fact, removal of the lens does usually produce a hyperopia of between 10 and 11 D. In aphakia produced by cataract extraction this hyperopia is usually accompanied by an astigmatism, generally inverse, due to the flattening of the cornea that results from the cicatrization of the operation wound. This astigmatism regularly diminishes during the first months after the operation, but even so may amount to 2 or 3 D.

If the eye is affected with an axial ametropia of n D before removal of the lens, the strength of the correcting glass after removal is roughly equal to $10+0.4\ n$. That is a previous myopia of 24 D would now be corrected by $10-0.4\times24$ or $+0.4$ D; i.e.. the eye would be almost emmetropic.

The aphakic eye is also *destitute of accommodation.* Hence any one glass corrects it for a single distance only. It is true that as the correcting glass is usually very strong slight alterations in its position produce corresponding changes in its effect (see § 156) so that the eye may in this way be adjusted for different distances (pseudo-accommodation). Generally speaking, however, the aphakic eye requires two glasses, one for distance and the other, usually about 3 D stronger, for near. When, as occasionally happens, the aphakic patient can see distinctly at near with his distance glasses, the apparent accommodation is usually due to the fact that he is looking through a stenopæic aperture (§ 168), e. g., a very small pupil or a minute opening in an opaque capsule. But in some very rare cases an apparent accomodation is present in aphakic eyes to which this explanation does not apply.

Two varieties of axial hyperopia may be regarded as pathological, namely the form due to *protrusion of the retina* from tumors or exudations and that in which the eye remains excessively short all through life so that the hyperopia is *extreme.* In the latter case we regularly find that the eye is no longer normal as a whole. It is abnormally small even from the time of birth (slight degree of microphthalmus), and many of these eyes show other signs of disordered development, such as a strikingly small cornea, marked astigmatism, deficient visual acuity due to incomplete formation of the retina, and other congenital anomalies.

These cases of extreme hyperopia are very rare. In axial hyperopia, in fact, even moderately high degrees of refractive error—those in excess of 4 or 5 D—are uncommon. They are much rarer, indeed, than the high degrees of myopia. They can be recognized at once by external examination of the eye, which shows that the eyeball is distinctly diminished in size, and that the anterior chamber is shallower and the pupil more contracted than usual. If the eye is turned strongly inward, we see that the equatorial region of the ball, which comes into view in the outer part of the palpebral fissure, presents a particularly sharp curve as it turns backward, and thus give evidence of the shortness

[2] Aphakia from ἀ privative and φακός, lentil.

of the axis of the eyeball. The ophthalmoscope shows that the interior of the eye is sound. The ordinary hyperopic eye, accordingly, even when the hyperopia is of quite high degree is an optically defective but otherwise healthy eye.

194. Course.—As we have seen the hyperopia, originally present at birth in most eyes tends to diminish steadily in amount, often being transformed into myopia, so that the proportion of hyperopic children gradually decreases from 80 to 90 per cent at birth to less than 50 per cent at 16. After 20 hyperopia does not change much in amount, although in middle life it may show an apparent increase, because a part concealed in youth by the accommodation then becomes manifest (see § 195). In advanced life hyperopia tends to increase and some eyes, previously emmetropic or slightly myopic may become hyperopic (see § 192).

195. Latent and Manifest Hyperopia.—As we have seen (§178), a hyperope can correct his error by accommodation which enables him to get distinct vision for distant and even for near objects. This tendency to accommodate, which is involuntary and of which he is usually quite unconscious, becomes so habitual that he is unable to relax his accommodation completely even when convex glasses are placed in front of the eye, which render his accommodation superfluous, or even a disadvantage. Thus, with a glass which completely corrects his hyperopia, the hyperope sees at a distance no less badly, and as a general thing much worse than with his naked eye.

That portion of his hyperopia which he thus persistently conceals by accommodation is called the *latent* hyperopia (*Hl*); that portion which he can disclose by relaxing the accommodation and which is measured by the highest convex glass that he will accept is the *manifest hyperopia* (*Hm*). Both together constitute the *total* hyperopia (*Ht*).

The ratio between the manifest and the total hyperopia depends partly on the range of accommodation, partly on other factors. Children have a large range of accommodation, with which they are able to conceal a considerable degree of hyperopia; yet in them the accommodation is very often flexible and readily relaxed so as to permit nearly or quite all the hyperopia to become manifest. On the other hand, between 40 and 46, the eye just at a time when it is finding difficulty in maintaining its accommodative effort, sometimes shows considerable disinclination to relax, so that 2 or even 3 D of hyperopia may be latent then. Later on, however—after 50—the accommodative effort is given up, and then all the hyperopia becomes manifest.

Till the period when the compensatory accommodative effort thus fails altogether, the amount of total and therefore the amount of latent hyperopia can be determined only by putting the eye under a drug like atropine or homatropine, which paralyzes the ciliary muscle and thus abolishes all accommodative action.

196. Symptoms.—Hyperopes would have indistinct vision both for distance and near points if they possessed no accommodation. The hyperope, therefore—in contradistinction to an emmetrope or myope—must accommodate even *when looking at a distance,* as in order to see distinctly he must correct his hyperopia by means of his accommodation. Whether this is possible or not depends upon whether the accommodation when compared with the amount of hyperopia is powerful enough to bring the near point within infinite distance (Fig. 96 A). If this is not the case, and the near

point lies behind the eye, it is impossible for the hyperope to see distinctly at distance without a glass (*absolute hyperopia*). If the near point lies within infinity, distinct vision for distance can be attained by an appropriate output of the accommodation (*facultative hyperopia*).

The extent to which hyperopia can be concealed by accommodation depends not only upon the degree of the hyperopia, but also upon the strength of the accommodation. But this latter changes with age, diminishing steadily with advancing years, and in old age becoming almost zero.

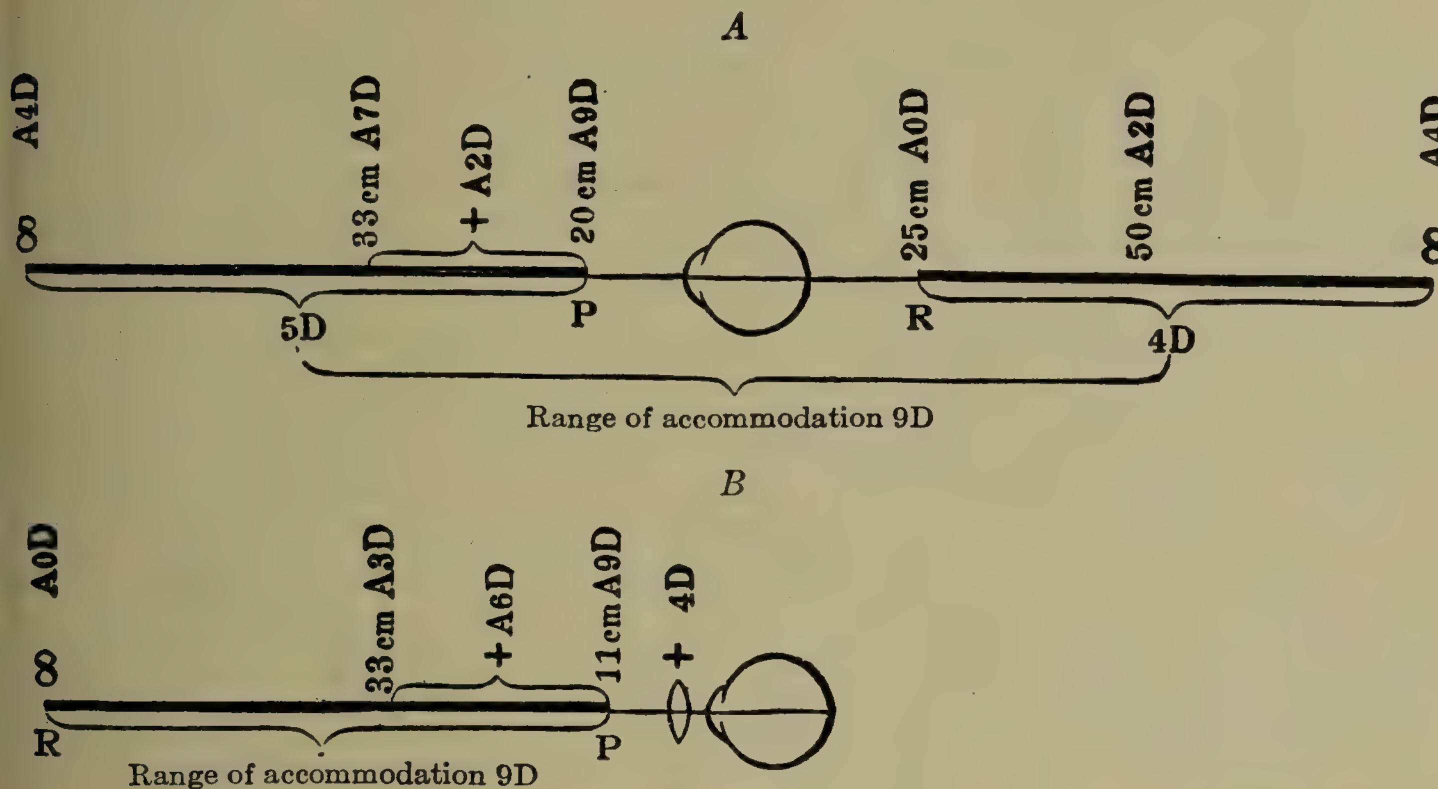

FIG. 96.—REGION OF ACCOMMODATION OF THE EYE OF A MAN 23 YEARS OF AGE WITH A TOTAL HYPEROPIA OF 4 D AND A RANGE OF ACCOMMODATION OF 9 D.

A. If the accommodation is completely at rest, the eye is adjusted for its far point, *R*, which lies 25 cm. behind the eye. With an expenditure of accommodation amounting to 2 D the point for which the eye is adjusted shifts to a distance of 50 cm. behind the eye, and if the range of accommodation of this eye amounted to no more than 2 D, the near point of the eye would be situated here (§179, Note 11). There would then be an absolute hyperopia, i. e., the eye would not be able to see distinctly in the distance. It is only by an expenditure of 4 D of accommodation that the eye is adjusted for infinite distance (facultative hyperopia). By further exertion of the accommodation the point for which the eye is adjusted shifts from infinity to a finite distance from the eye; e. g., with an expenditure of 7 D of accommodation to 33 cm. and with an expenditure of 9 D of accommodation to 20 cm., which, therefore, represents the near point, *P*, when $A = 9$ D. To produce an adjustment for 33 cm. an expenditure of 7 D of accommodation is required, so that of the total range of accommodation but 2 D are left for the positive portion of the range, while 7 D are already put into play i.e. are left for the negative portion of the range. In this case, therefore, continuous work at 33 cm. would be impossible.

B. Displacement of the region of accommodation by a correcting convex glass. The far point now lies at infinite distance, the near point at 11 cm., as in the case of an emmetrope in whom $A = 9$ D. For a working distance of 33 cm., 3 D of accommodation are required, so that 6 D are left for the positive portion of the range and 3 D for the negative.

Even the small amount left is usually no longer used in correcting distant vision so that even a slight hyperopia, becomes absolute in advanced age.

197. Near Vision.—While the hyperope needs accommodation even for distant vision, this is much more the case for *seeing near by*. The amplitude of accommodation, *A*, of the hyperope is the same as that of the emmetrope and myope. To be sure, the near point in hyperopes lies farther off from the eye, but this is simply because a part of *A* is employed in the correction of hyperopia, and only the remainder is left to effect the adjustment for shorter distances. For the same reason the hyperope requires for a certain working distance more accommodation than the emmetrope, the excess, in

fact, corresponding precisely to the amount of the hyperopia. Let us assume that work has to be done at a distance of 33 cm. For this purpose the emmetrope must use an accommodation of 3 D. A hyperope with $H = 4$ D also uses the same amount of accommodation; but he must employ 4 D more to conceal his hypermetropia, so that altogether he must make an accommodative effort of 7 D (Fig. 96 A). Now, as his range of accommodation is no greater that of an emmetrope, this great amount of accommodation causes him proportionally more trouble. He may be said to be always dragging about with him a deficit in his accommodation, namely, the quota of the latter necessary for the correction of the hyperopia—a deficit which causes him to tire quickly when doing near work (asthenopia). At first, vision near by is distinct and the work goes on well; but after a little while the objects, print, near work, etc., begin to grow indistinct and are blurred as though enveloped in a slight haze. This is owing to the fact that the over-strained accommodation gives way, and the eye then ceases to be properly focused. A short period of rest, during which the eyes look at distant objects or are kept closed, enables them to continue the work. But the same obscuration soon sets in again and compels another pause. These periods of enforced rest are the more frequently repeated and are of greater duration the longer the work is kept up. With them are associated pain in the eyes, and more especially pain in the forehead and headaches. The symptoms just described at first make their appearance only after quite prolonged work—i. e., toward evening. But afterward they keep coming on earlier and earlier all the time, so that the work has to be set aside after even a short period of exertion. After quite a prolonged rest—for example, after the repose of Sunday or upon laying work aside for several weeks—the symptoms probably disappear for a number of days in succession, but only to appear again in the old way and in an even more aggravated fashion. They are dependent upon an exhaustion of the ciliary muscle, and are hence comprised under the name of *asthenopia accommodativa* to distinguish them from asthenopia muscularis (see § 340), and asthenopia nervosa (see §245).

The injurious effect of hyperopia upon vision at near points furthermore finds expression in the fact that *presbyopia* sets in earlier than in emmetropic eyes. At the same age—i. e., with the same range of accommodation—the near point of the hyperope is situated farther from the eye than is that of the emmetrope. In an emmetrope of about forty years with a range of accommodation of 5 D, P lies at 20 cm. ($100 \div 5 = 20$). A hyperope having a hyperopia of 2 D would at the same age and with the same range of accommodation have his near point at 33 cm. (corresponding to 3 D), since he has to use 2 D of his range of accommodation for correcting his hyperopia. Such a hypermetrope, therefore, would be already on the threshold of presbyopia at the age of forty, i. e., some six or seven years earlier than an emmetrope.

Myopia is a state which is the direct opposite of hyperopia, and yet there are cases in which these two conditions might be confounded with each other. When the hyperopia has attained a very high degree, even the strongest effort of accommodation proves

insufficient for rendering the vision at near points distinct. Then the hyperope gives up altogether the attempt to focus his eye for near points and holds the object as close as possible so as to get large retinal images, just as amblyopic patients do (see § 189). In this way very small print is often read well at a distance of a few centimetres, and, as at the same time distant vision is pretty bad, such a condition may easily be regarded as myopia. Examination with glasses and with the ophthalmoscope, however, will at once rectify the diagnosis.

The difference in the capacity of the emmetropic, the myopic and the hyperopic eye for close work is explained by the difference in the position of their *region of accommodation* (see § 179). In emmetropia it has its normal position, in myopia it is carried inward (3, Fig. 85), and in hyperopia it is displaced outward. For example, suppose that an emmetrope twenty-three years old has a range of accommodation of 9 D. His region of accommodation then extends from ∞ to a point 11 cm. from the eye where his near point is situated. Let us compare with this an eye having the same range of accommodation but with a total hyperopia of 4 D. In this case R lies 25 cm. ($100 \div 4 = 25$) behind the eye (Fig. 96 A). To change its adjustment from this far point to infinity, the eye must employ 4 D of its accommodation. Hence, out of its whole range of accommodation of 9 D the eye has only 5 D left, and by means of this it is enabled to approach to within 20 cm. of the object. This distance, therefore, is the near point (P) of the eye. Hence the region of accommodation of the latter is displaced in comparison with that of an emmetropic eye having the same range, and in such a way that the near point is made to recede 9 cm. farther away (from 11 to 20 cm.), while at its other end a part of the region of accommodation is made to lie behind the eye. But as this latter portion can not be utilized, and, on the other hand, the recession of the near point hinders the vision of objects close by, the displacement of the region of accommodation is unfavorable to the availability of the eye for working purposes. The use of a convex glass removes the accommodative troubles, since it brings the region of accommodation into a more available position—it now, in fact, lying entirely in front of the eye (Fig. 96 B).

The calculation of the range of accommodation in hyperopia is made according to the same rules as in emmetropia. P can be found directly, R is determined by the convex glass which corrects the error. $A = P - R$; hence in the example selected, $A = 5D - (-4D) = 9D$. R must be taken as a negative because it lies behind the eye, i.e., represents a deficiency in refractive power (§ 172).

III. Myopia

198. Varieties.—The fact that parallel rays come to a focus in front of the retina, which constitutes the essence of myopia, may in general arise in two ways:

1. The *refractive power of the eye is abnormally great*, so that parallel rays are made too convergent, the retina in this case being in its normal situation (*refractive myopia*). The cause of the increased refractive power may lie in the cornea or in the lens. In the *cornea* it is increased *curvature* that leads to myopia. This is found most markedly in ectases of the cornea of the most diverse sort, and especially in keratoconus. In this case the myopia is always associated with considerable astigmatism.

The *lens* can cause abnormal elevation of the refractivity of the eye either through increased curvature, through changes of *position*, or through augmentation of density. The following cases fall under this head: (*a*) Accommodation, which increases the curvature of the lens, may be kept permanently in action, and as long as this spasm of accommodation continues myopia will be present (factitious or apparent myopia). The latter disappears when the accommodation is paralyzed by atropine. (*b*) In

luxation the lens takes on an increased curvature because the tension exerted by the zonula is removed. If the case is one of luxation into the anterior chamber, the forward displacement of the lens contributes to the increase in refractive power, since with the lens the principal focus of the whole dioptric system is shifted forward (see § 165). (*c*) Slighter changes of position in which the lens pushes forward as a whole so that the anterior chamber is rendered somewhat more shallow, cause a moderate myopia. (*d*) Myopia due to increase in density of the lens (so-called *index myopia*) not infrequently sets in at the beginning of senile cataract (see § § 192, 638). Whether the myopia that at times develops in diabetes is due to the same cause is uncertain. It is possible also that some of the myopia in incipient cataract is due to changes in the curvature of the refractive layers (isoindicial surfaces) of the lens. For the myopia of iritis, see § 681.

All the above varieties of myopia may be classed as *pathological.*

2. The refractive power of the eye may be normal, so that parallel rays come to a focus at the customary spot, but the retina may lie too far back. The cause of this is an elongation of the axis of the eye, hence this sort of myopia is called *axial.* The distention of the sclera, to which the elongation of the eye is due, usually affects the posterior division of the sclera, which is bulged out posteriorly (see § 203). This is the ordinary typical form of myopia, which in its lower degrees may be regarded as *physiological,* being the result of a regularly occurring, but in this case overacting developmental process (§ 191). In its higher degrees it is associated with changes in the background of the eye and must then be regarded as pathological.

199. Development and Course.—Myopia is only exceptionally congenital. At birth about 5 per cent of infants are myopic. The proportion increases in early life, so that at the age of 6 it amounts to perhaps 9 per cent. Thereafter it *increases* more rapidly and quite steadily until by the age of 16 a large number of children are myopic. During this period, which corresponds to the period of rapid bodily growth, not only do more and more cases of myopia develop all the time, but those in whom it has appeared become more and more myopic. Such an increase of myopia after it has once developed, although progressive, is often discontinuous, the refractive power of the eye remaining the same for a year or two, then suddenly increasing. Moreover, the progress of the myopia in the two eyes is often dissimilar. As a rule, the right eye is the more myopic.

The development of myopia during childhood is the net result of two opposing processes going on simultaneously at this time, viz., a progressive reduction in refractivity produced by a steady flattening of the excessively curved infantile lens, and a progressive increase in refractivity produced by steady elongation of the eye (see § 191). In the development of the myopic eye, the former process apparently goes on as usual, but the latter goes on excessively, so that by the time full growth is reached, the eye has a normal optical system but is absolutely too long. This excess of length, in fact, accounts for all or nearly all of the refractive error, so that from the latter

we can compute how long the eye is. We simply have to remember that each addition of 3 D to the myopia means an addition of 1 mm. to the length of the eye.

In some cases, according to Risley, the distension in ordinary myopia affects the front rather than the back of the eye. In this case the above rule would not hold, since part of the increase in refraction is due to bulging of the cornea.

Most cases of myopia are of low degree (less than 8 D even at their maximum period of development), and come to a stop after the completion of the body growth (*stationary myopia*). In other cases, however, the myopia attains a considerable height (10 D or more) even in youth, and then does not remain stationary, but increases steadily during the whole life, so that finally it reaches the greatest possible degree (*progressive myopia*). In such cases the myopia may amount to 20 or even 30 D. It is mainly these cases that lead to destructive changes in the interior of the eye, and that cause myopia to appear in the light of a disease of the eye, and of a serious disease, too, which in advanced age often gives rise to amblyopia or even blindness.

Apart from these serious cases, a certain amount of progress occurs at times in even slight cases of myopia in adults. Such increase is usually very moderate and is probably due not to elongation of the eye but to changes in curvature of the cornea or lens or changes in position or density of the lens.

In advanced life myopia may diminish or if slight may disappear, or on the other hand may increase (see § 192).

In advanced life myopes often believe that they are becoming less near-sighted because they see better at a distance without glasses than they used to do, and yet tests show no diminution in the myopia. This improvement in vision depends upon the fact that in old age the pupils become more contracted, and hence, in looking with the naked eye, the diffusion circles are smaller.

200. Etiology.—A vast number of statistics have been accumulated showing, first, that, especially in the schools, myopia regularly increases both in prevalence and in degree during the growing period and, second, that it occurs especially among those who are using their sight continuously on fine print or on fine, close work. Thus, it occurs particularly among students, tailors, seamstresses, compositors, lithographers, etc. On the other hand, it is not often found in savages. Hence, the inference has been drawn that it is caused essentially by overstraining the eyes for near work—the effect being ascribed to excessive convergence or excessive accommodation. Attributed particularly to this cause is the form of myopia occurring in schools, and increasing in them steadily from the lower grades to the higher (*school myopia*). We must remember, however, that the mere fact that more cases of myopia are found in the later years of school life than in the earlier is no proof that the school work caused the trouble. Since myopia is due simply to an elongation of the eye carried to an extreme, and since up to the age of 16, at least, all eyes keep steadily elongating, some

more and some less, the number of cases of myopia must naturally increase year by year, even though the eyes are not used at all for near work. Moreover, statistics are not wanting that seem to oppose those above cited as to the special prevalence of myopia among the cultured classes. Thus, myopia of even high degree is found among those who never use their eyes for near work at all, e. g., among the Bedouins, Egyptians (among these it is very frequent), and other illiterates [3]. Some, therefore, argue that there is some congenital anatomical basis for myopia which would be effective in causing its progressive increase whether the eyes were used for near work or not. In any case, even if straining the eyes in near work is the main cause of myopia, it cannot be the sole cause, since not all, but only a fraction of those who are subjected to this strain actually become near-sighted. In this fraction, therefore, special additional factors must be present which favor the development of the myopia due to near work. The following are the factors of this sort that we know of: 1. A *predisposition* to myopia, which doubtless has its seat in definite anatomical conditions, such as too slight resistance of the sclera, peculiarities in the relations of the ocular muscles or of the optic nerve, etc. Since anatomical peculiarities are apt to be inherited, the hereditary character of myopia is also readily explainable. The children of near-sighted parents are not, to be sure, born near-sighted; but if they are exposed to those conditions which favor the development of near-sightedness, they show a greater tendency to become myopic than do the children whose parents have normal sight. 2. Those circumstances which compel *too great approximation of the work,* and thus require an abnormally great accommodation and convergence. This is the case when particularly fine work has to be done, or when work is carried on with insufficient illumination, and also when the visual acuity is diminished (by maculæ of the cornea, opacities of the lens, astigmatism[4], etc.), so that it becomes necessary to bring the objects closer than usual (see § § 189, 610, 645.

Since elongation is a process that takes place in all growing eyes and it is only when this process is carried to an excess that myopia results, we must conceive of myopia as being due essentially to such a disproportion between the elongating force and the resistance offered to it, as renders the former unduly predominant. In this view we may say that myopia develops either because the elongating force is excessive or because the resistance offered is too weak; in the latter case the coats of the eye at its posterior pole being too distensible. In all probability both factors are concerned, but from the considerations adduced in the preceding paragraph the deficient resistance of the eye would seem the more important of the two.

Among the theories which have been advanced to account for the development of the elongation of the eye which lies at the bottom of myopia are the following (1) inflammatory processes in the chorioid and sclera (sclerotico-chorioiditis posterior), by which the sclera is rendered softer and more yielding (Von Graefe). (2) A congenital defect in

[3] It must be admitted, however, that here the great frequency of corneal opacities among these people may have a good deal to do with causing their myopia.

[4] The effect of astigmatism is considered particularly important. Irregular astigmatism and, according to some, inverse and oblique astigmatism are of especial significance in causing myopia.

development causing diminished rigidity of the coats of the eye (Schnabel). (3) Distension of the eyeball from heightened intraocular pressure due to physical effort (Edridge-Green). (4) Elongation of the eye from pressure exerted on it by the external ocular muscles, especially in the act of convergence. It has been supposed that either the internal and external recti which in the act of convergence are stretched more tightly upon the eyeball, or the two obliques which surround the eyeball, like a sort of noose, may produce this effect (Stilling). The muscles last named are, furthermore, so situated that they press upon the points of exit of some of the venæ vorticosæ from the eyeball, and may thus produce venous congestion in the latter. Ochi thinks that for the external muscles to produce myopia the sclera also must be unduly weak. Besides causing pressure by the external muscles, convergence might produce distention of the posterior pole of the eye by displacing the latter outward so that it is pulled upon by the optic nerve. This action would be particularly looked for when the optic nerve is, relatively speaking, too short (Hasner, Weiss). (5) A. Wood believes that the small size of the ciliary muscle, which is so frequently found in myopia, is not, as stated in § 205 the result, but actually the cause of the nearsightedness. Accepting Thomson's theory (§ 77) that the ciliary muscle helps to effect outflow from the eye, he believes that congenital deficiency of this muscle by retarding outflow increases the intra-ocular pressure and hence causes the eye, which is distensible during early life, to expand. When the myopia is once established, the non-action of the ciliary muscle, due to the failure of myopes to use their accommodation, would tend to make it increase, and a contributing factor would be the dilatation of the pupil, which is usually present in myopes and which would also help to impede outflow from the eye. There are a number of clinical observations which seem to favor this theory—one being that correction of myopia by glasses, which call the accommodation into play, seems to retard the development of the near-sight.

Myopia is found equally among males and females. Its incidence in different nations varies greatly, it being specially prevalent in the peoples of central Europe.

201. Symptoms.—Myopes see indistinctly *at a distance*, because of the presence of diffusion circles. To make these smaller, and thus see better, they squeeze the lids together, and in this way produce a stenopæic slit. In fact, it was from this habit of blinking that the name myopia originated. In myopia as in other refractive errors the diffusion circles often cause monocular diplopia—test-types and like objects appearing double.

Myopes see well *near by*, and, moreover, have the advantage that they need use little or no accommodation for this purpose. The range of accommodation has the same relation in the myopic eye (if the high degrees of myopia are excepted) as in the emmetropic eye. Only, since the far point lies at a finite distance, the whole region of accommodation is placed closer to the eye, as can be seen from Fig. 85 (No. 3), in which the region of accommodation lies between 10 and 5 cm. in front of the eye. In working at close range, therefore, the myope needs to use less accommodation than the emmetrope, or even no accommodation at all. Suppose, for instance, that work has to be done at a distance of 33 cm. In this case the emmetrope requires an accommodation of 3 D ($100 \div 33 = 3$). A myope whose myopia equals 1 D needs only 2 D of accommodation, and one having a myopia of 3 D needs none at all, since his far point lies at the working distance. As soon, therefore, as myopia has reached a certain degree, the accommodation ceases to be used (it being presupposed that no glasses are worn). Hence, in myopia of high degree the range of accommodation, is as a rule, not normal, but diminished.

For the same reason, in myopes *presbyopia* sets in later than in emmetropes, or does not set in at all.

To be sure, the diminution in elasticity of the lens is produced in a myopic eye just the same as in any other, but practically it does not make itself so perceptible. If a man has a myopia of 3 D, his far point lies at 33 cm., and will always remain there though he be ever so old. During his whole life, therefore, there will be distinct vision at this distance, the only difference from what existed previously being that the patient, when at an advanced age he has lost his power of accommodation, will no longer be able to see closer than 33 cm.—a thing, however, which there is usually no necessity of his doing in any case. Such a myope, therefore, does not become presbyopic at all. Those having myopia of less degree do become presbyopic, but do so later than emmetropes.

202. Symptoms in High Myopia.—The *troubles* that myopes complain of, vary according to the degree of myopia. In the lower grades of myopia distant vision is indistinct, and yet often suffices for ordinary purposes, so that many myopes of this sort do not use glasses. For near work moderately near-sighted eyes are generally regarded as serviceable, because they do their work with less accommodation, and, moreover, either become presbyopic late or do not become so at all.

It is otherwise with the high degrees of myopia. In this case not only is the complaint made of indistinct vision at a distance, but also of inability to keep on with work near by for any length of time; for, owing to the short distance from the eyes at which the far point lies, a considerable effort of convergence is required—an effort which, moreover, is often rendered difficult because the impulse to converge is diminished, owing to the abolition of the accommodation. Hence, exophoria and, as a result of it, muscular asthenopia develop. By a transformation of this latent deviation into a manifest one, an exotropia may be set up, a condition, therefore, which is frequently met with as a result of high myopia.

In myopia of high degree it is often the case that satisfactory distant vision is not attained even by glasses, because morbid changes exist in the fundus. For the same reason, vision close by is frequently defective in spite of the great approximation of the object. Moreover, complaint is made of rapid tiring of the eyes, of great sensitiveness to light, and of muscæ volitantes. The last-named phenomenon is also, to be sure, found in healthy eyes (see § 668), but the myopic eye is more apt to see muscæ volitantes, and sees them in greater number. This arises from the fact that myopic eyes without glasses see everything indistinctly; and upon a hazy background, such as is caused by this indistinct sight, opacities are better projected. Moreover, in the higher degrees of myopia pathological opacities of the vitreous are apt to be present. Muscæ volitantes are not infrequently a source of constant annoyance and worry to myopic patients.

In myopia of 10 to 12 D the vision may still be $\frac{20}{20}$, especially in children. Usually, however, it is not better than $\frac{20}{30}$ and may be only $\frac{20}{40}$. In myopia of over 12 D vision is rarely better than $\frac{20}{40}$ even when there are no complications, and when the myopia exceeds 15 D, vision is generally $\frac{20}{70}$ or $\frac{20}{50}$ at best. Moreover all persons who have a great degree of near-sight see worse and worse as their age increases, because not only is the myopia augmented, but the complications of myopia tend to develop more and more.

203. Objective Signs.—Objective examination of a near-sighted eye shows that it is longer than normal, the elongation being due to the distention of the sclera at the posterior pole (Fig. 97). In marked myopia the enlargement of the eye-ball is recognizable even in the living eye. The eye projects far forward (pop-eye); and when it is turned well in toward the nose, so that the equatorial region appears in the outer portion of the palpebral fissure, this does not, as in the case of the normal eye, make a sharp curve in turning backward, but runs back without much curving—almost straight, in fact. Very myopic eyes are also usually characterized by a deep anterior chamber and a dilated pupil.

The most important changes in myopia are those which are situated in the *fundus*, and can be recognized by ophthalmoscopic examination. These, as a general thing, are the more extensive the higher the degree of the myopia. They affect above all the chorioid and retina, which become atrophic, both in the vicinity of the papilla and in the region of the macula lutea (see § 745). The latter spot, moreover, is the favorite seat of retinal hæmorrhages. Numerous floating opacities are formed in the vitreous, the latter at the same time becoming liquefied. The degeneration of the vitreous may cause detachment of the retina and opacities of the lens; also, through atrophy of the zonula, may cause tremulousness and even luxation of the lens.

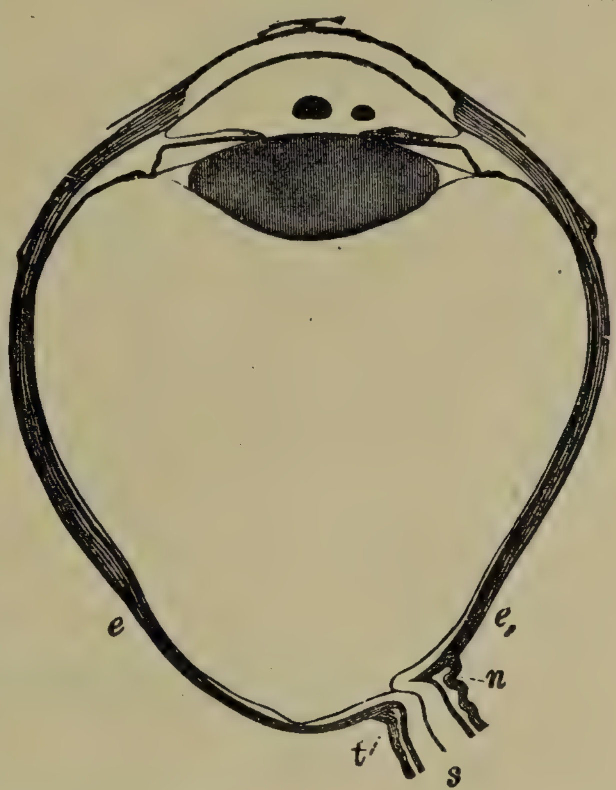

FIG. 97.—CROSS SECTION OF A MYOPIC EYE HAVING AN AXIAL LENGTH OF 28 MM. Magnified 2x1.

The ectasia (staphyloma posticum) which occupies the posterior segment of the eye extends from e to e_1. Over the area occupied by the staphyloma the sclera is very much thinned, and at the spot where it is continuous with the sheath of the optic nerve its layers have separated from each other, so that the intervaginal space is dilated at its extremity, and more so at the nasal side, n, than at the temporal side, t. The optic nerve, s, shows within the foramen scleræ an outward bend. The anterior chamber is very deep; the ciliary body projects but slightly.

In the lower degrees of myopia no noticeable change in the size and shape of the eyeball is found on dissection. But a comparative test of the refraction in different parts of the fundus shows even in these cases an approximation to the oval form (Salzmann). In myopia of higher degree the eyeball is considerably elongated. This elongation is due to a staphyloma posticum, a condition which was first discovered by Scarpa in 1807 but was not recognized as the cause of myopia until 1854 (by Arlt). When the myopia is not excessive, the ectasia is limited to the region of the posterior pole and the eyeball assumes thus an oval shape (Fig. 97). In extreme degrees of myopia the ectasia extends to the equator of the eye, and the eyeball back of the equator becomes almost cylindrical. The sagittal diameter of the eye, which in emmetropic eyes amounts to 26 mm. at most, may then increase to 32 mm. or over, and the transverse diameter is also somewhat enlarged. When the tension of the eye is lessened, as after death or in certain hardening fluids, the staphyloma collapses and becomes quite concave. The ectatic portion of the sclera is often only as thin as paper, and the chorioid and retina lining it are atrophic.

In the anterior segment of the highly myopic eye the only morbid changes found are in the ciliary muscle (Iwanoff). This is thinner than in the emmetropic or the hyperopic eye (see Figs. 98, 99, 100), since the circular fibres are less developed, and are sometimes, indeed, almost entirely wanting. Since the ciliary processes, too, are less prominent, the whole ciliary body looks abnormally flat and the root of the iris is further back than usual. Hence, the sinus of the anterior chamber is more patent and the chamber itself is deeper than in non-myopic eyes. According to some, this non-development of the ciliary muscle in myopia is due to non-use arising from the lack of accommodative effort in myopes; according to others it is a congenital defect, and Wood, accepting this view, argues that it is the cause and not the result of the myopia (§ 200).

Fig. 98.—Ciliary Body of an Emmetropic Eye.

Fig. 99.—Ciliary Body of a Myopic Eye.

Fig. 100.—Ciliary Body of a Hypermetropic Eye.

IV. Astigmatism

204. Regular and Irregular Astigmatism.—Owing to the complicated course that the refracted rays take in the eye and their consequent failure to focus sharply, all eyes have some astigmatism. The character of the images thereby produced, even in what is known as regular astigmatism, is quite irregular, so that the distinction between regular and irregular astigmatism is rather one of degree than of kind. Thus, some of the effects formerly attributed to irregular astigmatism, as, for example, the stellate figure that we see when looking at a distant point of light like one of the fixed stars,

are really the result of refraction by a regularly curved (sphero-cylindrical) surface (Gullstrand). Nevertheless, the distinction clinically is valid, since the images formed in ordinary astigmatism even of high degree not only have a general regularity of shape, which so predominates over their attendant irregularities that the latter are largely negligible, but also can be so well focused by correcting glasses as to appear quite distinct. On the other hand, in what is clinically known as irregular astigmatism the images are so distorted that no correction of them by glasses is possible. Such astigmatism, moreover, is usually of distinctly pathological origin, while in the great majority of cases regular astigmatism occurs in normal eyes and must be regarded as physiological.

205. Course.—Ordinary astigmatism is either congenital or is acquired very early in life as the result of developmental changes. It is present in most eyes and may have associated with it, especially if of high degree, a certain amount of irregular congenital astigmatism. It does not change much, as a rule, during childhood, youth, and middle life. Before the age of 50 astigmatism is oftener direct (or with the rule) than inverse (against the rule) or oblique. After 50 it not infrequently changes in amount and character in the sense that a direct astigmatism diminishes or is actually transformed into an oblique or more often into an inverse astigmatism, while an inverse astigmatism tends to increase. Such changes are usually slight (not over a dioptry at most). Higher degrees of astigmatism that develop at this time are generally pathological, being due to sclerosing or cataractous changes in the lens.

206. Corneal and Lenticular Astigmatism.—The seat of the irregularity which gives rise to astigmatism is found in both the cornea and the lens, but predominantly in the former. *Corneal* astigmatism is partly latitudinal, due to the aberration that each meridian of the cornea taken by itself displays, partly longitudinal, due to the fact that each successive meridian differs in curvature from its neighbor. In longitudinal corneal astigmatism the rule is that the more refracting of the two principal meridians of the cornea is nearly or quite vertical, so that a direct astigmatism is produced. This direct corneal astigmatism is generally more or less reduced by the action of the lens, which has a moderate degree of inverse astigmatism. The astigmatism of the *lens,* in fact, which is due to the peculiarities in the curvature of its exterior surface and more particularly in the curvature of the surfaces of its successive refractive layers (isoindicial surfaces) is in a measure such as to compensate for the astigmatism, both latitudinal and longitudinal, of the cornea (Gullstrand and others). The total astigmatism of the eye is therefore generally rather less than that of the cornea alone, and, moreover, often differs from it in the direction of its principal meridians, so that it would be corrected by a cylinder of different axis as well as of different strength

When an astigmatism of any noticeable amount is found in one eye, it is usually present in the other also, though not always to the same degree, and then the direction of the principal meridians in the two eyes in the large majority of cases is symmetrical.

Nearly all eyes have some astigmatism corrigible by glasses, although in very many cases this is very small (0.12 to 0.50 D). Corrigible astigmatism of 1 to 2 D is common, but astigmatism of 3 D is rather infrequent and one of 4 D quite unusual. It is rare indeed for regular astigmatism to exceed 6 D, although isolated cases have been reported of corrigible astigmatism of 20 D or more. Such extreme degrees of astigmatism, which are doubtless due in most cases to some considerable error of development, are apt to be associated with other developmental defects of the eye and with irregular astigmatism.

207. Pathological Astigmatism.—Some varieties of acquired or pathological astigmatism are more or less *regular* and hence more or less corrigible by glasses. This is the case when, as sometimes happens, the curvature of the cornea or the lens has been altered in quite a regular way by disease and especially by operation. Thus after every cataract operation, and in fact even after an iridectomy, a certain degree of corneal astigmatism develops, which indeed diminishes with the consolidation of the cicatrix, but seldom disappears entirely. Keratoconus, along with a high degree of irregular astigmatism, may cause an astigmatism of many dioptries corrigible by cylinders. The like is true of lenticonus and false lenticonus (§ 672). The lens also gives rise to regular astigmatism when it is obliquely placed as, for example, in case of subluxation, and also sometimes as a result of irregular action of the ciliary muscle, the astigmatism in that case being greater or less after atropine than before

Still more frequently pathological conditions cause *irregular* astigmatism. This occurs, for example, in the cornea from facetting after ulceration or from flattening or bulging of any kind due to inflammation or to degenerative processes (keratoconus, etc.). In the lens it results from cataract. In this, as the refractive power of the separate sectors of the lens becomes more and more different, the images produced by them recede farther and farther apart, so that ultimately they appear entirely distinct from each other. It is in this way that monocular polyopia develops in incipient cataract (see § 638). A very high degree of irregular astigmatism occurs in subluxation of the lens when the displacement is so considerable that part of the pupil still has the lens in it and part is aphakic.

208. Symptoms.—Regular astigmatism causes a peculiar distortion of vision due to the shape of the diffusion images (see § 170). With this are associated confusion of sight, asthenopia, and frequently reflex disturbances such as pain and irritation of the eyes, headache, and sometimes nausea and vertigo. In irregular astigmatism of any considerable degree objects appear irregularly distorted as though seen through a badly made window pane or often appear multiple (polyopia), and vision is correspondingly reduced.

The *vision* in regular astigmatism is distinguished from the vision in other errors of refraction by the fact that objects are distorted, and that not all their parts are seen with the same indistinctness. If the principal meridians are respectively vertical and horizontal, the horizontal strokes of the letter E will appear distinct, the vertical ones indistinct, or vice versa. The astigmatic subject then tries from the parts which he does see

to guess the rest. If we test the visual acuity of a myope at a distance of 6 metres, he will read Snellen's test types well down to a certain line, and then stops because he does not see any farther down. The astigmatic patient, on the contrary, often reads the whole card down to the bottom, but tells almost every letter wrong. In fact, he takes refuge in guessing—an attempt, however, which gives rise to a peculiar and very unpleasant form of asthenopia. Asthenopia and vertigo are further caused by the fact that the patient by accommodating can make first one set of lines distinct, then another, but cannot by any effort make both sets distinct at once. Thus if in the case represented in Fig. 73 his retina is at f_1 his horizontal meridian is emmetropic and he will see vertical lines distinctly, horizontal lines poorly. If now he endeavors to escape from the confusion thus caused by accommodating, he can make the rays passing through the vertical meridian, which originally focused at f, now focus at f_1, so that the vertical meridian is emmetropic and horizontal lines are seen distinctly. In this case, however, the rays passing through the horizontal meridian are also affected by the excessive accommodation so that they no longer focus at f_1 but focus in front of it. Hence the horizontal meridian is now out of focus and vertical lines therefore are no longer distinct. The patient, accordingly, has simply changed one bad condition for another. By focusing backward and forward in this way, making first one set of lines and then the other distinct, he gets confusion, asthenopia, and often vertigo. These unpleasant symptoms in some cases are accentuated by the fact that this alternate clearing and fading of lines make objects appear to move—wheel-like figures appearing to revolve and check patterns to dance.

As in other kinds of ametropia, objects like test letters may appear not only blurred but doubled (monocular diplopia).

209. Rôle of Astigmatism in Causing Myopia.—Since the astigmatic cannot see all the lines constituting a letter distinctly at once, he regularly tries to accommodate in such a way as to bring out most distinctly those linesby which he can judge the letters best (i. e., the vertical lines). In doing this he often makes himself temporarily myopic and if he keeps up the effort may become permanently so. To astigmatism, then, has been ascribed by many an important rôle in the development of myopia. In most of these cases, however, it is impossible to say that the myopia would not have developed anyhow as a result simply of the elongation naturally taking place during the growing period. Moreover, in many of these cases there are other conditions (opacities of the cornea, etc.) which are a more likely cause for the evolution of the myopia (§ 200).

V. Anisometropia.

210. Anisometropia.—In anisometropia one eye may be emmetropic and the other myopic, hyperopic, or astigmatic, or both eyes may be ametropic, but in a different way or to a very different amount. In this regard all possible combinations occur.

Anisometropia not infrequently is *congenital*, and then, at least in the higher degrees of it, often manifests itself even upon external inspection by an asymmetrical formation of the face and of the skull. *Acquired* anisometropia most infrequently originates from the circumstance that the change taking place in the refraction during life—that is, the decrease in the hyperopia or the development of a myopia—does not advance at the same pace in both eyes. The right eye usually has the higher refraction. Very high degrees of anisometropia develop when one eye is normal, but the other, in consequence of a cataract operation, has become very hyperopic.

A correction of anisometropia without the aid of glasses would be conceivable only as the result of an effort of the accommodation differing

in the two eyes; but this the eyes are incapable of doing, at least to any noteworthy extent. Accordingly, the anisometrope never sees distinctly with both eyes at once. This, however, gives him so little inconvenience that many persons do not become aware of the fact that they are not seeing equally well with both eyes until the tests of vision which the physician institutes are made. Moreover, if the difference in the refraction is not too great, binocular vision is not disturbed by it. Both images, even though they are of unequal distinctness, are superimposed and made to coalesce. In the high degrees of anisometropia, however, strabismus very frequently sets in. This may be either divergent or convergent, and is often alternating particularly when one eye is hyperopic, the other myopic (see § 282). Anisometropia, particularly when of high degree is rather frequently associated with hyperphoria.

CHAPTER XV

DETECTION AND TREATMENT OF REFRACTIVE ERRORS

I. General Principles

211. Scope and Routine of Examination.—In determining the refraction of the eye we must combine both subjective and objective tests and at the same time make a general examination of the patient, learning all we can about his symptoms, his physical condition, and his temperament, and determining his accommodation, motility, and the exterior and interior condition of the eye. From all these factors we shall decide as to the significance of the refractive findings and thus determine whether glasses should be prescribed, and, if so, what they should be, and also determine what other means besides glasses should be applied.

It is best to follow a definite routine in the examination. A good order is as follows:

1. Taking the history and determination of the glass the patient has been wearing (see § 158).
2. General inspection of patient. Examination of exterior of eye (§§ 386–393).
3. Determination of the vision of each eye separately and together (§§ 185–187).
4. Examination of refracting media and of fundus with ophthalmoscope (§§ 397 et seq.). (If case seems to be obviously one of refractive trouble, this examination may be deferred till after the next step.)
5. Subjective examination of refraction (preceded, if desired by a determination of the corneal astigmatism with the ophthalmometer). This combined with (*a*) examination of accommodation and determination of reading glasses (§ 215); (*b*) examination of motility (subjective and objective tests; §§ 302-310).
6. Subjective and objective examination under cycloplegia (§§ 217, 219-223).
7. Postcycloplegic tests (§ 218).

In the case of children too young to undergo the subjective examination we make an examination, objectively at least, of the motility of the eye, when we are examining the exterior; next, if possible determine at least approximately the vision; then instill atropine and combine an examination of the refracting media and the fundus with a determination of the refraction by skiascopy.

It is important in all cases to keep an accurate *record* of the findings, omitting nothing that may possibly be important for future reference. For the understanding of a case negative findings may be as important as positive and should therefore be set down in the record.

212. Manifest and Latent Errors.—In making both the subjective and the objective examinations we must always remember that under ordinary conditions a very large part of a patient's hyperopia or less often of his astigmatism may be concealed by the accommodation, i. e., may be latent, and that his myopia may possibly be exaggerated by the same cause (§ 178). Hence, as has already been noted, the mere fact that a person gets normal vision without glasses is simply a proof that he is not myopic but by no means a proof that he may not have a considerable degree of hyperopia and more or less astigmatism. Furthermore, he may obstinately hold on to the effort by which he is concealing his hyperopia and astigmatism, so that we have to use every artifice to get him to relax. In examining such a case subjectively, then, we add convex glasses of gradually increasing strength beginning with +0.25 D; aiming in this way to make him relax little by little. When we have by such gradual additions reached the highest convex glass with which he will still see clearly, or as we say the highest glass that he will *accept*, it is proof that he has at least that much hyperopia, which, latent before, has now become manifest by relaxation of the accommodation. He may, of course, have a good deal more which remains obstinately latent. This may be the case also in the objective tests with the ophthalmoscope or skiascopy provided the patient's accommodation is still active. We must then, in order to get the true finding, use a cycloplegic, which by doing away entirely with the accommodative effort renders all the error manifest.

So too in examining myopes, if we get normal vision with a certain concave glass we must ask ourselves if possibly this clear vision is not in part secured by accommodative effort which exaggerates the myopia. We then try to determine whether the patient will not see equally well with a convex addition, i. e., with a weaker concave glass.

The rule, in fact, obtains that when we are examining the refraction, either by the subjective or objective method, of a patient not under a cycloplegic, the *highest convex* glass or the *lowest concave* glass which gives normal vision or which makes the patient objectively emmetropic is a measure of the manifest refraction. When a patient is thoroughly under a cycloplegic just the reverse holds good.

II. Subjective Examination

213. Determination of Refraction with Glasses.—In determining the refraction with the trial case, it is important to proceed according to some systematic plan. A good way is as follows: Unless we know the patient is myopic, we begin with a convex spherical glass (one of 0.50 D if the vision is already good, one of 1 or 2 D, if the vision is poor). We change this rapidly until no further improvement in sight is secured or, if we are making the examination without a cycloplegic, until the added convex glass causes blurring. Then we begin adding to this strongest convex glass accepted cylinders, weak if the vision is good, stronger if the vision is poor, and at axes of 90°, 180°, 45°, and 135°, respectively. We try convex cylinders first, and if these do not help, concave cylinders. If any cylinder improves,

we leave it on in the axis indicated. If neither convex spheres nor cylinders improve at the outset we try concave cylinders or put in concave spheres, and when the maximum vision attainable has been secured with these, add cylinders to them. Thus, in one way or another, we get an approximate spherical or sphero-cylindrical correction. Or, we may get our first approximation with skiascopy or with the ophthalmometer. Suppose this approximate correction to be +2.50 sph. ○ +1.25 cyl. 90°. To this we add in turn—

+ 0.50 sph.	making	+ 3.00 sph. ○ + 1.25 cyl. 90°
+ 0.50 cyl. 90°	"	+ 2.50 sph. ○ + 1.75 cyl. 90°
+ 0.50 cyl. 180°	"	+ 3.00 sph. ○ + 0.75 cyl. 90°
− 0.50 cyl. 90°	"	+ 2.50 sph. ○ + 0.75 cyl. 90°
− 0.50 cyl. 180°	"	+ 2.00 sph. ○ + 1.75 cyl. 90°
− 0.50 sph.	"	+ 2.00 sph. ○ + 1.25 cyl. 90°

If any of these additions improves the sight we make the corresponding ubstitution and start with this as a new basis. Thus, if the addition of —0.50 cyl. 180° was an improvement (and the other additions were not), we substitute +2.00 sph. ○+1.75 cyl. 90° for our first trial combination, nd then to this new combination make systematic additions as before. Pretty soon we come to a combination which is not improved by any ddition (even of a 0.25 D sph. or cyl.). Then we try shifting the axis of he cylinder a little one way or the other. If this does not help, then we an be fairly sure that the combination last found is the best that can be ad. If we are making the examination under a cycloplegic, we confirm this esult by skiascopy (§§, 220-223), and if the latter affords any indication or it, modify our combination and try again.

If with the trial combination used at the outset we get comparatively oor sight (less than $\frac{20}{40}$) we would make additions of 0.75 or 1.00 D exchanging this for a 0.50 D, when the vision had been improved to $\frac{20}{40}$ or better. When the vision is about $\frac{20}{20}$ we can make additions of 0.25 D or even 0.12 D.

If no addition brings the vision above $\frac{20}{40}$ or $\frac{20}{50}$, we re-examine the eye arefully for pathological changes (faint opacities of the cornea or lens, lisease of the fundus) and if these are absent, examine the patient for a entral scotoma, especially a color scotoma.

Some instead of working up gradually to the highest convex glass a patient will ccept, put on at once a strong glass; with which he sees indistinctly, and then reduce his gradually till clear vision is attained. This *fogging* method is used particularly in esting for astigmatism (§ 214), but finds its special application in the postcycloplegic xamination (§ 218).

When we are examining a patient who is not under a cycloplegic, and ave found the highest convex or lowest concave glass that he will accept vith each eye separately (see § 212), we can sometimes get him to relax till more if he looks with both eyes together (May). In that case we lter the correction of each simultaneously, making successive additions of +0.25 D to both at once, until the vision begins to blur. Sometimes the wo eyes together will thus accept an addition of a dioptry or more above he limit of the unilateral manifest correction.

In any case when the examination of each eye by itself has been completed, whether with or without a cycloplegic, we should take the vision with both eyes, when corrected, in order to see whether, as should be the case, binocular sight is better than monocular (§ 187).

In making the subjective test it is important that the glasses in the trial frame should be at the same distance from the eyes as the glasses worn by the patient. If the glasses in the trial frame are at a wrong distance they exert an effect stronger or weaker than that indicated by their number.

214. Subjective Detection of Astigmatism.—As a general thing astigmatism is readily detected and accurately measured by the empirical test with cylinders described in § 213. But various supplementary subjective tests are used which may help in doubtful cases or may confirm a result otherwise obtained.

One test much used is the *astigmatic chart.* This usually consists of a series of lines or groups of parallel lines radiating from a common centre. There may be a fixed disc

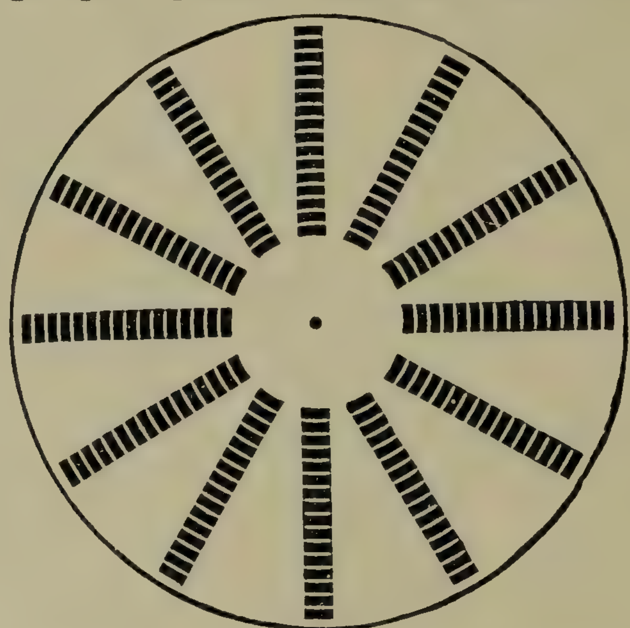

Fig. 101.—Test Object for determining the Position of the Principal Meridians in Regular Astigmatism. In most Charts used in America, Parallel Radiating Lines Replace the Cross Bars here Shown.

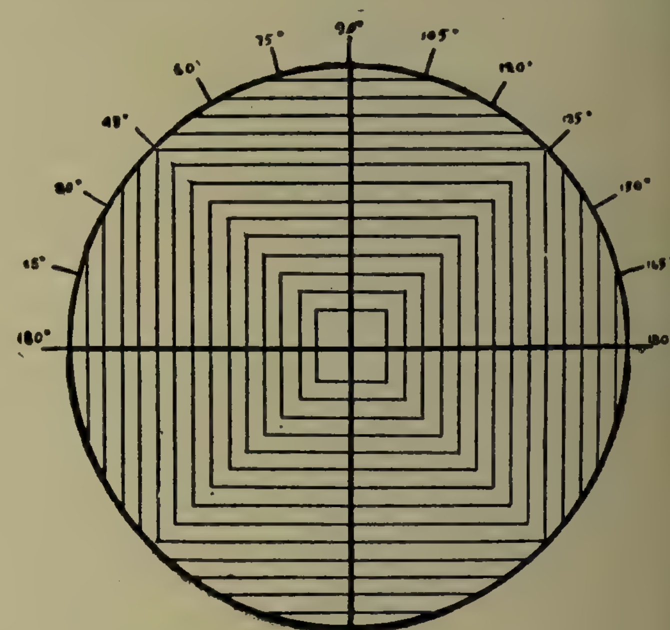

Fig. 102.—Verhoeff's Chart.

containing 4 or 6 sets of such lines making angles of 45° or 30° with each other (one of the latter kind is shown in Fig. 101); or there may be two sets of lines at right angles set on a disc which can be rotated so as to make the lines point in any desired way. To an astigmatic, particularly if he has been reduced by a spherical glass to a condition of simple astigmatism (§ 174) the set of lines that happens to lie in line with one of his principal meridians will appear distinct, while the line at right angles will appear blurred. The blurred lines in this case lie parallel to his emmetropic meridian (§ 170), and the correcting cylinder should therefore be placed in line with them. A better test chart still is that of Verhoeff (Fig. 102) which is used as follows:—The chart is rotated until all lines look equally blurred, when the two main lines should occupy a position 45° from the principal meridians. The disc is again rotated 45°. Then one set of lines comes out most distinctly, making the two corresponding segments stand out black and sharp, while the other two segments look gray. Cylinders are then applied with their axes parallel to the less distinct lines of the chart. The cylinder which, with its axis in this direction makes all the lines look alike is the one that corrects the astigmatism. Other devices, such as discs or letters (Pray's letters) composed of lines running in different directions are used for the same purpose.

In the translator's experience the chart is useful rather as a corroborative test, than as one to begin with. A great many patients fail to recognize the distinctions between

the two sets of lines, even when their accommodation is relaxed; and when it is not relaxed they frequently focus from one set of lines to the other so as to give confusing answers.

The *fogging method* (§ 213) is generally used in conjunction with the astigmatic chart. That is, an overcorrecting glass is put on with all the lines blurred; then this is gradually reduced in strength until one set of lines comes on sharply. The subsequent correction is then made with a cylinder.

The *stenopæic slit* is also used for determining astigmatism, and especially the axis of the correcting cylinder. Of the various methods of applying the test the best is as follows. When an approximate correction has been secured by spherical lenses, the slit is put in the frame and revolved until the clearest vision is secured. The spherical glass is then changed until the vision obtained through the slit is normal. Suppose this glass is a +2.00 and the slit is set at 70°. The +2.00 then represents the spherical part of the correcting sphero-cylinder and 70° is the axis of the cylinder. To determine the strength of the latter we remove the slit and leaving the +2.00 spherical in place add cylinders at axis 70° until full vision is secured.

Sometimes the detection of minute degrees of astigmatism is facilitated by making the subjective test under greatly *reduced illumination*. Ferree and Rand have shown that when the light thrown on the test card is very much reduced the patient affected with low degrees of ametropia will sometimes at least fail to discriminate letters which he can make out as soon as the proper correction is put on. This test seems to help particularly in determining the axis of weak cylinders, the patient detecting the letters when the cylinder is at the right axis and failing to do so when the cylinder is rotated.

215. Testing the Accommodation; Reading Tests.—The accommodation should be tested in every case of refraction. The test with the rule and card should be made for each eye separately and then for both eyes together with the glasses found. If on repeated tests the accommodation is found to be below the normal limit for the age (§ 182), there must either be a corresponding amount of latent refractive error or the accommodation must be actually subnormal. Such deficiency in accommodation may itself be the cause of the symptoms which had been ascribed to the refractive error and had not been relieved by its correction.

If with the full correcting glass for distance the accommodation as measured by the rule is less than 3 D, or at any rate if it is less than 2.5 D the patient will in general require an additional correction for near work. This addition must be determined by trial. What it should be depends on:

(*a*) The patient's *working distance*. Naturally we must adapt the glass to this, trying one strength, for example, if the patient desires a glass for ordinary reading, another if he wishes it for desk work, and still another if he is using it for card playing, the piano, etc. In this regard we endeavor to make the test under conditions, which, particularly as regards distance, shall simulate those under which the patient works.

(*b*) His range of accommodation and consequent *range of clear vision* with the glass. This should be such that all the near objects with which he ordinarily has to deal shall be within the limits of clear vision, i.e., within the far and near points artificially produced by his reading glasses. For reasons already given (§ 183) this region is often smaller than would be expected theoretically. The stronger the added glass, the more contracted it is. It is important that it should not be contracted too much, and hence, other things being equal, the weakest glass with which a patient can read should be given. In many cases he will prefer range to distinctness of

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reading vision, and in any event careful trial should be made to see which strength gives the best combination of range and distinctness.

(*c*) The *visual acuity*. Persons with sharp vision will be able to see fine print farther off than those with poorer sight, and hence will not require so strong a reading glass. Hence, if a patient can read only No. 3 or 4 Jäger we must give him a stronger reading correction than if he reads No. 1 readily.

(*d*) The *fineness of the work*. One who has to do with small objects (draftsmen, engravers, etc.) will need a stronger glass than those who use their eyes for ordinary reading. It is often well in these special cases to examine with a style of test-object that shall be like that with which the patient has to do.

(*e*) The *illumination*. A patient who works a good deal by artificial light often requires a stronger glass than one who works by good daylight. In some cases it may be necessary to try the patient's reading ability by artificial light.

(*f*) The condition of the *muscles*. Patients with a large exophoria for near and a remote convergence near-point will often be troubled by a glass which brings the print rather close, and will see a little less sharply but more comfortably with a weaker glass that does not call for so much approximation and therefore so much convergence as a stronger one. Sometimes a change of 0.25 D makes a marked difference in this regard. This, again, should be determined by trial.

It will be seen that the determination of the proper reading glass in presbyopia requires much care and patience. First the full correction for distance must be determined accurately. Then the patient wearing this correction is placed as nearly as can be under his normal working conditions and with his work at the habitual distance. Then different convex glasses are added to the distance correction until an addition is found that gives the most serviceable and comfortable (which is not always the sharpest) vision. With each addition it is well to determine the near point and corresponding amount of accommodation with the accommodation card. As stated above the correction will vary greatly with circumstances, but, as a rule, except in the younger presbyopes with a range of 2 or 3 D, a glass which brings the near point within 28 cm. (over 3.5 D) is not well borne, and, other things being equal, the glass that gives the widest and most serviceable range of vision is the best. This should be determined by a sufficiently lengthy trial. A glass, which seems comfortable at first may after a few minutes give a sense of pull or strain above or in the eyes. This is usually a sign that the glass is too strong. Sometimes the accommodation test shows a constant difference in the maximum accommodative power of the two eyes, and in that case it may prove best to make a somewhat stronger addition for reading in one eye than in the other (see § 383). Whether this is so or not must be determined by trial.

216. Tests of Motility.—It is essential in testing the refraction that the *motility* be examined at the same time. Sometimes symptoms, apparently due to refractive errors are really due to motor anomalies, and, moreover,

the presence of the latter may have some bearing on the question cf the glass to be prescribed (see §§ 215, 227,). In any case, the amount of deviation, if any for distance and for near, both with and without glasses and the situation of the convergence near-point should be determined. It is well also to note the size and mobility of the *pupils*.

217. Application of Cycloplegics.—In this country a cycloplegic is very frequently used in determining the refraction whether the case is one of hyperopia, myopia, or astigmatism. *Homatropine* is the agent mainly in use, although some prefer *scopolamine* (in ⅕-per-cent solution). Homatropine in 2-per-cent solution, instilled four or five times at intervals of ten minutes suffices in almost all cases to produce in an hour or an hour and a half a sufficiently complete relaxation of the accommodation. As the cycloplegia proceeds, a convex glass of 3 D or more should be added to the manifest correction and then the far and near points and the range determined with the accommodation rule and card. When the range becomes less than 1 D, the cycloplegia may be regarded as fairly complete. The effect begins to abate pretty soon, but does not disappear completely until two or three days at least have elapsed. Some measurable effect, indeed, is often apparent five or six days after the instillation. The period of blurred sight may be abridged by repeated instillations of ¼-per-cent eserine with 1-per-cent pilocarpine.

The *practice in regard to the use of a cycloplegic* varies, although oculists here may be said to employ one in from 40 to 90 per cent of their refraction cases. The translator's own practice is to use a cycloplegic whenever practicable, especially insisting upon its employment in children and where there is a suspicion of spasm of accommodation (difference between subjective and objective tests) or where there are evidences of convergence-excess (see § 330). Contrary to the statements generally expressed he has found it advantageous to paralyze the accommodation in patients between forty and fifty. It has a number of times been his experience to find patients of this age in whom the refraction could not have been determined accurately without a cycloplegic. He has found the latter particularly serviceable at the time between forty and forty-six when the accommodation is changing fast and when the patient, in struggling to use his failing accommodation, often over-exerts the latter for distance and conceals some part of his ametropia. Of course, in using homatropine for these elderly cases, we must exclude any suspicion of glaucoma, and be particularly careful to avoid using a cycloplegic when the pupil in a non-myopic patient is unnaturally dilated.

The gain in certainty both for the physician and the patient that we get by using a cycloplegic in both the objective and subjective tests is so great in comparison with the moderate inconvenience produced, that it seems proper to employ it in all cases except in the very old and in those who are likely to develop glaucoma

Occasionally homatropine fails to produce complete cycloplegia. This will be evident from the failure to obtain satisfactory or consistent results by subjective or even objective testing, from a continued discrepancy

between the results of the objective and subjective tests (particularly between the test with the trial case and the shadow test) and from the persistence of over 1 D residual accommodation. This *failure of homatropine* to produce complete paralysis occurs especially in cases of spasm of accommodation. Here, as well as in convergent strabismus, *atropine* (1, 1½, or 2 per cent) should be instilled two or three times a day for one, two, or many days. Even with this we may not be able to secure complete relaxation.

After we have found the glass which gives the best vision for 15 or 20 feet, it is well to see while the patient is still under a cycloplegic if this also gives the best vision for far distance (100 feet or more). We often find that for the latter the patient needs a −0.25 D added, and that the glass with this addition represents his real correction for distance.

218. Postcycloplegic Tests.—In hyperopia and presbyopia it is usually necessary to make a postcycloplegic test five to seven days after the homatropine. If made earlier the test often misleads, because the accommodation has not returned completely. To determine the glasses for distance by the postcycloplegic test we put on the full correction determined under the cycloplegic. Then, making the patient look with both eyes, we ascertain, first, whether after keeping the correction on awhile he will not accept it; if not, then what is the minimum reduction that he will take in order to see clearly. We determine the reading glass in the usual way, putting on the full correction found under the cycloplegic and adding to this the proper convex glasses.

III. Objective Determination

219. Determination with Ophthalmoscope; Direct Method.—As will be shown later, an observer using the ophthalmoscope by the direct method, gets a clear image of the patient's fundus, if the patient's eye and his own are either actually emmetropic or are made so, and if neither uses his accommodation. In that case the rays emanating from the illuminated area of the patient's fundus are parallel as they emerge from his eye (§ 172) and are, therefore, focused sharply by the observer's eye which being emmetropic is adjusted for parallel rays. To make both patient and observer emmetropic, if not already so, the ophthalmoscope contains a series of lenses, which can be brought in succession before the aperture in the mirror; or better still, both observer and patient wear their correcting glasses (§ 402). When the image appears distinct the observer knows that his eye is adjusted for the rays emanating from the patient's fundus. The glass put up in the ophthalmoscope, has then, rendered both observer and patient emmetropic—i. e., corrected the refractive errors of both simultaneously. Deducting from the strength of this glass the strength of his own correction the observer obtains at once the strength of the glass correcting the patient's ametropia. If the observer is wearing glasses that make him emmetropic, the glass put up in the ophthalmoscope indicates the patient's refraction directly.

There are very few who do not accommodate more or less when using the ophthalmoscope. In so doing they render themselves virtually myopic and have to neutralize

their accommodation with a concave glass. They have to do the same thing if the patient uses his accommodation. Even if the latter is abolished by a cycloplegic, the determination of the refraction by the direct method is pretty uncertain, and there are very few ophthalmologists who can measure the refraction in this way to within one dioptry.

If we examine by the direct method an eye with a notable amount of *astigmatism* the disc appears elongated into an oval shape (§ 405) and other details of the fundus are similarly drawn out and deformed. Furthermore vessels running in a certain direction, e.g., the vertical vessels, will come out distinctly with one glass while the vessels at right angles will be seen best with another—the difference in strength between the two glasses being approximately equivalent to the amount of the astigmatism. In this case we must recollect that the glass with which we see one set of vessels gives the refraction of the meridian at right angles to them (§170). Thus, if we see the vertical vessels with a +3 D, and the horizontal vessels with a plane glass, the vertical meridian is emmetropic and the horizontal meridian is hyperopic 3 D. If the patient during the examination wears the glass correcting his astigmatism, the apparent deformation of the disc is corrected and all details of the fundus come out distinctly.

The ophthalmoscope may give evidence of ametropia when used in other ways than by the direct method, e.g., when we see the details of the fundus clearly by *axial illumination* (§ 399) and when in the *indirect method* the disc appears to change shape or size as the object glass is approximated or withdrawn (§ 405).

220. Skiascopy (Shadow Test).—The determination of the refraction by means of the *shadow test* was discovered by Cuignet, who called it keratoscopy. It is also known as retinoscopy and skiascopy (*σκιά*, shadow). It is the most accurate of our objective methods of determining the refraction—far more so than the determination by the direct method—and has the advantage over the latter that the refraction and accommodation of the observer do not need to be considered. It is done in the dark room and usually requires dilatation of the pupil and the relaxation of the accommodation with a cycloplegic. In using it, the observer places himself at a determinate distance from the patient, and throws light into his pupil by means of a mirror. When the mirror is in a certain position, the whole pupil appears a vivid red; then if the mirror is turned a little on its vertical axis, a black shadow appears at the edge of the pupil and, as the mirror is rotated still more, passes over the whole area of the pupil, until the latter is completely dark.

221. Skiascopy with Concave Mirror.—If a concave mirror is used on applying skiascopy, the shadow in the patient's pupil moves in the same sense as that in which the mirror rotates, provided the far point of the patient's eye lies between his eye and the observer's, and it moves in a direction opposite to that in which the mirror is rotated, provided the far point of the patient's eye lies behind the observer's eye (in low degrees of myopia), at infinite distance (emmetropia) or behind the patient's eye (in hyperopia).

Without going into the details of the refractive principles involved, we may say that this is essentially what happens. Rays emanating from a source of light behind the patient fall upon the mirror and are concentrated by the latter at its focus (1) just in front of it. Rays diverging from 1 impinge on the patient's eye and are by the latter concentrated more or less sharply on the retina at a spot (2) the location of which is determined by a line drawn from 1 through the nodal point of the eye to the retina. The spot 2, being thus illuminated, sends out diverging rays which are refracted by the eye so as to meet in its far point, i.e., so as to emerge from the eye parallel if the eye is emmetropic,

or converge to some point in front of the eye if the latter is myopic, or diverge from a point back of the eye if it is hyperopic. If now we tilt the mirror down, its focus, which we may regard as the actual source of light, moves down too. The illuminated area of the retina (2) therefore moves up, and the image of 2 which the eye projects to its own far point moves also. Whether to an observer looking through the peep-hole of the mirror it appears to move up or down depends on the refraction of the eye. It is, in fact, the movement of the illuminated retinal area as projected by the eye and referred to the plane of the pupil that we see, although we generally speak of seeing the movement of the shadow which forms its edge.

In applying the shadow test we usually stand just beyond a metre from the patient. If the shadow then moves against the mirror, we know that the patient is either hyperopic, emmetropic, or myopic with a far point beyond 1 metre. We can, however, make him myopic with a far point of less than 1 metre by adding sufficiently strong convex glasses. When we do this the shadow will move with the mirror. It is obvious that the glass that *just* effects this change (or reversal) will have made him myopic exactly 1 D, since it will have brought his far point from a little back of a metre to a metre. To find his true refraction, then, we have simply to subtract 1 D from the convex glass added.

If the shadow moves with the mirror we add concave glasses until it begins to move against. The last glass with which the shadow still moved with the mirror evidently made the patient myopic just one 1 D, so that to find the true correction we have to add 1 D to that glass.

We have, thus, the following simple rule for using skiascopy with the concave mirror. We apply glasses, convex if the movement of the shadow is "against", and concave if the movement of the shadow is "with" the mirror, and we increase these successively in strength until we find the lens that just suffices to produce a movement "with" the mirror (any lens that is more concave or less convex giving us a movement "against"). This lens we call the *reversing glass*. If we then add −1 D to the reversing glass we get the glass that corrects the patient's refraction.

The observer may stand at *any distance* from the patient other than a metre. For example, he may stand at two metres. In that case, since the reversing glass obviously puts the patient's far point just in front of the observer's eye, the patient with the reversing glass on will be myopic 0.5 D and to find the true correcting glass we shall have to add −0.5 D instead of −1 D to the reversing glass. So if we stand at half a metre we shall have to add −2 D to the reversing glass to get the proper correction.

If the pupil is dilated we must try to observe the movement of the shadow in the area which corresponds to that occupied by the pupil when normally contracted. This *visual area*, or central portion of the pupil, which the patient habitually uses for seeing, sometimes differs markedly in refraction from the peripheral area of the pupil. Unless attention is paid to this point and the movement of the shadow in the visual area is taken instead of that in the periphery, a false estimate of the refraction may be had.

In skiascopy the best results are obtained if we use a *slit* or *hole lamp* for the source of light (Gullstrand).

222. Detection of Astigmatism by Skiascopy.—We determine *astigmatism* with the shadow test by ascertaining the refraction of the two principal meridians separately. For example, if with the concave mirror at one

metre we obtain reversal in the vertical meridian with +2.5 D and in the horizontal meridian with +4.5 D there is an astigmatism of 2 D and the correcting glass is +1.50 D sph. ⊃+2.00 cyl. ax. 90°.

A more accurate way of determining the astigmatism by the shadow test is to ascertain the refraction of one of the principal meridians with a spherical glass; then leaving this glass in situ, to place a cylindrical glass with its axis in the meridian thus corrected and keep on changing the cylinder until with the two glasses, spherical and cylindrical, a complete and uniform reversal is obtained in all meridians at once. This method has the advantage of indicating both the axis and the amount of astigmatism with great precision.

If the spherical glass is too strong, reversal will take place beyond or within a metre. We must then change the spherical glass until reversal takes place at just a metre. If the cylinder is of wrong strength, no added spherical glass will produce reversal in both the axis of astigmatism and in the meridian at right angles to it at the same time. We must then change the cylindrical glass until reversal is the same for the two meridians. Finally if the axis of the cylinder is wrong, the shadow will make a skew movement, i. e., a movement oblique to the direction in which the mirror is tilted. We must then turn the cylinder until the skew movement disappears.

Skiascopy is often used to *confirm the result* of the subjective test (see § 213). To do this we add + 1 D to the correction found, and then check the spherical and cylindrical elements in the combination in the way just stated.

Regular astigmatism of any amount is shown in the shadow test by the presence of a *band of light* which always runs in the direction of one of the two principal meridians. This appears as follows: When a glass is put on which is about equal to the reversing glass for either one of the principal meridians, the illuminated area of the pupil instead of appearing round is transformed into an elongated oval or into a band of light which runs in the direction of the meridian thus corrected. Then, no matter how the mirror is moved, the movements of the shadow tend to take place at right angles to this band. This band of light is an important indication since it runs in the precise direction in which the axis of the correcting cylinder is to be placed.

The presence of *irregular astigmatism* is shown in the shadow test by confused shadows which move irregularly and in various directions across the pupil; often by the presence of two shadows which come simultaneously from opposite sides of the pupil to meet each other (scissors movement).

223. Skiascopy with the Plane Mirror.—The shadow test can also be conducted with the plane mirror. With this the image of the flame (source of light) lies behind the mirror, and hence, when the latter is rotated, moves, not with the mirror, as is the case when this is concave, but in the opposite direction. Accordingly, the movement of the pupillary shadow with relation to the rotation of the mirror is just the reverse of that which obtains with the concave mirror. That is, in hyperopia, emmetropia, or low myopia the shadow moves with, and in myopia of 1 D or over moves against the mirror. The reversing glass in this case is the first convex glass or the last concave glass with which the shadow moves against the mirror. Otherwise the same rules apply as with the concave mirror.

224. The Ophthalmometer; Principle and Construction.— The ophthalmometer is an instrument for determining the corneal astigmatism by measuring the radii of curvature of the two principal meridians of the cornea i. e., the meridians which have respectively the greatest and the least refrac-

tive power. It does this by measuring the images of an object reflected on the cornea, first in one meridian then in the other. From the size of these images the curvature of the cornea in each of these meridians can be deduced; for the greater the curvature of a convex mirror, such as the cornea represents, the smaller are the reflected images.

The reflected images which serve for making the measurement should be quite large, because then the changes in their size which take place when the radius of the cornea alters have also large absolute values and hence are more readily demonstrated by measurement. For producing the reflected image, therefore, an object is selected whose image on the cornea is so large as to be equivalent to at least a fourth of a meridian of the cornea. If we should take for such an object an upright white cross, the reflected images of the two arms of the cross would be equally long only in case the cornea had a precisely spherical curvature. If, as is generally the case, the vertical meridian is more c rved than the horizontal, the vertical arm of the cross would look shorter in the reflected image than the horizontal; and from the difference between the two we might compute the difference between the radii of curvature of the two principal meridians. It is an easier matter to take, instead of the whole cross, only one arm of it, which is first placed horizontally, then vertically, and is measured in both instances. We, accordingly, give the object a considerable elongation, so that its reflected image shall extend mainly over only one meridian of the cornea; e. g., if the object is horizontal its reflected image will belong to the horizontal meridian (Fig. 104). In order to be able to vary the size of the object and therefore of the image we use for the former two white plates α and β which are attached to the circular arc B, and are movable on it (Fig. 103). By carrying them nearer together or further apart, the object as a whole is diminished or enlarged and so too is its reflected image on the cornea. If the size of the object is constant and its distance from the eye that is being tested is also constant, the size of the reflected image will vary only with the degree of the corneal curvature. Hence the latter can be computed if the size of the reflected image is known. *Helmholtz* was the first to measure this precisely, using the method of duplication of the reflected image. The apparatus designed to effect this is placed in the telescope which passes through the centre of the arc B, and shows the reflected image in a magnified form. In the ophthalmometer of *Javal and Schiötz*, which is now generally used, the doubling of the image is accomplished by a prism of double-refracting Iceland spar, and in the instrument made by Kagenaar by means of two glass prisms whose refracting angles meet in the centre. Hence, if we look through the telescope at the cornea we see the latter and also the images upon it double. The doubling is so contrived that the two images partly overlap (in Fig. 105 the image $a_1 b_1$ partially overlaps the image ab). If now by approximating the two plates α and β on the arc B we make the object smaller, its reflected image also becomes smaller; a advances toward b, and a_1, toward b_1. At the same time a_1 also comes closer and closer to b, until at last when the plate α has been brought sufficiently close to plate β, the inner edges of a, and b, which face each other come in contact (Fig. 105). We now read off on the arc the distance between the two plates, i. e., the size of the object; and since the distance of the latter from the cornea on which the reflection is cast and also the amount of the doubling are known, we can compute the size of the corneal image and from this the curvature of the cornea. For convenience, the arc B is so graduated that from the position of the plates the radius of the cornea in millimetres or the refractive power of the cornea in dioptries can be read off directly on it.

If now the arc is placed vertical its reflected image will lie in the vertical meridian of the cornea. If this has the same curvature as the horizontal meridian the reflected image will remain unaltered. If, however, there is a greater curvature in this meridian as is the case in a direct astigmatism, the reflected image becomes smaller. a and b come closer together, and so do a_1 and b_1, whence it follows that now a_1 slides over on b and partly overlaps it (Fig. 106). By enlarging the object, i. e., by separating the plates α and β from each other, we can now enlarge the double reflected image to such an extent that a_1 and b again come just in contact. If then we again read off on the arc the curvature of the cornea, we shall ascertain how much the latter has increased in comparison with

the horizontal meridian, i.e., we shall ascertain the amount of the astigmatism. But we can also deduce this directly from the amount of overlapping of a_1 and b. For this purpose plate a does not constitute a rectangle but a stair figure with six steps so measured off that an overlapping of one step corresponds to an increase of refractive power of one dioptry. Thus in the case represented in Figs. 105 and 106 the vertical meridian would be 3 D more refractive than the horizontal.

225. Application of the Ophthalmometer.—In using the ophthalmometer, we render the patient's head immobile by placing it in a chin rest

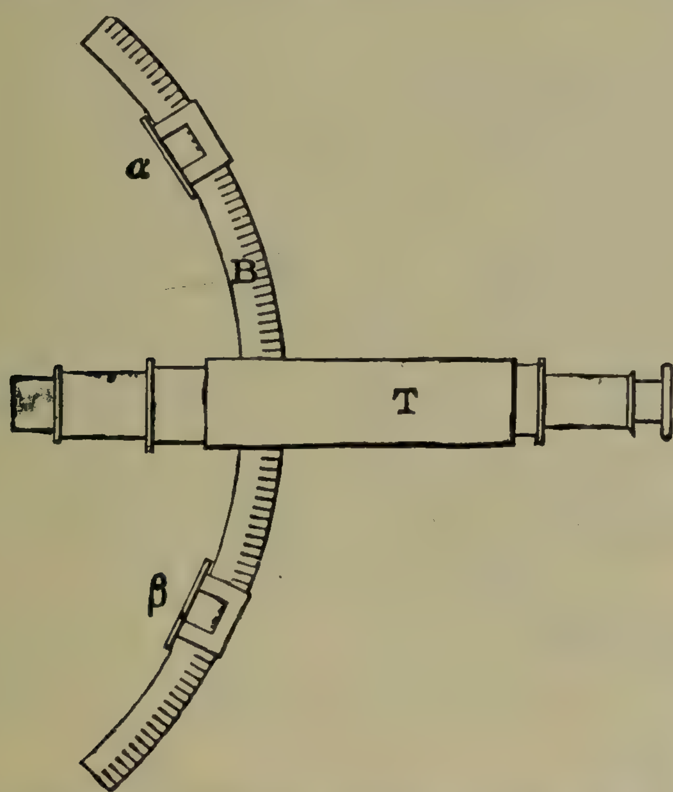
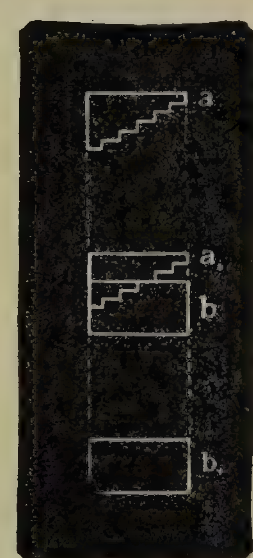


FIG. 103.—OPHTHALMOMETER OF JAVAL-SCHIÖTZ

FIG. 104

FIG. 105.

FIG. 106.



FIGS. 104 TO 106.—REFLECTED IMAGES ON THE CORNEA AS SEEN BY THE OPHTHALMOMETER.

and direct him to keep his gaze straight forward. We then look through the telescope at his cornea and rotate the arm B until the meridian is found in which the images of α and β are furthest apart. This is the meridian of least corneal curvature or first *principal meridian.* The direction of this is read off on a scale attached to the instrument. The plates (mires) having been moved until the images are in absolute contact, the arm is rotated until the images overlap the most, i. e., are closest together (meridian of greatest corneal curvature or second principal meridian). The difference in refraction between the two meridians is then measured in the way described, i. e., by counting the number of overlapping steps in the second position. This will give the number of dioptries of astigmatism, and the direction of one or the other of the principal meridians will show the axis of the correcting cylinder.

The ophthalmometer gives only the difference in refraction between the two principal meridians, but not their absolute refraction; it does not tell us whether either one of these meridians is hyperopic or myopic or to what extent they are so. Moreover, it shows only the astigmatism of the cornea, which usually differs from the total astigmatism in amount and sometimes in axis (see § 206). Hence, although it is often a useful auxiliary, it is not as reliable an objective test as skiascopy.

A very useful instrument for determining whether there is actually any great amount of corneal astigmatism present is *Placido's keratoscope* (see Fig. 107). This consists of a disc of cardboard, one surface of which bears a number of black concentric rings upon a white ground. An aperture in the centre of the disc and corresponding to the centre of the rings, allows the observer to look through the disc. The disc is held so that the side with the rings upon it is turned toward the eye that is beng examined, and so that the plane of the disc is parallel to the base of the cornea. If now we look through the central aperture at the eye we see the rings mirrored upon its cornea. If the cornea has the normal curvature, these rings appear perfectly circular; if not, the rings are transformed into ellipses, or show irregular bulgings, according as regular or irregular astigmatism is present. (See Figs. 108, 109.)

Fig. 107.—Placido's Disc.

IV. Treatment of Refractive Errors.

226. General Considerations.—In order to reach a decision as to the treatment of a refractive case, we must first have all the facts at our disposal. By a careful subjective and objective examination we must have gained precise and accurate information as to the patient's refraction, accommodation and motility, and as to the other facts already enumerated as being helpful (§ 211). Our final decision will depend on several considerations. In this regard, we must remember that we are treating patients, not eyes, and must, therefore, take into consideration all the physical and mental factors which may affect his outlook on life and determine his need for refractive or other correction. Among the factors to be regarded are—

(*a*) The *age* of the patient. The younger the patient, the more we may leave for his accommodation to correct. Moreover, in the case of young children we may sometimes omit glasses, when in an adult similarly affected we would prescribe them at once. For in childhood headache, asthenopia, etc., are often transitory and depend on the effect produced on

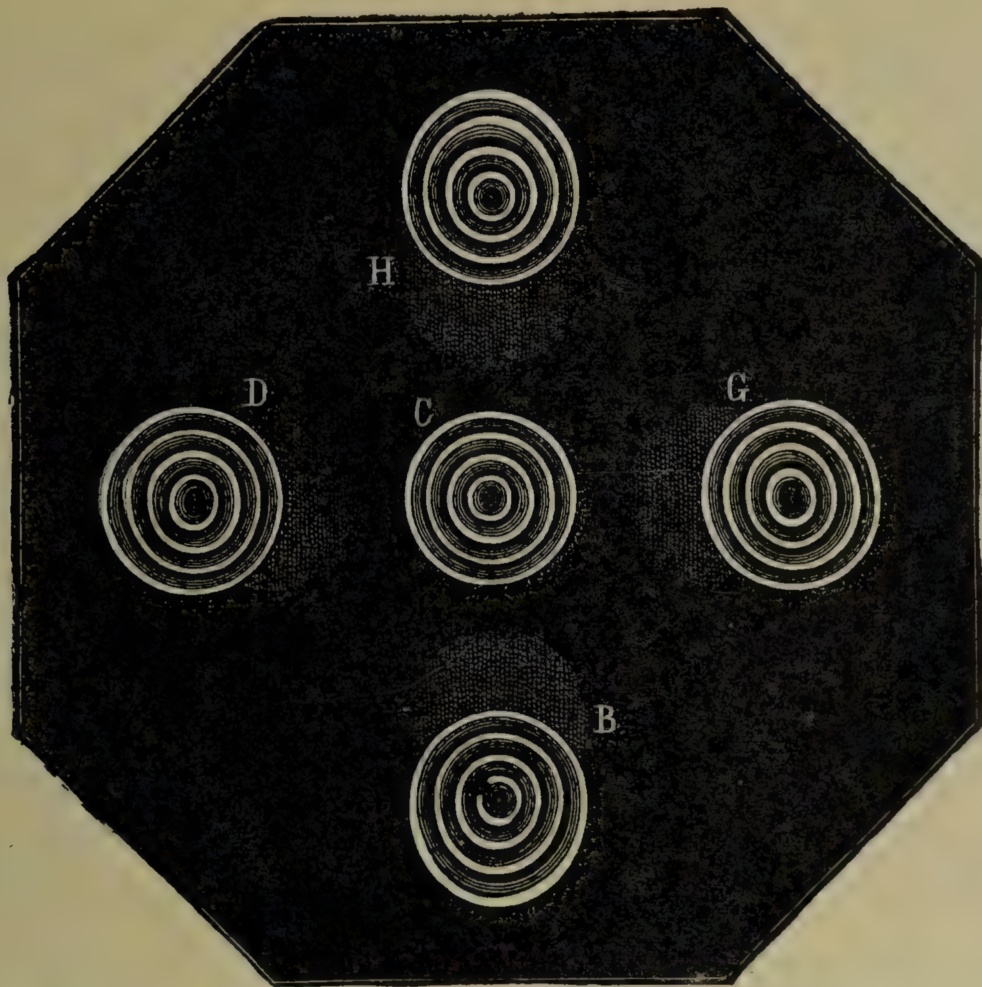


Fig. 108.—Keratoscopic Images in a case of Regular Inverse Astigmatism. After Javal (from Norris and Oliver).

C, reflex, from centre of cornea. The reflex is an ellipse with the vertical diameter the longer. *B*, *D*, *H*, *G*, reflexes from outlying portions of the cornea where the astigmatism is more marked and is also slightly irregular.

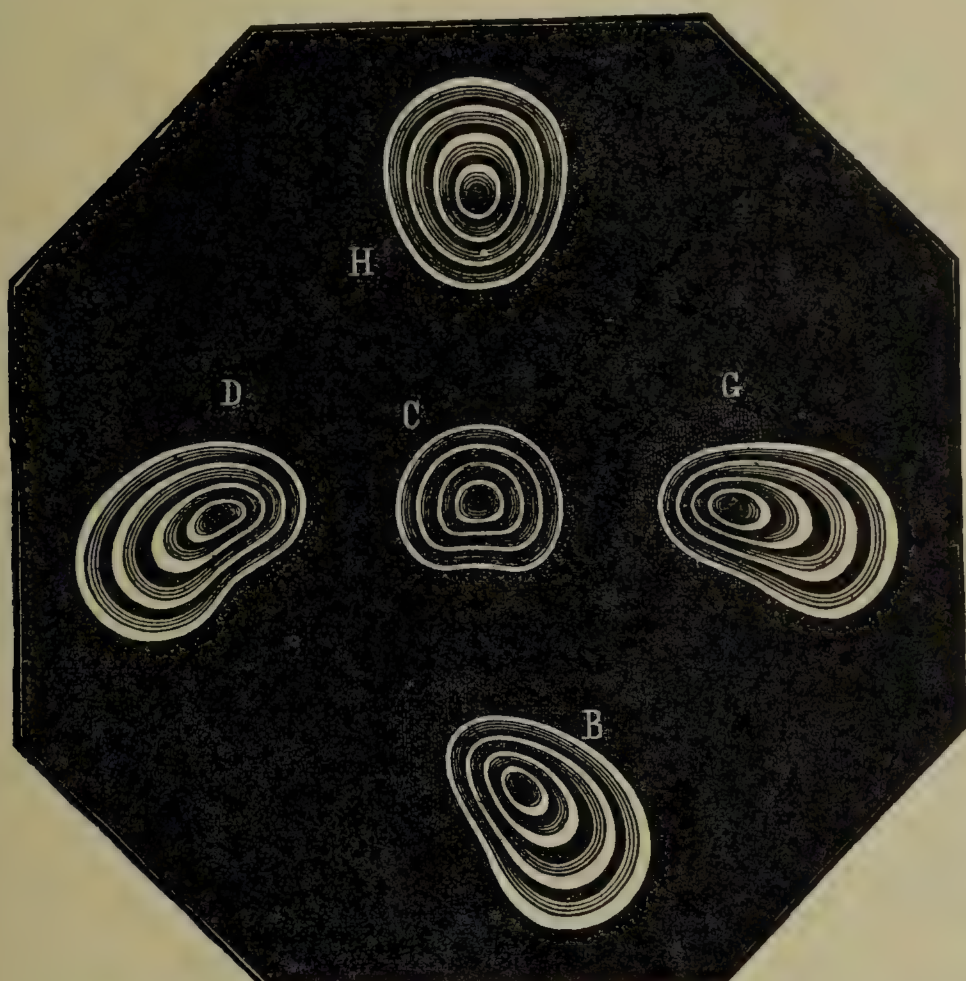


Fig. 109.—Keratoscopic Images in a Case of Irregular Astigmatism. After Javal (from Norris and Oliver).

the sharply reacting nervous system of the child by various influences, of which the refractive trouble may be only one. Unless the refractive error is considerable or there is some other good reason for giving glasses at once, one may refrain from prescribing them in children, when there is a hyperopia of 1 D or more or an astigmatism of 0.50 D−0.75 D—this, however, under the condition that the patient is watched to see whether the symptoms do or do not persist under this temporizing policy. On the other hand, even the slightest degree of myopia found in children should be corrected at once and fully (see § 227)

b) The *vision.* Patients who have sharp vision for distance and who can readily recognize slight distinctions in the strength and axis of added glasses naturally require a particularly accurate correction. With such patients we shall be especially concerned to get the glass right to a 0.12 D, and in these a weak glass often affords a relief which we should not expect and which in other persons, less discriminating, we fail to obtain. On the other hand it is undeniable that others—and particularly those with reduced vision—often get on satisfactorily with an approximate correction. This is fortunate since in just these cases an approximate result is often all that we can get.

Certain people demand sharp vision and suffer real annoyance unless they get it. In these cases we must be particularly careful not to give an over-correction of hyperopia or an under-correction of myopia for distance. Others, particularly myopes of medium degree (4 to 8 D), are not only satisfied with a somewhat blurred vision for distance but really prefer it, saying that the sharp definition is actually disagreeable. Again, many in office work are willing to forego sharp distant vision if they can see distinctly at a yard or two. In such it is often best to give a bifocal, in which the upper part represents the distance glass with a + 1.00 D added and the lower the regular reading correction.

Lastly, as already mentioned, patients with reduced vision for near will usually require a somewhat stronger reading glass than those with sharp sight.

(*c*) *Amount and Kind of Use to which Eye is Put.*—Obviously a person who is engaged in hard continuous eye work under trying conditions needs correction more than one who is not.

In this regard, we must remember, first, that some forms of distant vision, e. g., looking at objects in a theatre, museum, or stadium, using the eyes on a train or in a motor car, etc., are more trying even than near work; second, that some forms of near work are more trying than others. Many find card-playing more taxing to the eye than either reading or writing, and, in certain forms of close work (foreign language study, proofreading, reference work of all kinds, fine embroidery, fine drafting, etc.) the demands made on the eye are such that the most particular correction of even slight degrees of error, especially astigmatism, is required. Similarly a patient who uses the eyes much by artificial light will require a correction where he might not otherwise do so; or, if a presbyope, will require a stronger addition than he would by daylight. As Ferree and Rand have pointed out, those who work by low illuminations will require the most accurate correction of their refraction and especially of low degrees of astigmatism.

In considering what is and what is not trying eye work we must remember that to children of six or eight, reading of English means what reading of a foreign language does to an adult and they may require help from glasses for what would seem to us an easy task.

(*d*) *The Symptoms and their Relation to the Use of the Eyes.*—We give glasses not because a patient has this or that refractive error, but because

he has this or that symptom or because we feel assured that without glasses he is reasonably certain to develop trouble.

Again, no matter what the patient's symptoms are, we must, in prescribing glasses for their relief, first be fairly confident that the symptoms are either caused or aggravated by the eye condition.

In this regard we are likely to make two mistakes. First, we may consider as of ocular origin certain symptoms, such as eyeache and asthenopia, which though ocular in seat are really dependent on other causes, especially nasal obstruction or dental infection, and, on the other hand, we may fail to recognize the influence of refractive, accommodative, and muscular errors of the eyes in causing all sorts of symptoms, some of them not apparently ocular. Thus an asthenopia of nasal origin may be aggravated by the use of the eyes and require help from glasses as well as nasal treatment.

We must also bear in mind the considerable difference between individuals with regard to their reaction to strain of any kind. Some seem to be able to stand all kinds of stresses; others suffer intensely from slight abnormalities and their consequences. Slight blurring of vision, almost indefinable symptoms of discomfort, annoyances arising from maladjustment—difficulties which seem to us very trivial—may in sensitive individuals produce what is to them intolerable annoyance. These troubles are quite real. Patient attention to them and their correction and extreme care in the selection, adjustment, and verification of glasses are required and should be given.

(e) *The Accommodation* and (f) *Associated Muscular Condition of the Eyes.*—As already stated careful attention should be paid to these points in every examination of the refraction, and steps should be taken to correct any outstanding defects which are giving symptoms. In this regard it is often difficult in the presence of a muscular anomaly to tell whether this or the refractive error is the cause of the symptoms. In this case the refractive error should be corrected first, and after a reasonable interval given to ascertain what this alone will accomplish, measures should be taken to relieve if necessary the effects of the motor anomaly.

(g) *The Associated General Symptoms and General Condition of the Patient.*—The influence of the patient's general condition on his ability to do eye work is obvious. A patient depressed by overwork or in other ways may develop eye symptoms, the relief of which may require both treatment addressed directly to the eyes and treatment designed to remove the cause of the systemic trouble.

Patients who are mentally depressed or mentally backward and patients who are nervous or supersensitive require special care in examination and special attention in treatment. The relief of their eye condition may be an essential element in their improvement and we must give our best efforts to secure good results in this regard. At the same time in our zeal to correct the eyes we must not forget the individual and his idiosyncrasies nor the fact that the correction of his other physical and his nervous ailments claims equal attention with the ocular treatment.

227. Glass Prescribed.—The glass prescribed after the refraction has been determined under a cycloplegic will depend upon various factors. Each case must be judged by itself and in accordance with the effect we wish to produce. In general we may say:

1. We correct the total amount of *astigmatism* found under a cycloplegic. The only exception is when the astigmatism is very high (5 D, or

over) in which case a partial correction is occasionally less annoying to the patient than is a complete correction which gives only slightly better vision. With properly adjusted glasses, however, it will usually be found that even very strong cylinders will be worn with comfort and advantage. Slight degrees of astigmatism (0.25 to 0.50 D) may be left uncorrected if they do not appear to be giving trouble. They should be corrected (*a*) whenever a glass has to be used anyhow (as in presbyopia); (*b*) in most cases also when the patient has to use his eyes excessively for near work; (*c*) when there are well-marked symptoms of eye strain, such as headache, asthenopia, and obstinate blepharitis or conjunctival irritation.

2. We undercorrect the total *hyperopia* by an amount which depends upon—(*a*) The age of the patient. The younger he is the more we leave for his accommodation to do. (*b*) The amount of manifest hyperopia. The less this is in proportion to the total hyperopia, the more we usually have to undercorrect the latter in our prescription. In general, we give a glass somewhere between the manifest and the total hyperopia and as near the latter as the patient will accept on a postcycloplegic test (§ 218). (*c*) The patient's requirements. If he uses his eyes excessively for near work, we correct more of the hyperopia than if he is leading an out-of-door life. (*d*) The symptoms. In the presence of marked asthenopia, headache apparently due to eye strain, neurasthenia, and general muscular weakness (particularly the accomodative weakness after exhausting diseases), and especially in the presence of a tendency to excessive convergence, we correct more and more or even the whole of the hyperopia. When there is an actual tendency to convergent squint or when there is a spasm of accommodation, we correct the whole of the hyperopia and insist upon the continuous use of the glasses combined if need be with the use of atropine (see § 382). On the other hand, if well-marked exophoria is present, we may undercorrect the hyperopia somewhat, so as to stimulate the accomodation and with it the convergence. Generally, however, the more completely a hyperopia is corrected, the better. Many hyperopes, indeed, do not secure relief from their symptoms until a full correction is applied. As a rule, children accept a full correction very readily, and adults generally can be got to do so too, if they are willing to put up with some temporary blurring of sight. But with many patients, particularly those of middle age, the blurring produced by a full correction often causes great annoyance and even asthenopia; and much relief is experienced when the correction is reduced by even 0.25 D. It must be remembered that a convex glass may seem too strong and a concave glass too weak because too far from the eyes (see § 173). This is one reason why toric glasses and automobile goggles sometimes cause blurring and have to be reduced in strength.

As a general thing, unless he is presbyopic or his accommodation is otherwise subnormal, a hyperope, if he wears a glass at all, should wear one (and the same one) for distance and near.

3. We correct the total amount of *myopia* found under a cycloplegic. The main exception will be when the myopia is excessive and the patient has not worn very strong glasses hitherto, as in this case the sudden change

may cause discomfort. Moreover, if the patient is under the presbyopic age, we try, as far as can be, to make him use the same glass for distance and near. Statistics seem to prove unequivocally that the full and early correction of myopia tends to check the progress of the process. It is therefore becoming a more and more prevalent practice here and also in Europe to correct myopia fully, even when of slight degree, and, as far as possible, to get the patient to use the same glass for distance and near. In this way the eyes are placed under more normal conditions both for distant and near vision and also as regards their accommodative function. The result of this practice in individual cases has proved eminently satisfactory. It is especially important to make this full correction in children.

4. In the large majority of cases of *anisometropia* the patient will readily tolerate and find satisfaction in glasses fully correcting each eye, even when the difference in refraction is very great. There may be a period of temporary discomfort but even this is absent in many cases, and the final outcome is more satisfactory than when an incomplete correction is used. Moreover, by the use of this full correction combined with exercise of the poorer-sighted eye, the vision of the latter may be materially improved. In anisometropia it is particularly important that the glasses should be so centred as to produce the minimum of prismatic deviation. When the patient will not bear the correction applied to both eyes we correct the better eye and put a plane glass before the other.

In many cases the obstacle that opposes our attempts at correction of both eyes is a muscular error. This produces diplopia, which, as long as the image of one eye is indistinct, is not obtrusive and hence can be neglected, but which becomes annoying as soon as both images are made clear by the use of correcting glasses. In a few cases the trouble lies in the unequal prismatic effect of the unequally strong glasses. This causes diplopia when the patient looks through the periphery of the glasses and can generally be obviated by careful adjustment.

For the prescription of reading glasses, see § 183.

228. Adjustment of Glasses.—It is important for clear and comfortable seeing that the glasses should be properly adjusted. The visual line of each eye should pass through the optical centre of the glass, otherwise the latter acts as a prism (see § 152), which with the two eyes acting together may cause diplopia, confusion, or strain. For distant vision the distance between the optical centres of the glasses should be equal to the intercentral distance of the eyes. (For the method of determining this see § 387 and for the method of determining the optical centre of the glass, see § 158). In reading the visual lines are converged and directed downward. Hence in a reading glass the optical centres of the lenses should be approximated 4 to 5 mm. and dropped 3 to 4 mm., and the lenses themselves should be tilted forward about 10°, so that the visual lines shall strike them perpendicularly and in their centres. Even for distance glasses this tilting is advisable, since most of our seeing is done somewhat below the horizontal plane. For this reason too, particular care has to be taken in adjusting bifocal segments, so that the upper line shall not be too high and interfere with distinct vision when the eyes are directed somewhat down as in walking on the street. Adjustment of glasses along these lines is particularly important in anisometropia (see above).

229. Hygienic Treatment of Refractive Errors.—In patients with asthenopia or other symptoms due to refractive errors and particularly

patients whose vision is poor, we are often called upon to regulate the amount of eye work which they can do—determining this by careful consideration of the individual patient's symptoms and reactions.

In healthy eyes even an extreme asthenopia may be no bar to their use. In some subjects such an asthenopia is but enhanced by disuse, and graduated exercise of the eyes, with increasing use of them in spite of discomfort is the proper treatment.

Some patients with obstinate photophobia do well with Crookes' or similar glasses (§ **114**). Care should be taken not to overdo the use of such glasses, as the patient may easily get more and more dependent on them.

We must always remember that a patient's general physical and mental condition may have a good deal to do with his eye symptoms and that relief of an extra-ocular condition may enable him to use his eyes with the proper glasses or in some cases do away with glasses altogether.

230. Hygiene of Myopia.—In myopia the *regulation of the patient's habits*, both as regards his general condition and his eyes, must be attended to. This is the more important the greater the myopia is, and the more reason there is to apprehend its progressive advancement and the development of complications. And, first of all, near work must be restricted as much as possible. Such work as has to be done under any circumstances should be performed at the greatest possible distance from the eyes. To effect this we must see to it that the print of the books is good, that the illumination is sufficient, that the correct position is maintained in reading and writing, etc. Work in the evening by artificial light must be restricted as far as possible. It is very advantageous to interrupt the work at frequent intervals and rest the eyes by looking at a distance. If it is apparent that the near-sight is making rapid progress and threatens to reach a still higher degree, it is advisable to drop all studies for quite a long time. Young people with markedly progressive myopia should be warned to take the condition of their eyes into account in choosing a profession. An occupation, like that of clerk, accountant or one of the learned professions, that requires constant reading and writing, is not suitable for people of this sort.

The following means are advised to *put a stop to the extension* of myopia in schools. First, the excess of work which many scholars have at present to struggle with should be reduced to the proper standard. The way in which scholars are overtasked, both in school and at home, is prejudicial not only to the eyes, but also to the child's whole mental and physical development. Instruction ought not to be begun too early (if possible, not before the completion of the sixth year), and more time should be allotted to bodily exercise, especially in the open air, than has hitherto been the case. The hours set apart for this latter purpose should alternate suitably with the hours devoted to sedentary occupations, so as to serve as a rest from both mind work and eye work. That amount of work which absolutely has to be done should be done under the most favorable conditions. The requisites, which in many modern schools are already carried out, are: 1. Good illumination—i. e., illumination of sufficient strength and falling upon the work in the proper direction; the light should come mainly from the scholar's left side. The illumination should be diffuse—never very intense in any one spot—should present no glaring points of light, and should reach all parts of the room, so as to offer no violent contrasts of light and shade anywhere. By observing these precautions we lessen the tendency to eye strain as well as to the development of myopia. 2. Well-constructed seats and tables, which, furthermore, should be adapted to the varying size of the scholars, so that they may not be forced to adopt a bad attitude of the body. 3. A proper method of

instruction in writing which will enable the pupil to keep the head and body straight while writing (upright script). 4. Good print. Books having too fine print, and also too fine fancywork for girls, should be banished from schools.

A very important means of preventing school myopia and checking its advance is the systematic examination of children in the primary schools to determine and rectify refractive errors, especially astigmatism and myopia, and bring the sight as nearly as can be to the normal. Those children in whom the sight because of opacities in the media of fundus disease can not be brought to the normal should not be allowed to take the full course prescribed for others, and should be trained for some out-of door avocation. In a beginning near-sightedness, the hygienic rules laid down for myopes in general—restriction of the hours of study, restriction or absolute prohibition of study by artificial light, frequent intervals of rest for the eyes during the day, and insistence on out-of-door exercise—should be followed out with particular stringency. The younger the child, the more important these regulations are. Children with rather high myopia should be segregated so that they can receive instruction specially adapted to their needs and in classes by themselves (Harman).

In the highest degrees of near-sightedness, we may *remove the lens* by discission, even if it is still transparent (Fukala). The operation is a suitable one for young persons, whose myopia amounts to more than 15 D, and who do not show excessive pathological changes in the fundus. The eye may thus be brought nearly to the point of emmetropia (see § 193), so that it is able to see distinctly at a distance without any glass. We must not, however, overlook the fact that by this operation we sacrifice the accommodation and that the operation does not act to check the increasing elongation of the eyeball and the consequent changes in the fundus.

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PART IV.

SENSORY AND MOTOR FUNCTIONS AND DERANGEMENTS; EXAMINATION OF THE EYE.

SENSORY AND MOTOR FUNCTIONS AND DERANGEMENTS; EXAMINATION OF THE EYE

231. The sensory apparatus of the eye consists of the retina and optic nerve, which are simply an outlying part of the brain, and of the connections of the optic nerve in the brain. This afferent system is intimately connected in the brain with the corresponding efferent system, comprising the nerves to the exterior and interior muscles of the eye, the two systems thus forming a sensorio-motor reflex arc. The functions and derangements of these two systems—in other words, the neurology of the eye—will be considered in the following chapters.

CHAPTER XVI

SENSATION AND ITS DISTURBANCES; SUBJECTIVE EXAMINATION OF EYES

I. Physiology of the Retina and Optic Nerve

232. Function of the Retina.—The objects of the outer world throw their images upon the retina. The position of the image is always directly opposite to that of the object. Thus objects in the right half of the field (*G*, Fig. 110) throw their images on the left half (l, l_1,) of each retina; those situated above throw their images on the lower part of the retina, etc. It is the function of the retina to convert the rays of light, by which the images are formed, into nervous stimuli. What takes place accordingly, is a transformation of one sort of motion—the vibrations of the luminous ether—into another, namely, nervous excitation. The place in which the conversion of luminous vibrations into nerve excitation occurs is the rods and cones.

In what way this conversion takes place is not known, but we do know that a part of the vis viva, which the luminous vibrations represent, is used up in the production of chemical and physical changes. The chemical changes consist in the transformation of the visual purple, contained in the rods, into a colorless substance by the action of light (Kühne). It is very probable that besides the visual purple still other "visual substances"—i. e., substances which undergo chemical changes under the influence of light—exist in the retina. The physical changes partly consist in variations in the strength of the electric current that normally passes from the retina to the brain (Holmgren), partly are motile phenomena occurring both in the cells of the pigment epithelium and in the rods and cones. The rods and cones shorten under the influence of light; and in the pigment epithelium the pigment granules, which in the dark lie in the most posterior portion of the cells, push forward into the cilia-like processes which extend between the rods and cones.

The excitations set up in the retina give us our appreciation of form, color, and light intensity; and the fact that they are set up all through the retina and are thus conveyed to distinct portions of the occipital cortex enables us to secure peripheric as well as direct vision.

In order that the excitations in the retina shall accurately represent the objects that originate them, the retinal areas stimulated must correspond in size and shape to these objects, and the intensity of the impression must correspond to the intensity of the original impulse. This can be the case only if the refractive media (cornea, lens, vitreous), with the anterior portion of the retina itself, which lies in front of the percipient elements, have the proper optical character as to transparency, etc. (see §§ 69, 70) and if there is no notable uncorrected error of refraction (see Part III).

233. Transmission of Visual Impulses to Cortex.—The optic nerve conveys the nerve impulses generated in the retina, partly to the oculomotor nucleus, partly to the occipital cortex. Those going to the oculomotor nucleus initiate reflex motor impulses, which in particular effect the

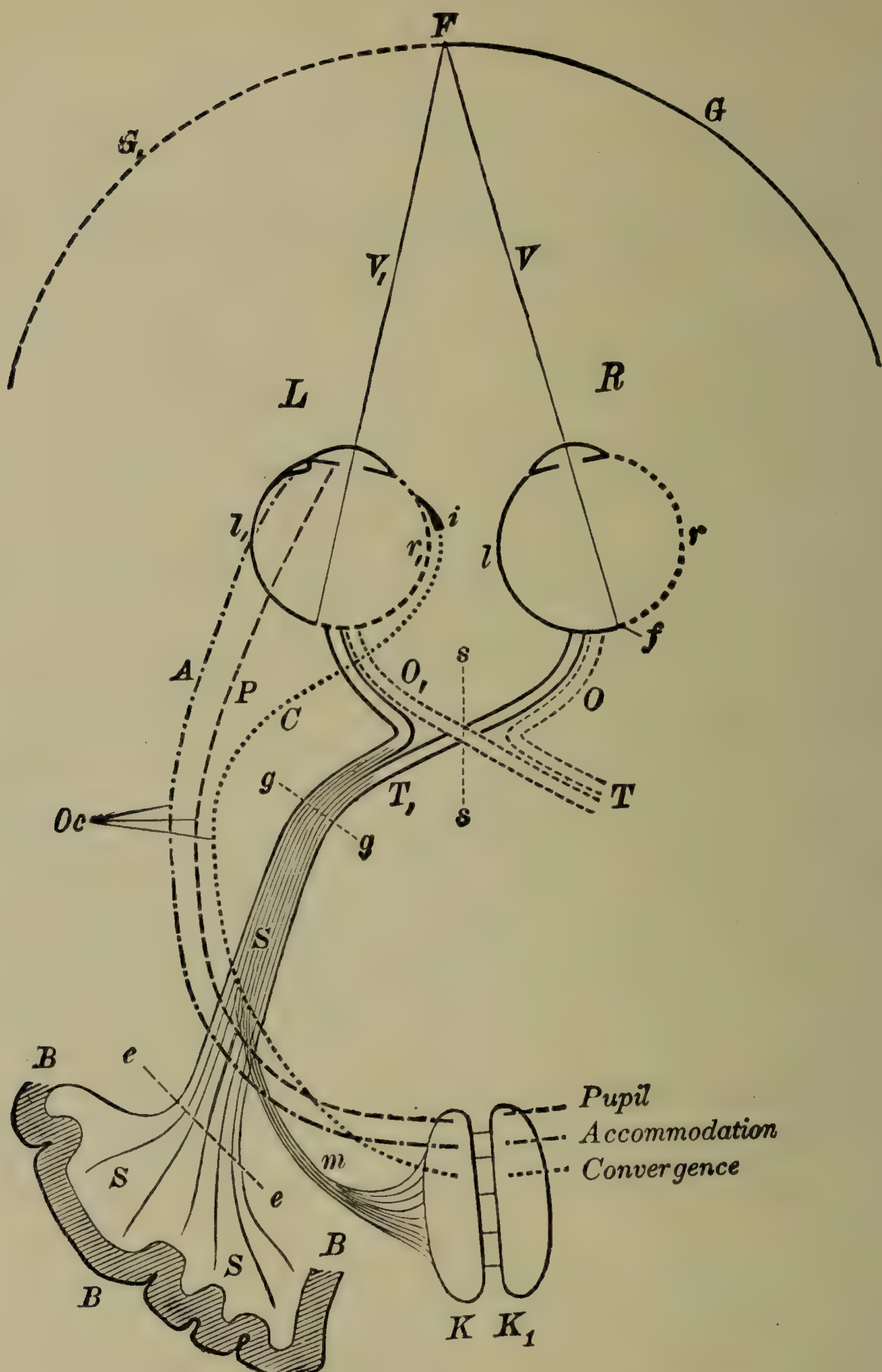

FIG. 110.—SCHEMATIC REPRESENTATION OF THE PATHWAYS.

The field of vision common to the two eyes is composed of a right half, G, and a left half, G_1. The former corresponds to the left halves, l and l_1, of the two retinæ, the latter to the right halves, r, and r_1. The dividing line between the two halves of the retina is formed by the vertical meridian. This passes through the fovea centralis, f, in which the visual line (V) drawn from the point of fixation, F, impinges upon the retina. The optic-nerve fibres arising from the right halves, r and r_1, of the two retinæ (indicated by the dotted line) all pass into the right optic tract, T, while the fibres belonging to the left halves, l and l_1, of the two retinæ pass into tract T_1. The visual fibres of each optic tract pass to the cortex of the occipital lobe, B, forming Gratiolet's optic radiation, S; the pupillary fibres, m, go to both oculo-motor nuclei, K and K_1. Each of these latter consists of a series of partial nuclei, one of which sends fibres, P, to the sphincter iridis, another sends fibres, A, to the ciliary muscle (muscle of accommodation), and a third sends fibres, C, to the converging muscle (internal rectus) i. All three fibres run in the trunk of the oculo-motor nerve, Oc (cf. Fig. 141). Division of the optic tract at gg or ee produces right hemianopsia, and in the former case there would theoretically be no reaction to light on illuminating the left half of either retina (hemikinesis). Division of the chiasm at ss produces temporal hemianopsia. Division of the fibres, m, abolishes the reaction of the pupil to light, but leaves the associated reaction of the pupil to accommodation and convergence unaffected (see for these relations Fig. 168).

direct and consensual reaction of the pupil, the act of accommodation and probably also the act of convergence. The fibres to the cortex convey visual impressions. Both sets of fibres pass together in the optic nerves, O

and O_1, (Fig. 110) to the chiasm, where both undergo a semidecussation. That is, fibres from the temporal (left) half, l_1, of the left retina continue on in the left optic tract, T_1, where they are joined by decussating fibres from the nasal (left) half of the right retina, l. The right optic tract similarly consists of direct fibres derived from the temporal (right) half, r, of the right retina, and decussating fibres from the nasal (right) half, r_1, of the left retina. The fibres of the optic tract terminate in neurones in the primary basal centres, some going to the pulvinar and anterior corpus quadrigeminum, but by far the most to the lateral geniculate body. From the basal ganglia the visual fibres ascend in the optic radiation, S, to ganglion cells in the occipital cortex, B. The sensory impulses arriving at these cells either initiate there a reflex motor impulse which is conveyed to the abducens, oculo-motor, and trochlear nuclei, or set up an excitation which in some way so corresponds to the object from which the light stimulus emanated, that the visual characters of the object (situation, extension, color, luminosity) are consciously apprehended. In ganglion cells which have once been subjected to excitation permanent changes remain (memories), which be-

FIG. 111.—THE PRIMARY VISUAL AREA
After Lenz, (from Salzmann).
I. Mesial surface of right hemisphere. II. The left occipital lobe seen on its lateral aspect. (The visual area—shaded portion—slightly overlaps the posterior occipital pole).

come so intense, particularly upon a pretty frequent repetition of the same excitation, that by means of them we are able to reproduce in our consciousness an object formerly seen (optical memory-pictures).

Wilbrand, Henschen, and others think that each portion of the retina and particularly the central portion (macula) has a definite, quite localized representation in the occipital cortex. To account for the fact that in complete hemianopsia macular vision is often preserved (see § 275), Wilbrand believes that the macula has a representation in both occipital lobes. This view that the calcarine cortex is a projection of the retina is rejected by Monakow and others, who assert that the macula finds its representation in the whole occipital cortex. But the experience of the late war seems essentially to confirm Wilbrand's contention and to show that the visual zone, or using a better term, the cortical centres for optic localization are limited to a strip lying within and along the lips of the posterior part of the calcarine fissure (see Fig. 111 I). In most this strip extends round the occipital pole over upon the lateral surface of the hemisphere (See Fig. 111 II) According to various authors the centres for peripheral vision lie round the front portion

and those for macular vision round the back portion of the calcarine fissure, and the upper part of the retina is represented in the area above, and the lower part of the retina in the area below the fissure.

It seems proved that the points in the visual cortex which represent any two corresponding points (see § 283) of the right and left retinæ are not identical but are in close inter-connection.

Association fibres connect these primary visual centres with higher centres in the cortex of both hemispheres, thus making it possible for us to utilize our visual perceptions for acts requiring the interposition of the intellect, e. g. in reading.

234. Cortical Representation of Right and Left-sided Vision.—As we have just seen, the excitation, from the left halves of both retinæ (l, l_1) are conveyed to the left cerebral cortex, B. But these excitations themselves are set up in the case of both eyes by light stimuli coming from the right half (G) of the field of vision, in which therefore the object apprehended by B is situated. Hence, the perception of all objects situated to the right of the median line is conveyed along the left optic tract to the cortex of the left hemisphere; the latter is thus designed for the apprehension of the right half of the external world. The converse is true of the right hemisphere. Thus the nerve subserving the sense of sight is in harmony with other nerves, all of which terminate in the hemisphere of the opposite side. This is the case both with centripetal and centrifugal nerves. What we touch with our left hand becomes an object of consciousness through excitation of the cortex of the right side of the cerebrum; and destruction of a certain portion of the latter entails a loss of the voluntary movements of the left arm. The sense of sight appears to form an exception to this rule, since each eye is directly connected with both hemispheres. This exception ceases to exist if we distribute the visual sensations in accordance with the halves of the field of vision to which they correspond. *Everything which the observer sees on the left side of him becomes an object of consciousness through excitation of the right occipital cortex, and vice versa.*

It is evident from the above statement that the right eye is not governed by the left brain in the sense that the right hand is. The visual apparatus corresponding to the left brain is a forked sensorio-motor organ, (Fig. 112), consisting of: (1) the right half of the cornea and lens in the two eyes forming the two ends of the sensory fork. These collectively are the true sensory organs and may be called the right lens-terminal. (2) The left half of the retina in both right and left eyes. This double organ, being an outlying portion of the left brain (see § 57), may be called the left brain-terminal. Between 1 and 2 the rays of light, a, a, emanating from an object on the right, cross over from right to left (exterior decussation a b). (3) The crossed bundle of the right and the uncrossed bundle of the left optic nerve. These passing over to (4) the left optic tract make the sensory decussation (semi-decussation), c. (5) Lower and (6) higher optic centres in left cortex. (7) Efferent fibres from 5 and 6 crossing over to right side (motor decussation, d) to (8) the right subcortical association centre. From 8 fibres pass to (9) and (10) motor nerve nuclei on the right side. From these the nerves (11) pass forward, going partly to the right side partly to the left (motor semidecussation, e, e) to form the two ends, (12) of the motor fork. These two ends comprise the motor nerve terminals and the muscles which they supply and which are attached to the right lens terminal (1). These muscles turn 1 in the direction of the arrow f, i.e. toward the object on the right. The right eye, then is not a visual representative of the left brain, but is composed of the fused representatives of both brains. A man who is left-brained and consequently right-handed is not "right-eyed." At most he is "right-seeing," i. e., sees and sights objects on the right rather better than on the left.

In the lower vertebrates and even in most birds and mammals there is a complete decussation of the optic nerves, but in many of the higher vertebrates there is a partial decussation as in man. This depends on the position of the eyes and the field of vision common to the two. In animals with complete decussation the eyes lie so far to the side that the fields of vision are altogether distinct, the right eye seeing only things that are on the right side of the animal, and vice versa. Hence all impressions affecting the right eye must be conducted to the left hemisphere, and the optic nerves must undergo

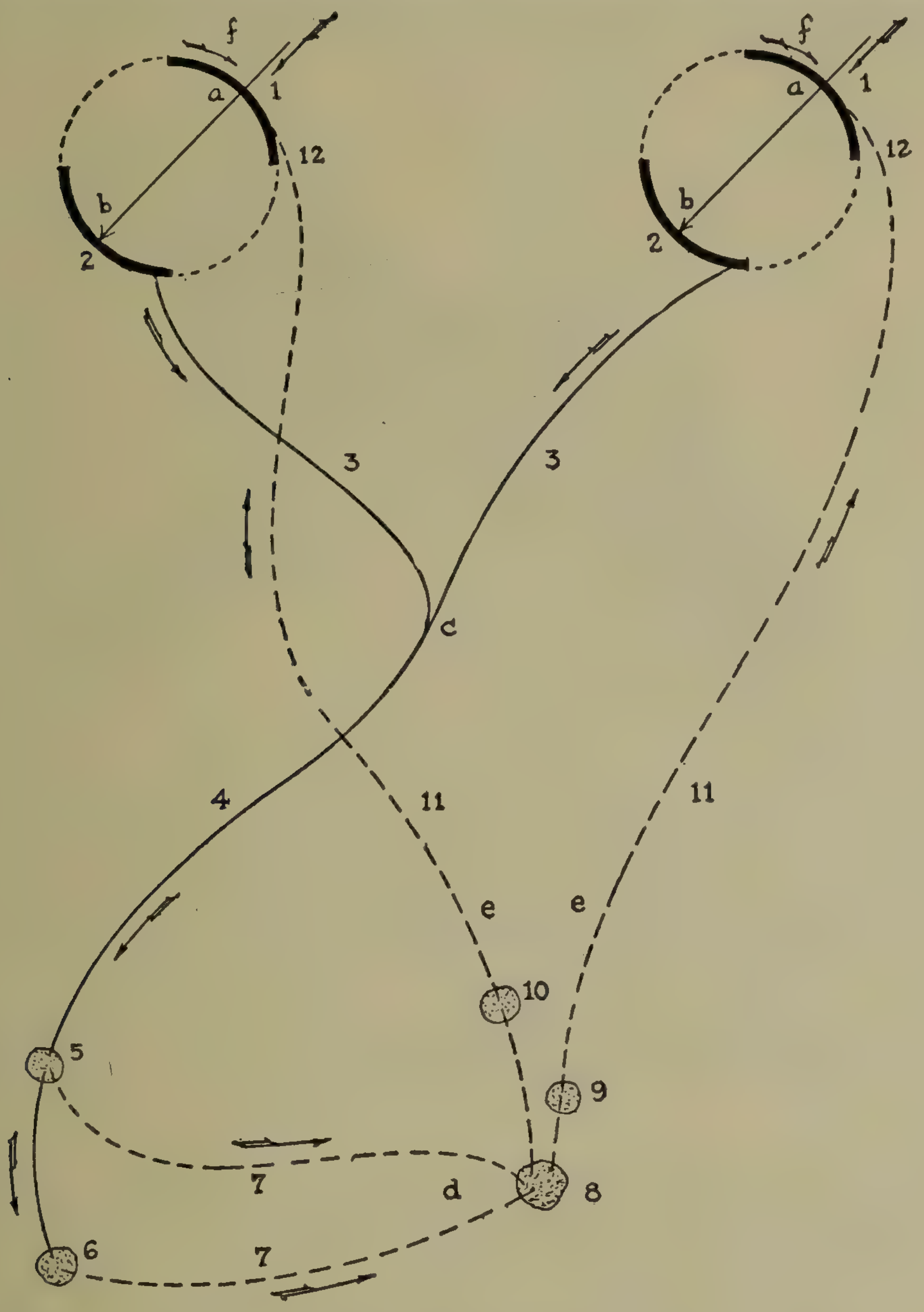

Fig. 112.—Diagrammatic Representation of the Sensorio-motor Organ Forming the Half of Each Eye.

The left organ is here represented. Both terminals, sensory and motor, are bifurcated, one arm of each fork merging in the right eye, the other in the left. The two organs, right and left, by their overlapping and fusion form the two eyes.

complete decussation. In proportion as the eyes are placed further forward, and the fields of vision therefore overlap more and more, the non-decussating bundle becomes more and more marked. In man the non-decussating bundle contains two-fifths, the decussating three-fifths of all the fibres of the nerve. The determination of this relation in man was made possible only by accident. People came to autopsy, who had lost an eye in childhood. One optic nerve was then found to be completely atrophic (Fig. 113), while the fibres of the other nerve, which were well preserved, were divided between both tracts but in such a way that the opposite tract always contained more fibres than the tract of the same side.

235. Monocular and Binocular Fields of Vision.—In man both eyes lie in the frontal plane, so that almost all objects are seen with both at the same time. Accordingly, the visual fields of the two eyes are to a large extent coincident, so that there is formed a large

binocular field of vision (the area left white in Fig. 114). Each eye sees objects both upon the right and upon the left side of the body, for which reason one part of the fibres of the optic nerve belonging to it go to the right, another part to the left hemisphere. To be sure, the visual field of each eye extends farther to the temporal than to the nasal side (see § 268). Hence, it follows that the nasal portion of the retina is larger than the temporal, and hence also, the number of decussating fibres in the optic nerve is necessarily somewhat larger even in man than the number of those which do not decussate.

The lateral portion of the visual field which subserves only *monocular* vision (the shaded portion in Fig. 114) is called by Behr the temporal crescent. The fibres arising

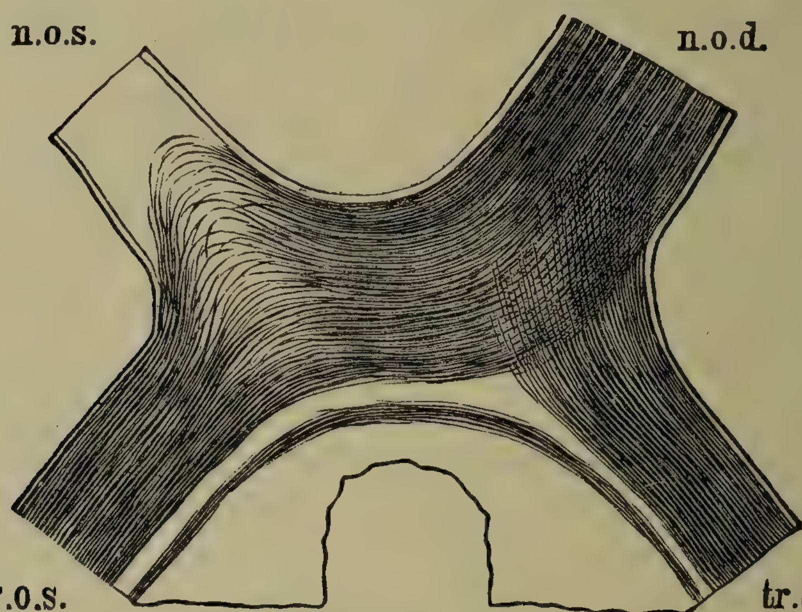

FIG. 113.—CHIASM IN ATROPHY OF THE LEFT OPTIC NERVE. Magnified 3x1.

The specimen is from a man, sixty-six years of age, whose left eye had been blind from childhood because of a total staphyloma of the cornea, due probably to blennorrhœa neonatorum. The cut represents a section passing through the chiasm in the horizontal plane, and stained with hæmatoxylin by Weigert's method, so that the normal (medullated) nerve fibres look black, while the atrophic fibres are unstained. The left optic nerve, *n. o. s.*, is completely atrophic, being both unstained and also considerably narrower than the right, *n. o. d.* The greater part of the fibres composing the latter pass transversely through the chiasm into the left tract, *tr. o. s.* On their way they make a looplike bend into the left optic nerve. The smaller portion of the fibres of the right optic nerve remain on the right side of the chiasm, and pass over into the right optic tract, *tr. o. d.* Since the non-decussating fibres are less numerous than the decussating, the right tract appears somewhat narrower in cross section than the left. The bundle of nerve-fibres running in a curve along the posterior border of the chiasm, and separated from it by a light-colored zone, is Gudden's commissure (or commissura inferior) which contains no optic-nerve fibres.

from the portion of the retina that corresponds to it run in a separate bundle from the chiasm to the cortex and terminate in a special area in the latter.

Animals that have their eyes placed on the side of the head, so that they have no binocular field of vision, may be said to have *lateral vision*, as distinguished from the *frontal vision* enjoyed by man. Some birds are able to move their eyes in such a way as to have either lateral or frontal vision, and some have two foveæ in each eye, one for each type of vision. An animal having lateral vision moves at right angles to his line of sight and hence gets a large parallactic displacement of objects that he passes—just as a man does when looking out of the windows of a railway train at objects whirling by. This large displacement gives the animal a much better idea of the relative distance of the objects seen than it could get by frontal vision, in which the parallactic displacement of the objects toward which it is moving is comparatively slight (Trowbridge). When the animal is at rest this advantage of lateral vision disappears, and even when it is in motion the advantage is partly compensated for by the fact that the animal with frontal vision sees the same object with both eyes at once and from this gets an idea of its spatial relations (see § 286).

236. Varieties of Visual Sensation; Central and Peripheral Vision.—Our visual sensations are of three different kinds, inasmuch as in looking at objects we take cognizance of their form, their color, and their bright-

ness. The faculty by which we recognize the form of objects is called the space or *form sense,* the faculty by which we distinguish colors constitutes the *color sense,* the faculty by which we distinguish different degrees of brightness constitutes the *light sense.* These faculties are resident in the retina throughout its entire extent, although in very different degrees. In this regard we must distinguish between central and peripheral vision.

Central or direct vision is vision with the fovea centralis. When we wish to see an object distinctly, we "fix" or "fixate" or "sight" it—that is, we turn the eye in such a way as to make the image of the object fall upon the fovea centralis, as the latter, on account of its peculiar anatomical structure, gives us the sharpest vision that we are capable of. *Peripheral* or indirect vision, i. e. vision with the other parts of the retina, is much less distinct.

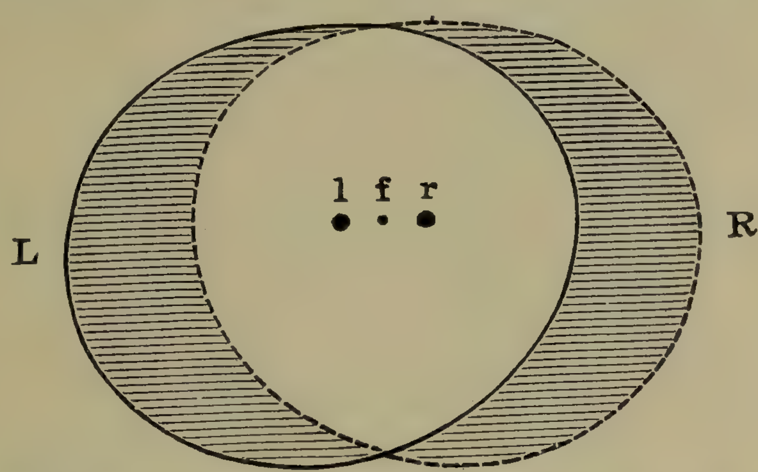

Fig. 114.—Binocular Field of Vision. (After Baas.)
The undotted line, *L*, bounds the visual field of the left eye, the dotted line, *R*, the visual field of the right. The median portion of the two visual fields overlap to an extent shown by the surface left white. This is, accordingly, the binocular field of vision, all objects in which are seen by both eyes at the same time. In its centre lies the point of fixation, *f* and at either side of the latter the blind spots, *r*, and *l*, of the right and left eye. Adjoining either side of the binocular field of vision are the temporal divisions of the two visual fields (the shaded areas in the figure) objects in which are seen with one eye alone.

We will first consider the different varieties of visual sensations and their derangements in so far as they concern central vision; afterward their behavior in indirect vision.

II. The Form Sense and its Derangements.

237. Form Sense.—The form sense is measured by determining the visual acuity (see § 185). This is affected, first and very largely, by the state of the refraction and the accommodation (see §§ 168-171 and 176) and furthermore by any condition affecting the transparency of the refractive media or interfering with the functions of the percipient parts—by all sorts of diseases, therefore, of the cornea, aqueous, lens, vitreous, uveal tract, retina, and optic nerve.

The visual acuity, while it serves as a measure of the form sense, is affected by other visual factors and especially by the state of the light sense.

238. Blurring due to Derangements of Form Sense; Metamorphopsia.—Derangements of the form sense are evidenced by blurring of the visual image, which in some cases is blotted out altogether (§ 271). In many cases also it is distorted. This distortion is called *metamorphopsia.*

Metamorphopsia may be *refractive,* i. e., due to refractive defects, especially astigmatism (see §§ 170, 171), or *retinal,* i. e., due to displacement of the retinal elements. Retinal metamorphopsia occurs when the retina is lifted up by an exudate or underlying liquid or mass or is pulled by a scar. It is found therefore in retinitis, chorioiditis, detachment of the retina, and tumors of the retina and chorioid. If the process is

such that the retinal elements are spread apart, as for instance occurs in a recent chorioiditis or retinitis, an object looked at will look smaller than it is (*micropsia*). This is because a retinal image, that would normally occupy three retinal elements, now, owing to the distention of the retina, occupies but two, and hence affords an impression corresponding to that afforded by an object of only two-thirds the actual size of the object looked at. On the other hand, when the retinal elements are abnormally crowded together, as occurs in the atrophic stage of retinitis and chorioiditis, objects look larger than they really are (*macropsia*). Retinal metamorphopsia if due to chorioiditis or retinitis is usually combined with a complete or partial scotoma.

The best test for retinal metamorphopsia is a series of parallel lines like the lines of music (Fig. 115).

Micropsia and macropsia also occur in disorders of accommodation (see § 380) but are not then associated with distortion like that shown in Fig. 115.

239. Disturbances of Vision without Apparent Lesion.—The expressions amblyopia[1] (weak sight) and amaurosis[2] (absolute blindness) are at present used to denote conditions in which no changes in the eye itself can be found to account for the loss or impairment of vision.

Occasionally the term amaurosis is still used in a wider sense to denote total blindness, even when there are external changes.

The following conditions are classed under this head:

I. Disturbances of sight seated in the eye itself.
  (a). Congenital amblyopia.
  (b). Amblyopia ex anopsia.

II. Disturbances of sight of central origin.
  (a). Amblyopia and amaurosis in central nervous diseases.
  (b). Scintillating scotoma.
  (c). Hysterical and neurasthenic amblyopia.
  (d). Psychic blindness.

240. Congenital Amblyopia.—We assume this to exist in those cases in which weak sight has existed for a long time, and in which all other causes for it can be excluded. We are justified in making this assumption whenever other congenital anomalies are also present in the amblyopic eye, such as an extreme degree of hyperopia or astigmatism, coloboma of the iris or the deeper membranes, albinism, microphthalmus, etc. For such eyes almost always display a reduction of the visual power, which cannot be brought to the normal even by the correction of the error of refraction present.

Congenital amblyopia is usually unilateral; the effected eye is then very apt to fall into a state of squint. If the amblyopia affects both eyes, nystagmus develops (see § 356).

In view of the great frequency of retinal hæmorrhages in the new-born (see § 775), it is assumed that some of the cases of congenital amblyopia are due to a hæmorrhage of this sort which destroyed central vision and disappeared without leaving a trace of its presence.

241. Amblyopia ex Anopsia.[3]—Amblyopia from non-use occurs when there has been present from earliest childhood an obstacle to vision, which makes the formation of sharp images upon the retina impossible. Such obstacles include opacities either congenital or acquired early

[1] Properly blunt-sightedness from ἀμβλύς, blunt, and ὤψ, sight. [2] ἀμαυρός, dark.
[3] From ἀ priv., and ὤψ, sight.

in life, situated in the cornea, lens, or region of the pupil (pupillary membrane). A similar amblyopia from disuse occurs in eyes affected with high uncorrected refractive errors (especially high astigmatism) and particularly in the more ametropic eye in anisometropia. Amblyopia also develops in an eye which has squinted since childhood, because in this case the perception of the retinal images in this eye is suppressed, and the eye is thus purposely excluded from participation in the act of vision. In all these cases, the retina, owing to lack of exercise, fails to attain to that delicacy of function which belongs to normal eyes, or the functional capacity which has been already acquired is lost; but absolute blindness never occurs. If the condition causing the trouble is removed in childhood, the amblyopia may be then relieved by persistently exercising the vision in the amblyopic eye. This is especially

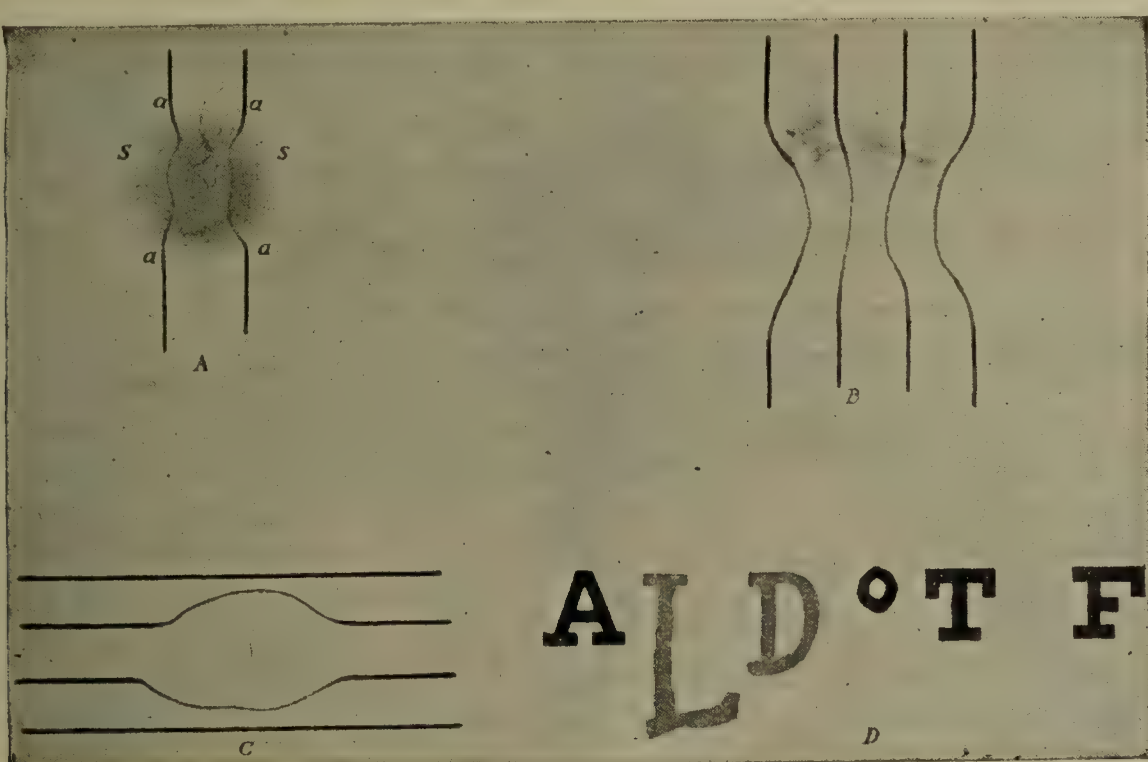

Fig. 115.—Retinal Metamorphopsia.

A, *Micropsia*, evidenced by an irregular bowing in of the parallel lines at *a*, *a*. Due to some cause producing distention of the retinal elements (detachment of retina, recent central chorioiditis or retinitis). The case depicted was one of central chorioiditis with a positive central scotoma, *s*, *s*. Within the scotoma the lines are curved in and are distorted. B, metamorphopsia in same case nine years after; positive scotoma still present. C, *Macropsia*, evidenced by an irregular bowing out of the parallel lines, *c*. Due to any cause producing shrinking of the retinal elements (atrophic chorioiditis, retinitis). The case depicted was one of solar retinitis in the translator's practice. D, the distortion and change in hue produced in a line of test-types by the metamorphopsia in this case. The letters are all really of the same height and blackness.

true of squint. Treatment is most efficacious if begun before the age of five but may give excellent results if undertaken two or three years later. If left untreated until adolescent or adult life, the amblyopia is generally not relieved, even though the cause of the visual disturbance is removed by operation.

Cases, however, have been reported, some surely authentic, in which an amblyopic squinting eye has acquired good vision either through correction of the refraction or because loss of sight in the good eye has compelled the use of the amblyopic eye.

The statement above made with regard to amblyopia from non-use is opposed by many who believe that in squint the poor sight is the cause not the result of the latter. But the weight of evidence seems to be for the view given in the text.

When—as in an adult—the development of the retina has once been completed, an obstacle to vision may last for many years without the retina's suffering any harm. Thus cataracts which have formed in adults have been operated upon with perfect success after lasting twenty years or more.

Treatment consists in the earliest possible removal of the obstacle to vision. This is particularly the case with infantile cataracts, upon which we may operate with the best results even in children at the age of a few months. Exercising the amblyopic eye is of service in bringing up the functional power of the retina. This is particularly employed in cases of strabismus, where by bandaging or atropinizing the sound eye we force the eye which squints to see (see § 342). In high astigmatism and in anisometropia proper correction of the refraction and exercising of the amblyopic eye may sharpen the sight considerably.

242. Amblyopia and Amaurosis in Brain Diseases.—Disturbance of vision may be set up by disease of the brain without there being any ophthalmoscopically perceptible changes in the eye, such as neuritis or atrophy of the optic nerve. If the disease affects but one side of the brain, the visual disturbance often appears under the form of hemianopsia (§§ 275-278), but if it is bilateral it may produce complete blindness.

Blindness of this sort even when complete, may be transient. *Uræmic amaurosis* which is produced by retention of the urinary constituents is a good example of this. In this condition the patient declares that everything suddenly becomes dark before his eyes; the disturbance of sight increases so quickly that the blindness gets to be complete within a few hours or a day. Even, however, when the blindness is absolute, the results of examination of the eye are usually negative. After one or more days the sight is gradually restored. Simultaneously with the attack of visual disturbance other nervous symptoms are found, such as headache, vomiting, dyspnœa, loss of consciousness, and convulsions—in short, the symptoms of uræmia. In other cases in which gross lesions of the brain, such as inflammatory processes or new growths, give rise to the disturbance of vision, the latter is permanent, and ophthalmoscopic changes, usually under the form of a descending atrophy of the optic nerve, are often associated with it later.

Disturbance of sight due to a central cause may originate in the cortex (*cortical blindness*), in the fibres ascending to the cortex, or, rarely, in the optic tract. If the lesion is seated in the cortex or in the supranuclear fibres ascending to the cortex, the reaction of the pupil to light is retained even if the blindness is total.

243. Psychic Blindness.—If the areas devoted to optical memories are destroyed, the stimuli reaching the brain no longer evoke the recollection of things already known. Objects in that case are seen but are not recognized (*psychic* or *mind blindness*).

Varieties of psychic blindness, also called object blindness are *word-blindness, letter-blindness*, and *number-blindness*, or inability to recognize or learn printed or written words, letters, or numbers. It or any of its sub-varieties may be congenital.

244. Scintillating Scotoma (scotoma scintillans, amaurosis partialis fugax).—The patient who suffers from this, notices besides a feeling of vertigo a sparkling light that appears before his eyes, and rapidly increases until finally he can scarcely see at all. Persons who are more accurate observers of their sensations usually aver that the sparkling originates from

a small spot situated not far from the point of fixation, and that within the scintillating area external objects are invisible (hence the name scintillating scotoma). The scintillation and with it the gap in the visual field spread rapidly, the boundaries of the scintillating area being often formed by lines zigzagging in and out, so as to form projecting and re-entrant angles. After a quarter or half an hour, the attack abates, the visual field beginning to clear up at the point first affected. Scintillating scotoma is usually accompanied by headache and sometimes also by nausea, and frequently a regular attack of migraine is joined with it.

The central origin of scintillating scotoma is apparent not only from the accompanying and following headache, but also from the fact that it almost always affects both eyes in the same way, and frequently occurs as a hemianopsia—i. e., occupies but one half (and that too, the homonyous half) of the field of vision in each eye. The symptoms, on account of their short duration can be caused only by disturbances of circulation, whose transient character indicates that it is not anatomical changes but disorders of innervation in the vessels that lie at the root of them. With extension of the angioneurotic disturbance to the other areas, other disorders of central origin, such as formication in the lips and arm, weakness or paralysis of one of the extremities, aphasia, etc., set in. These come and go with the scintillating scotoma. But scintillating scotoma is most intimately associated with the most frequent angioneurosis of the brain, i. e., migraine, so that is has also been called by the name of migraine oculaire. The circulatory disturbance sets up an irritation of the optical elements—an irritation which according to the laws of projection is referred to the external world, and appears under the form of a colored scintillation while at the same time the perception of peripheral impressions is abolished. So also at the beginning of a fainting attack, which is likewise due to circulatory disturbances in the brain, symptoms appear which are perhaps identical with scintillating scotoma; the patients averring that everything looks green and blue, or scintillates, or grows dark before the eyes.

Scintillating scotoma is very common. It may occur only at long intervals or on the other hand, recur very often—even several times a day. The causes alleged for the attack are excessive physical or mental exertion, straining of the eyes, dazzling light, or great hunger; often, however, no definite cause can be made out. In ordinary cases there are no evil consequences of any kind, and it is the exception for scintillating scotoma to be the precursor of a serious affection of the brain (brain tumor, progressive paresis, apoplexy, etc.). Treatment is addressed to the apparent cause of the condition (migraine, etc.); otherwise is symptomatic consisting in avoidance of excessive exertion and in measures to increase the general strength. If the attacks are frequently repeated, we may order quinine in small doses to be taken for some time.

245. Hysterical Amblyopia.—In hysteria we sometimes find a marked amblyopia or even amaurosis, the disturbance of sight being often associated with contraction of the visual field (see § 274), diminution in light sense and color sense, and asthenopia.

Hysterical amblyopia is found to the most marked degree in those cases of hysteria that are associated with disturbances of sensibility (hemianæsthesia, etc.). It is generally present in both eyes, although for the most part to a greater degree on the side upon which the general sensibility is affected.

The diagnosis is based principally upon two points. The first of these is the absence of any demonstrable changes in the eye which might explain the enfeeblement of sight. The second is the failure of the separate symptoms constituting the disturbance of vision to show that agreement with each other that they ordinarily present. Thus, the acuity of vision and the extent of the visual field change frequently (usually doing so as the other hysterical symptoms grow better or worse); the field itself shows a

peculiar character (see § 274); and persons with marked amblyopia or whose visual field is unusually contracted still move with perfect security and without stumbling in a space which is not well known to them; in fact, even those who are absolutely blind sometimes do so, too, if they think that they are not observed. Furthermore, the pupillary reflex to light is retained even when blindness is complete. It can be seen from this that it is often difficult to draw the line between simulation and a hysterical blindness—i. e., one having an actual existence in the imagination. In the latter case there may be other evidences of hysteria associated with the amblyopia which will render the diagnosis more certain.

Hysterical amblyopia chiefly attacks young people, particularly of the female sex. It is sometimes produced by injuries, even when they do not affect the eye itself (*traumatic neurosis*). Quite analogous is the condition erroneously called *shell-shock*, in which a profound nervous shock or the exhausting and nerve-racking conditions incident to modern warfare set up amblyopia or other symptoms.

Hysterical amblyopia affords a good prognosis, as ordinarily a complete cure takes place. The disease, however, usually lasts for a long time, often for years. Treatment consists in the management of the causal lesion, re-enforced mainly by suggestion and locally by hypodermic injections of strychnine and the application of the constant current. The brilliant results sometimes obtained by the two last-named remedies are, however, mainly ascribable to their psychic effect.

Along with hysterical amblyopia may be mentioned the condition known as *nervous asthenopia*, which occurs both in hysterical subjects and in neurasthenics, and consists in an incapacity of the eye for any continuous exertion, in spite of there being good visual power.

Some complain that after reading or working for even a short time everything becomes covered with a cloud so that the work has to be laid aside. Others, again, allege that after pursuing their occupation for a little while, indeed even after reading a few lines, they have violent pains in the lids, eyeballs, or head, which render the continuance of the work, impossible. When no strain is put on the eyes, there is generally no trouble; in other cases, however, the pains never entirely disappear, or a great sensitiveness to light is constantly present.

In making the diagnosis, proof must first of all be forthcoming that there is no error of the refraction or of the muscular equilibrium to cause the trouble. These cases are frequently associated with convergence-insufficiency and with insufficiency of accommodation, both of which may require treatment (see §§ 332 and 379). Other cases are due to reflex disturbances and remote infections (especially infection or irritation in the nose and its accessory cavities, in the teeth, and in the tonsils). Nervous asthenopia, like hysterical amblyopia, with which it frequently is associated, is often extremely obstinate and sometimes for years prevents the patient affected by it from engaging in any serious occupation. In it, too, the psychic factor plays a great part in the treatment. Electricity is particularly serviceable in this condition (Fuchs).

246. Simulation of Blindness.—In testing the sight we shall at times have to reckon with the fact that the patient is purposely trying to lead the physician astray by simulating blindness or weakness of sight when these do not exist. This most frequently occurs with those persons who wish to be relieved of military service or who wish to get damages for an accident. Quite similar are the symptoms in hysteria, in which, however, it is not a question of intentional deception, but of unconscious imagination.

We are first led to suspect simulation by the lack of agreement between the results of the functional testing and of the objective examination; an eye, for example, which is alleged to be perfectly blind, presenting no pathological changes of any sort. Or the

ests of the individual functions give contradictory results, inasmuch as the visual acuity, he field of vision, the color sense, etc., do not stand in the right relation with each other nd with the result of the objective examination.

Complete blindness of both eyes is rarely simulated; much oftener it is simply uniateral blindness that is alleged; and still more frequently a feebleness of sight actually present in one eye is exaggerated. In the case of an alleged complete blindness of one or both eyes we regard in the first place the reaction of the pupil to the light. If this is well preserved, it will always afford a strong ground for suspecting simulation, although there are rare cases in which in the presence of actual blindness the pupillary reflex for light is still retained (see § 367).

The methods, beside the pupillary test, used for detecting simulation of unilateral blindness or amblyopia depend mostly on the fact that one does not perceive with which of his two eyes he is seeing, if both eyes are kept open when the tests of sight are made. A man could get at this, if his two eyes were equally good, only by quickly closing one eye or the other for a moment while the sight was being tested, and care must, therefore, be taken that this is not done by the person who is being tested for simulation.

1. Bar-reading test.—We make the patient read, and then hold a pencil in a vertical direction between the eye and the book. If there is vision with but one eye the pencil conceals certain words from it, and thus interferes with reading. If, however, there is good vision with both eyes, those letters which are concealed from one eye by the pencil are visible to the other, and reading is carried on without difficulty (Cuignet). (Cf. § 305.)

2. A convex glass of 6 D is placed before the sound eye. In this way the eye is made artificially myopic, so that its far point lies at a distance of about 17 cm. (it being presupposed that the eye is emmetropic). The eye can therefore read fine print only at a distance of 17 cm. or less, but no farther. After placing the glass before the eye we first make the patient read at quite a short distance, and then slowly and imperceptibly move the book farther and farther away. If it is possible in this way to withdraw the book considerably farther than 17 cm. without the patient's ceasing to read, it proves that he has been reading with the eye alleged to be bad That is, he began reading with the good eye and, when the book was carried too far off for that, continued with the other eye, without noticing the alternation in the employment of the two eyes.

3. Snellen has constructed a board with test types which are alternately red and green. Before the patient is allowed to read it, a pair of spectacles is put upon him, in which are introduced a red glass for one eye and a green glass for the other. Through the red glass the red letters alone, and not the green, can be seen, because green is the complementary color of red, and therefore green rays are not transmitted through red glass. For the same reason the red letters cannot be perceived through the green glass.[4] If, therefore, any one who is blind in one eye looks through these spectacles at the test types, he will read off only the red, or only the green letters, according as the red or the green glass of the spectacles is placed in front of the eye which alone can see. He will not once suspect that still other letters of a different color lie between the letters that he has read. Should the patient, on the other hand, read all the letters, it proves that he sees with both eyes and in such a way as to recognize the red letters with one eye and the green with the other.

4. Letters are written on white paper with a black and a red pencil alternately. The subject under examination is then told to read the writing rapidly, while a red glass is held before the sound eye. If he reads the whole correctly, it is a proof that he is able to read with the eye alleged to be blind, for the sound eye, looking through the red glass, cannot see the red letters since these now offer no contrast to the background upon which they are viewed, which appears as red as they.

5. In the methods above given the malingerer may readily outwit us if during the examination he slyly closes the alleged poor eye and thus gets an idea of how he ought to see were it really poor. We can prevent this trick if we make him read out loud, and

[4] In this test of Snellen's the red and green letters are transparent and placed upon an opaque ground and are hung up before a window so as to be seen by transmitted light. In this case, as stated in the text, only the red letters are seen through the red glass, and only the green through the green glass. The same will occur if the red and green letters are opaque, and placed on a dull black ground, and viewed by reflected light. In either case the conditions are opposite to those which exist in the test next mentioned, in which opaque red letters are viewed by reflected light on a white ground.

then, while he is reading quite fast and is wholly occupied with what he is doing, suddenly place a prism of 2 *d* with the base down before the poor eye. Of course, we make sure in doing so that the eye is open at the time. Now, if the eye is really very poorsighted, it will make little difference to him whether the prism is placed before it or not, and hence he will read on as before; but if he sees fairly well with this eye the interposition of the prism will cause very great confusion by the production of superimposed double images and he will at once involuntarily pause and stumble in his reading and perhaps will be unable to go on at all.

III. The Color Sense and its Derangements.[5]

247. Physical Basis of Color Vision.—By the color sense we appreciate differences in the hue of objects.

Colors, in the widest sense of the term, include the colors proper and the neutral shades (white, black, and gray). These latter give rise to sensations essentially the same in character as the colors proper. Mixtures and transition forms between the neutral shades and the colors proper occur. Accordingly a color has in general three distinguishing characters, namely, (1) *hue* or color tone, dependent on the wave length of the light producing it; (2) intensity (*brightness*, luminosity); and (3) *saturation*, which increases in proportion as the amount of admixed white light diminishes. As the intensity (brightness) diminishes, the color becomes darker (fainter) and ultimately is replaced by blackness. As the saturation diminishes, the color becomes lighter (less colored) and ultimately becomes white.

An eye with normal color sense viewing a spectrum sees the well-known rainbow series of colors from red to violet. The transition from one color to another is effected by a change of hue and brightness only—there being no admixture anywhere of neutral shades. The brightness is greatest in the yellow (light having a wave length of 570 $\mu\mu$[6]) and diminishes from this point gradually in both directions. At the ends of the spectrum, on the contrary (in the red and the violet), the hue no longer changes but the brightness diminishes rapidly. These terminal portions in which this occurs extend from 655 $\mu\mu$ to 800 $\mu\mu$ in the red and from 430 $\mu\mu$ to 400 $\mu\mu$ in the violet.

248. Color Mixtures; Primary Colors.—A color like purple not contained in the spectrum can be obtained only by mixing red and violet. Again, if we mix two spectral colors, that are not too far apart in the scale, we get a color of intermediate hue.

If the two component colors are too far apart, the mixture formed is unsaturated, and if they have a certain degree of separation and the conditions are proper, the mixture is a neutral shade. Two colors like these (e. g. red and bluish-green) which thus form a neutral shade on mixture are called *complementary colors*. While therefore, the yellow, which lies between the red and the green, may by its mixture with either give rise to other hues (orange or yellowish-green), the complementary colors are mutually destructive, each excluding the other from sensation. The phenomena of contrast always occur under the guise of complementary colors.

Only a limited portion of the normal color system can be represented by a mixture of two spectral colors. If we wish to construct the entire color system by mixtures of this sort, we must select at least three colors (e. g. red, green, and violet). On this basis the color system of the normal person is called a *trichromatic system*.

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[5] The sections on the color sense and the light sense are with slight exceptions taken from Salzmann.

[6] The abbreviation $\mu\mu$ denoting micromillimetres or millionths of a millimetre, is used especially in denoting the length λ of light waves. An Ångström unit (A. U) is a tenth of a micromillimetre.

It does not follow from this, however, that there are only three *primary color sensations* (as, for example, is assumed in the Young-Helmholtz theory). The relations in the periphery of the visual field would rather lead us to assume with Hering that there are four primary color sensations, the colors concerned consisting of two complementary pairs, viz., red and green, and yellow and blue. Many, however, still believe the Young-Helmholtz theory the most satisfactory. A number of theories of color vision have been advanced, but none has served to explain convincingly all the phenomena observed.

249. Relation of Color Sense to Illumination.—The functioning of the color sense is more or less dependent on the illumination. The brighter the light, the more color there is in the world. Thus the landscape in full sunlight appears more full of color than when the weather is overcast. If the illumination sinks below a certain limit the perception of colors fails altogether, and everything appears gray on gray as in a photograph (*scotopic vision*). We then are aware of nothing but what Hering calls the "white value" of the colors. This is very slight in the red end of the spectrum, reaches its maximum in the bluish-green (at 510 $\mu\mu$), and from this point on to the violet diminishes but comparatively little. In the twilight, therefore, blue appears much brighter than red (Purkinje's phenomenon).

250. Affections of the Color Sense; Congenital Color Blindness.—Disturbances of the color sense are known as color blindness, and occur in a congenital and an acquired form. Congenital color blindness is not a disease but an imperfection of the visual sense due to unknown causes. It may be total or partial.

251. Partial Color Blindness.—Partial color blindness is characterized by the fact that but one pair of primary colors is perceived. All hues and variants of them of different degrees of saturation perceived by a person who is partially color-blind can be produced by mixing two spectral colors. His color system, therefore, is *dichromatic.*

In the spectrum the dichromate perceives only two primary colors, a "warm" and a "cold." As these are complementary colors, they merge into each other by a zone of neutral shade. Following von Kries we may distinguish three forms:

1. *Protanopia* (red-blindness of the older nomenclature). The neutral zone lies at 490 to 499 $\mu\mu$ (in what is the blue-green of the trichromates). At its red end the spectrum is shortened, and the terminal portion extends in to 540 $\mu\mu$. The brightest portion is at 570 $\mu\mu$ (in the green-yellow of the trichromates).

2. *Deuteranopia* (green-blindness of the old nomenclature). Like the preceding, but the spectrum is not shortened, and the brightest part lies at about 600 $\mu\mu$ (in the orange of the trichromates).

3. *Tritanopia.* The neutral zone lies at 575 $\mu\mu$ (in the yellow of the trichromates).

Protanopia and deuteranopia are very closely allied. To those affected with either, all hues which the trichromate distinguishes as red, orange, yellow, and yellowish-green, appear of the same color (probably yellow), but of varying degrees of brightness. Bluish-green appears to them a colorless gray. For this reason the two conditions are comprised under the name red-green-blindness, and they are accounted for on the supposition that one pair of primary colors (red and green) is absent, while the other pair (yellow and blue) is present. In tritanopes probably the primary sensations of

yellow and blue are wanting but red and green are present. This condition, which is hence called blue-yellow-blindness (Hering) or violet-blindness (Helmholtz), is so rare that it has been but insufficiently investigated.

252. The three kinds of dichromates seem to be sharply differentiated from one another, there being apparently no transition forms between them. But there are a series of transition forms between red-green blindness and the normal condition of trichromasia. Persons thus affected are said to have a weak color sense or to be *anomalous trichromates* (König). They can perceive red and green if the object is seen under a large enough visual angle, is well illuminated, and is under view sufficiently long. They become uncertain, however, if the visual angle is small, the colors are not very bright nor quite saturated, and particularly if only a short time is allowed for the observation (e. g. if colored lights suddenly flare up and then disappear). According to Köllner, the contrast between red and green is increased, so that white lights alongside of red are easily taken for green.

253. Total Color Blindness.—Those who are totally color-blind can distinguish no hues at all but simply differences in brightness. Their color system can be represented by varying the intensity of a single spectral light, i. e., it is monochromatic. The spectrum looks like a colorless band, the greatest brightness of which is at the point where the trichromate sees the green. The variation in the degree of brightness in different parts of the spectrum is the same as in the scotopic vision of a normal person (see § 249).

254. Frequency of Congenital Color Blindness; Symptoms.—If we include the anomalous trichromates and make the tests for color blindness sufficiently searching, we find that from 10 to 11 per cent of men and about 1 per cent of women are affected by it (Schiötz). It is transmitted by sex-linked recessive heredity. It is occasionally unilateral. Tritanopia and total color blindness are very rare. On the other hand, red-green blindness is very frequent and anomalous trichromasia probably more frequent still, so that these conditions have considerable practical significance.

In respect of other functions of vision (visual acuity, light sense), the partially color-blind show not the slightest deviation from normal, but the totally color-blind regularly have diminished visual acuity, photophobia, and nystagmus. Sometimes, too, a central scotoma can be demonstrated. Their dark adaptation, however, is normal or actually increased.

Since their vision in other regards is normal, those who are affected with red-green blindness suffer no inconvenience from it except that it unfits them for certain *callings*, like painting, decorating, etc., which require a precise discrimination of colors. Their unavailability for such pursuits, however, is not absolute; there are, for example, color-blind painters, whose work even with regard to color stands among the highest in the art. On the other hand, it would be a great mistake to impute the peculiarities in coloration or drawing that are constantly exhibited by certain artists to any deficiency in their visual sense. But, such occupations apart, it is of the greatest importance that those concerned with the *railway* and *marine services* should have normal color vision, since the signals which are used in these services are mostly red and green and any confounding of these signals may result in the most calamitous accidents. As a matter of fact it was a railway accident in Sweden, the cause of which was attributed to color blindness in the engine driver, that started the first compulsory examinations of the color vision. At

present such examinations have been introduced into almost all countries, and only those are admitted to the service whose color vision is good.

Many of those affected with red-green blindness are not aware of their defect, and are much grieved or affronted if they are charged with it. In dealing with such persons one should not forget that they lack not only certain sensations but the concepts that go with the latter; that they cannot, in fact, understand that there are men who see more colors than they themselves do. There is the further source of misunderstanding that they can really distinguish up to a certain point between red and green not, of course, by the hue but by the brightness of saturation of the "warm" color which they perceive in the less refractive portion of the spectrum. In ordinary life they rarely make mistakes in naming colors. In many cases the relations between objects and their colors (or the color names assigned to them) are fixed, and daily experience thus teaches the color-blind to call colors by the same names as other people do. But if one shows them colors under conditions in which their experience fails them and if by changing the brightness and saturation of the test-objects one deprives them of their only means of distinguishing red and green, it will then turn out that they will confuse colors which to a normal trichromate appear radically different. Thus, those affected with red-green blindness confuse bright red with dark yellow, brown, or olive green, a rose or carmine red with gray, bluish-green or a brown-green, and a green with a gray-yellow, brownish-yellow, or a gray-rose.

Some believe that congenital color blindness can be helped by *exercise*. This may possibly be true of the anomalous trichromates, but even here for the reasons just given it may be difficult to tell whether an apparent improvement is real or spurious.

255. Acquired Color Blindness.—Acquired color blindness (achromatopsia) is a frequent symptom of disease or injury of the sensory apparatus i. e., of the retina, optic nerve, and cerebral conduction paths.

The disturbance of color sense in these cases is at first limited to a portion of the visual field, and at this stage is discoverable only by determining the visual field with colored objects (§ 269). It is only when the disturbance has extended over the entire field of vision that we call it color blindness. Those thus affected and also those affected with acquired color defects confined to a part of the field (e. g. those who have a central color scotoma) differ from the congenitally color-blind in that they can give correct answers as to their lack of perception of this or that color, since they once had normal color sense and with the color names associate the same concepts as normal persons do. There is this further difference that in acquired color blindness the brightest part of the spectrum is in the yellow, so that the vision in such cases corresponds to vision in the outermost, color-blind area of the visual field. In acquired color blindness the other visual functions, such as the visual acuity, peripheral vision, and the light sense, are often greatly impaired.

Particularly important from a practical point of view is the partial acquired color blindness produced by a tobacco or alcohol amblyopia (§ 820). This causes a central color scotoma, which in the railway and marine services has the same evil results as an congenital color blindness.

256. Tests for Color Blindness.—The tests for color blindness depend mainly on the use of confusion colors. In *Holmgren's wool test* the person examined is required to select from a large number of colored skeins of wool those which match a given sample. If in so doing he makes gross errors, e. g., if he puts wools of a gray, light brown or reddish hue with a pure green, or violet or blue wools with a bluish-red, he is proved to have red-green blindness.

The wool test is unsatisfactory because not a few color-blind persons are able to pass it successfully but may be used, especially in the self-registering modification devised by Jennings, as a rapid test to sift out the obviously color-blind, doubtful cases being reserved for the more delicate tests. Of these latter the best are Nagel's and Stilling's.

Stilling's pseudo-isochromatic plates are made with the aid of color-blind painters. The confusion colors are put on in round or irregular spots, those of one color forming the background and those of the other forming numbers. A man with good color vision can read all the numbers because for him they show a difference in hue, but the color-blind person cannot read the plates if they are constructed with the colors that he confuses. Quite the same principle underlies *Edridge Green's* cards used in the English marine service.

In *Nagel's test*, which may also be used as a quantitative test (see below), there is a set of cards, each bearing a series of little color discs arranged in a ring. In some rings the discs are all of one color, but of slightly different shades; in others the discs are of two or three different colors (confusion colors). By making the patient state which rings are monochromatic and then making him pick out in the dichromatic or trichromatic rings all the discs that are of one special color, we can readily ascertain whether he is color-blind and what sort of color blindness he has. This and Stilling's test, supplemented by the lantern test (see below) afford the simplest and most satisfactory means of determining the presence of color blindness.

For examining persons in the railway and marine services, one of the best tests is a *lantern* having a revolving diaphragm containing different colored glasses and provided with devices for varying the aperture and reducing the illumination so as to simulate the appearances gotten in bad weather (fog, etc.).

Since the colors that are confused by the color-blind are not the same for everybody thus affected, it may happen that a color-blind person will pass the tests mentioned, which are limited to certain hues and combinations. Such persons can be detected only by an apparatus, by which what is called the normal color equation can be produced. Such an apparatus is Nagel's *anomaloscope*. In this the observer, looking through a tube, sees a circular field divided by a horizontal line into an upper and a lower half. The lower half is illuminated by homogeneous yellow light of a hue corresponding to the sodium line of the spectrum, (sodium yellow), the upper by a mixture of lithium red and thallium green light. By turning a graduated screw at the left side of the apparatus the relative proportion of the red and green lights can be continuously altered and thus all hues from a pure red to orange, yellow, and a yellowish-green can be produced. The only variations that can be made in the lower half are those of brightness, and these are effected by turning the screw on the right hand side. In this way with any eye the upper and lower halves of the field can be made to match exactly, making thus what is called Rayleigh's equation. What this is in the given instance is indicated by reading the position of the graduations on the screws. For the normal person these readings vary within very narrow limits. In red-green blindness it is possible to make a match, if a pure red or pure green is set in the upper half. This, of course, a normal person cannot do. Anomalous trichromates, as a rule, in order to make a match, require a mixture of red and green different from the normal or betray themselves by their uncertainty in adjusting the apparatus.

The acuity lantern devised by Ferree and Rand may be used for making color mixtures and detecting even minor deficiencies of the color sense.

The demonstration of the existence of color blindness requires accurate and careful testing. Many of the color-blind who are aware of their defect try to conceal it from the examiner, especially if some material advantage, such as, for instance, a business position, depends upon the result of the testing. Accordingly, in the case of such persons we must be on the lookout for all sorts of artifices, and particularly on the lookout for previous practice in the ordinary methods of testing the color sense. On the other hand, people with a good color sense might be considered to be color-blind if from want of education

or practice they call the colors that are set before them by incorrect names. We should not, therefore, undertake to test the color sense by setting colored objects before the person and asking him the *name of the color*. If we do so, the man who is color-blind will in many cases by using a little attention give the right answer, while on the other hand, an uneducated man will not infrequently call the colors wrong.

A *quantitative test* for color blindness may be made either by determining the smallest visual angle under which an object appears as colored or by determining in the same way as with the light sense the threshold and the minimum difference of color distinction. Apart from Wolffberg's diagnostic color apparatus, none of these methods has been developed into a means actually applicable to clinical diagnosis.

257. Perversions of Color Vision.—Sometimes objects appear abnormally colored (*chromatopsia*[7]).

The most frequent perversion of this sort is that in which objects look red (erythropsia)[8]. This occurs in normal eyes which have been exposed to a very bright light, especially light reflected from snow. It occurs particularly in those where pupils have been enlarged by atropine or an iridectomy and is very often present after a cataract extraction. In these aphakic patients its persistent recurrence may cause much concern. Sometimes, e. g., in jaundice and in cases of santonin (rarely in digitalis) poisoning, objects appear yellow (*xanthopsia*[9]). Much less frequent are blue vision (*cyanopia*[9]), violet vision (*ianthinopia*[10]), and green vision (*chloropia*[10]). These occur sometimes in diseases of the retina and chorioid, sometimes in general diseases and in poisoning (digitalis). Cyanopia may occur as a result of removal of the lens, developing then immediately after the operation and regularly disappearing within a month.

IV. The Light Sense and its Derangements.

258. Light Sense; Adaptation.—The light sense is the faculty by which we distinguish between light and dark and between differences in brightness of illuminated objects. It may be regarded as the primitive element in the visual sensations, without which the other elements (form sense, color sense) cannot function and upon the activity of which they are to a considerable degree dependent. Thus if the illumination steadily diminishes, as in the dusk, the visual acuity diminishes too, then color sensation disappears, and finally nothing at all is seen, even though the absolute brightness has not yet been reduced to zero. Furthermore for the actual detection and discrimination of distant objects, particularly under the conditions obtaining in the military, marine, and railway services and in all sorts of out-door occupations the light sense is often more important than visual acuity itself. (cf. §§ 187, 188.)

It is thought that the appreciation of differences of brightness is effected by the rods in the retina.

The minimum degree of brightness which can produce a sensation of light is called the *threshold stimulus* or light minimum (L.M); the least difference in brightness which can just be appreciated is the minimum difference or *light difference* (L.D). The threshold stimulus has very different values, the differences depending on whether the eye under examination was in the light just before being tested or had spent some time in darkness. A man who goes from a brightly lighted room into a dark one at first sees nothing at all. But the longer he stays in the dark, the more his vision adjusts itself, so that finally he

[7] From χρῶμα, color, and ὤψ, sight.
[8] From ἐρυθρός, red.
[9] From ξανθός, yellow.
[10] These terms are derived respectively from κυάνεος, blue, ἰάνθινος, violet, and χλωρός, green.

can get about quite well. If he then goes back into a light room, he is at first so dazzled that he again sees quite badly, and again it takes some time for the sense of dazzling and the disturbed vision to disappear. This process of adjustment of the eye for to the illumination present is called *adaptation,* a term which comprises *light-adaptation,* or the adjustment from dark to light, and *dark-adaptation,* or the adjustment from light to dark. The condition obtaining when the eye is completely adapted for bright light is called by Parsons *photopia* and that when it is adapted for the dark as *scotopia.*

Light sensitivity and adaptation are *less at the fovea* than in the periphery of the retina, hence when the illumination is reduced central vision is first affected and if the eduction is carried far enough a central scotoma develops.

259. Measurement of the Light Sense and Adaptation.—Tests of the light sense are usually made under conditions of dark adaptation. Among the instruments used are the *photometer* of Förster (Fig. 116) and the more

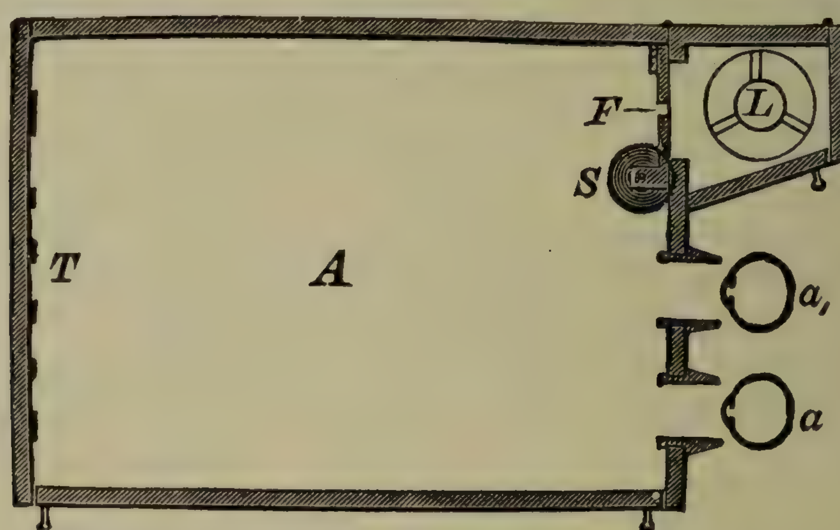

FIG. 116.—INSTRUMENTS FOR MEASURING THE LIGHT SENSE.

Försters photometer. The photometer consists of a box, A, blackened on the inside and having in its front two orifices for the eyes of the patient (a, a_1). T, the object consisting of broad black stripes on a white background. This is illuminated by a normal candle, L, shining through a window, F, covered with greased paper. By turning the screw, S, the area of F can be varied by an amount varying from zero to 5 cm. square.

serviceable *adaptometer* of Nagel. In the main, the method of examination with the two instruments is the same. The subject is brought into a perfectly dark room and directs the eye that is to be tested at a surface, the illumination of which can be varied within wide limits. The least degree of illumination with which the surface can be distinguished against the darkness about it gives the *light minimum.*

A handy method of measuring the light minimum for clinical purposes is a rotating black disc with white sectors. When the disc is rapidly rotated the white sectors are converted into gray circles diminishing in intensity from the centre of the disc outward. The ability to perceive these circles measures the light minimum. Thus one who can just see the outermost, faint circle has a light minimum of 200 (normal); one who can distinguish only the innermost circle has a light minimum of 50, etc. A similar disc, but white with black sectors measures the *light difference,* the normal here being represented by the ability to distinguish the middle ring (light difference of 100). This test has the advantage that it can be made in daylight without a preliminary period of dark adaptation, but its reliability under these conditions is still to be proved.

Ferree and Rand's acuity lantern in which the illumination of the test-object used in determining the visual acuity may be altered by a measurable amount, can also be used as a test of the light sense.

In order to determine the rate of the *adaptation,* we first make the patient stay in the bright daylight until he is completely adapted for light (has photopic vision). We

then test his light sensitivity directly after coming into the dark room and keep repeating the test at short intervals until he is completely adapted for the dark (has scotopic vision).

With an instrument like Förster's the light minimum is measured by the size of the aperture F, with which the object, T, just becomes visible. It is usually assumed in interpreting such measurements that the *light sensitivity* is inversely proportional to this light minimum, i. e., to the minimum illumination just producing visibility. This, however, is an erroneous assumption. Usually in those who start in with an eye fully adapted for light an illumination of 1 metre candle is required for the minimum stimulus, while after 45 minutes an illumination of only.00001 metre candle may suffice (Nagel). This does not, however, mean that the light sensitivity has increased 100,000 times in the interval. When the illumination is great, large differences in it produce no more effect than minimum changes when the illumination is small. The ordinary adaptation curves, therefore, according to which there is practically no increase in light sensitivity during the first ten minutes, a marked increase during the next ten, and an extreme increase during the ten succeeding, gives a quite erroneous conception of what takes place. Rather should we say that there is a steady and considerable increase during the first twelve minutes, then a gradually diminishing increase until about thirty minutes have elapsed, after which the sensitiveness changes very little, although some increase can still be noted for hours later (Best and others). In any event, whichever view we may adopt, we may say that in normal persons a practically complete dark adaptation is reached in 48 minutes, so that the light minimum taken at this time gives a full measure of his light sensitivity. Hence often but a single test is taken after this interval; but if the test then shows a low degree of sensitivity (high threshold) it should be repeated later to determine what the ultimate sensitivity is.

The light sense may appear to be defective simply because the pupil is narrow even in the dark. In such cases the pupil should be dilated when the test is made (Grafe).

260. Disturbances of the Light Sense.—Even in normal persons there are considerable differences in the light sensitivity and in the rate of adaptation. Both also may be pathologically affected, either the adaptation being too slow or the ultimate light sensitivity after adaptation has been effected being below the normal. Such changes are by no means necessarily in relation with the visual acuity, and they may in certain occupations cause a relative incapacity more important than that which a moderate reduction in visual acuity would produce (cf. § 188).

Disturbances of the conducting apparatus (optic nerve and retinal connections) act particularly to cause a reduction of the light difference (LD): disturbance of the percipient apparatus (rods of the retina) cause reduction of the light minimum. The former, therefore, is particularly affected in optic neuritis and atrophy and in toxic amblyopia, the latter in chorioiditis and deep retinitis. In diseases confined to the transparent media (cataract, corneal opacities, etc.), the reduction in light sense is comparatively slight and in any case affects the light minimum and light difference equally. Accordingly, if in opacity of the media, the light difference is markedly reduced as compared with the light minimum, we may infer some lesion of the optic nerve and in the contrary case, some lesion of the chorioid or deeper parts of the retina (Percival.)

Considerable disturbances of the light sense are evidenced at once by the fact that even without any special tests the relation between vision by bright light and vision by reduced light is obviously different from the normal. These disturbances we group under the names of hemeralopia and nyctalopia

261. Hemeralopia [11] (Night Blindness).—By hemeralopia, in the widest sense of the term we understand that condition in which one sees well by

[11] From ἡμέρα, day, and ὤψ, sight.

day, but by night or under feeble illumination from any cause, e. g., if the room is darkened, sees poorly or not at all.

In hemeralopia tests with the photometer show that the light sense is considerably reduced. In those affected with it the retina functions normally if set into action by sufficiently strong stimuli, but as soon as these stimuli sink below a certain limit it no longer reacts to them (*torpor retinæ*).

Hemeralopia is not a substantive disease, but simply a symptom which may occur in various diseases. It might be caused either by opacities of the refractive media, or by disease of the sensory apparatus.

(a) *Opacities in the media* excite the symptom of hemeralopia when they occupy the periphery, and leave the centre free, as in the case of peripheral opacities of the cornea and lens. Under brilliant illumination, when the pupil is contracted these no longer fall within the area of the latter, while, when the illumination is diminished and the pupil is dilated, they project into the pupil and interfere with sight. Again, when there are faint diffuse opacities distributed uniformly over the entire cornea, the sight is often better when the pupil is contracted, because the dazzling due to diffused light is then less.

(b) Hemeralopia occurs in diseases of the *sensory apparatus*, especially in retinitis pigmentosa and in atrophy of the retina due to retinitis, chorioiditis, and glaucoma, i. e., in conditions associated with marked reduction or absence of peripheral vision. As the light sense is especially active outside the fovea, any reduction in it is particularly marked in peripheral vision, producing thus an annular or concentric contraction of the field (see § 273). This sort of hemeralopia is permanent.

In another variety of hemeralopia due to sensory disturbance (*idiopathic hemeralopia*) no changes whatever are found in the interior of the eye. But in most cases there is a xerosis of the bulbar conjunctiva (see § 507); i. e., we find in the latter upon the outer and inner side of the cornea a small rounded or triangular spot, over which the surface of the conjunctiva looks dry and seems as if covered with a fine, whitish foam. Xerosis of the conjunctiva has no other connection with torpor retinæ than that both are symptoms of a reduced state of nutrition probably due to lack of vitamines in the food. It is found in people who in general are insufficiently nourished, as the inmates of workhouses, penal establishments, or orphan asylums, soldiers (very frequently affected in the recent war), sailors (in these occurring simultaneously with scurvy), and persons weakened by hæmorrhage (Terson). Furthermore, hemeralopia is sometimes observed with jaundice, with intermittent fever, with chronic alcoholism, and also in pregnant women. In persons who are thus predisposed to it the disease develops usually in spring. It often affects a number of persons at the same time, and men more than women (especially men of middle age).

The prognosis of idiopathic hemeralopia is favorable, as the disease usually gets well of itself after some weeks or months. It, however, leaves behind it a tendency to recurrences which usually occur in the spring or summer.

As regards treatment, the use of cooked liver and of cod-liver oil have for a long time enjoyed a great and deserved repute among the laity. We also seek to increase the nutrition by strengthening diet and by corroborative remedies and we protect the eyes from light, in the lighter cases by dark glasses, in the severer cases by keeping the patient in a dark room. By this treatment the disease is shortened.

Hemeralopia in conjunction with xerosis of the conjunctiva is also found as a precursor of keratomalacia, which likewise must be regarded as the consequence of a disturbance of the general nutrition (see § 559).

262. Nyctalopia[12].—This is the opposite of hemeralopia—i. e., is the condition in which the sight is better at night or in diminished illumination than in bright daylight.

It, too, occurs in two groups of diseases which have their seat either in the media or in the light-perceiving apparatus—only, in this case the site of the changes is just the

[12] From νύξ, night, and ὤψ, sight.

reverse of that found in hemeralopia. The opacities of the media causing nyctalopia are centrally situated (in the cornea, pupil, or lens). Hence, when the pupil is contracted they occupy its entire area; on the contrary, when, owing to diminished illumination, the pupil dilates, its peripheral portions which are still transparent can be used for seeing. The affections of the light-perceiving apparatus causing nyctalopia are those in which the outlying portions of the field of vision are normal, while in the centre there is a scotoma. In these cases, to be sure, the visual acuity is ordinarily no better with diminished illumination than it is in full daylight, but the feeling that central vision is blunted is less unpleasant, so that the patient imagines that he sees better in the evening. This symptom is most pronounced in toxic amblyopia (see § 820).

V. Peripheral Vision and its Derangements.

263. Peripheral Vision.—*Peripheral* or indirect vision is vision with those parts of the retina which do not belong to the fovea centralis and which comprise by far the greatest part of the retina. Vision with the peripheral portion of the retina affords a less distinct[13], a duller sensation, of which we can best get an idea by holding the outspread fingers of our hand to one side of the eye, while the latter is looking straight ahead. The farther from the fovea centralis is the image produced upon the retina, the more indistinct is the perception of its shape. For perceptions of movement, on the other hand, as well as of slight differences of luminosity, the periphery of the retina is quite sensitive.

Of what use, then, is peripheral vision, if we can get no well-defined perceptions with it? We can best understand this by observing persons who have lost peripheral vision to such an extent that only the fovea centralis and its immediate vicinity retain their functional activity, as happens in many diseases, especially in retinitis pigmentosa. Such persons can sometimes still read the finest print, and yet are in no condition to go about alone. We can put ourselves in this condition if we fasten in front of the eyes a long tube which allows us to see only the point lying directly in front of our line of vision. We cannot go about with such an apparatus because we strike against objects everywhere. Peripheral vision, therefore, is of service in orientation. How? If, as we are walking, we look straight before us and there is a stone lying in our path, the latter forms an image in the periphery of the retina of our eye, in this case in the upper part of it. The stone, to be sure, is not distinctly perceived, but still it excites out attention. Our gaze is then directed at it; it is seen directly; we recognize it as an obstacle and avoid it. The same thing happens if we go out upon the street and men come toward us from one side, etc. The images falling upon the periphery of the retina give us warning signals which make us cast our eye directly upon the objects which produce the images. And it is precisely moving objects that are most sure to attract our attention, since, as just stated, the peripheral portions of the retina have a high degree of sensibility for the perception of movement.

The maximum sensitivity to light is found in a zone 10° to 20° from the fovea. Hence, we can best distinguish a faintly luminous object (a star, for example), by looking 10° or so to one side of it.

264. Examination of the Field of Vision; Confrontation Test.—The examination of the field of vision—that is, of the limits of indirect vision—must be made for each eye separately. The eye examined is directed at a fixed point, in order that it may thus remain steadily in the same position, while the other eye is kept closed. We then determine the limits of the field for the perception of objects as objects (so-called form field, although the

[13] According to Burckhardt, the visual acuity of an excentric portion of the retina is 1 divided by $3n$, where n = the distance in degrees from the fovea (Leber)

perceptions involved concern the light sense much more than they do the form sense); and also the limits of the field for the perception of colors (color field).

The simplest way of investigating the extent of the field of vision is by the *confrontation test*. For this, if the patient's vision is still fair, we may use as a test object a white ball or a white card with a round black spot 1 to 3 mm. in diameter on each side of it; or for color tests a small colored square on each side of a gray card, the gray having the same light value as the color that it bears (Holden). The observer stations himself opposite the patient and in such a way that the background and the illumination are alike for

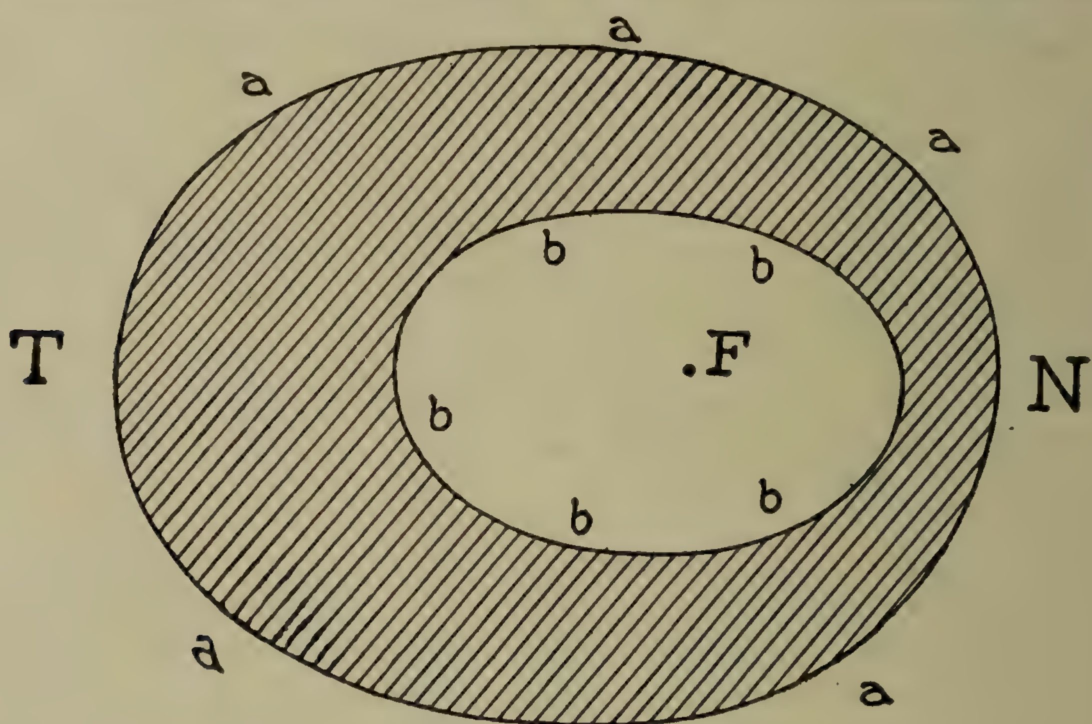

Fig. 117.—Plot of Field taken by the Confrontation Method.

The observer with one eye shut gets an idea of his field of vision in the other as a space bounded by an oval, *a*, *a*, *a*, which, reckoning from the point of fixation, *F*, extends apparently about twice as far to the temporal side, *T*, as to the nasal, *N*, and also extends somewhat further below than above. The patient in the present case sees the test object only half as far out as the observer does to the temporal side, and two-thirds as far out to the nasal, while his field above and below is contracted about one-half. His field, therefore, has the extent shown by the curved line, *b*, *b*, *b*.

both. The patient closes his left eye and with his right looks straight into the observer's left eye. The observer closes his own right eye, looks with his left straight at the patient's right eye, and holding the test card midway between the patient and himself, carries it from the periphery to the centre, noting the point where he himself first sees the dot and then the point where the patient sees it. If these two points coincide, the patient has a normal field in that special meridian; otherwise his field is abnormal Thus, if under the given conditions the observer begins to see the spot at a certain distance from the centre but the patient does not see it until it is carried half way in, the patient's field is contracted one-half in that meridian.

By repeating this test for the chief meridians in succession, quite an accurate plot of the field may be made as compared with the field of the normal eye (see Fig. 117). Indeed, for clinical purposes the test often gives quite as reliable and accurate results as the more seemingly scientific methods described later.

If the patient's vision is so poor that he cannot see the cards or ball, the surgeon uses his own hand as a test-object.

265. If the patient can no longer see even the hand, we must use a *candle flame* which we carry about through the field of vision. In this way, for instance, we test the visual field of a person who is blind from cataract. In doing this we place the patient in a dark room, heavily blindfold the eye not under examination, and direct the patient to keep the other eye fixed steadily in one direction. We then carry a candle in the different parts of the field, and, as we do so, alternately shade and uncover it. The patient, if his field is normal, must be able to tell not only when it is light and when it is dark, but also just where the light is at any moment.

The reason why it is essential that the patient should not only declare that he sees the light, but also say where it is, is this. It not infrequently happens that he recognizes the glimmer of the light as soon as the candle flame appears in the periphery of the visual field, but that he tells its place wrong. He says every time, for instance, that it is on the right hand even when it is held in some quite different spot. This is accounted for as follows: When an eye with transparent media is tested with a candle flame in a dark room, there is formed upon the retina, at a point opposite to the flame, an image of the latter, while the rest of the retina is not illuminated and has a sensation of darkness. If that part of the retina which is situated directly opposite the light were insensitive, no light would be seen at all. The case is different with an eye whose media are cloudy. In this the rays emanating from the light are so dispersed by the cloudy media that the whole retina is illuminated no matter where the light is placed. To be sure, the illumination of the retina is not perfectly uniform. There are always more rays falling upon that part of the retina which lies opposite the light than upon the other regions of the retina, and thus the patient is able to tell where the light is; but he would also see the light if the portion of the retina lying opposite the latter were insensitive, since the rest of the retina likewise receives light upon it. Let us assume that the whole retina has become insensitive, with the exception of a region situated on the temporal side. This latter region, no matter where the light may be, will receive diffused light and will perceive it, too. The patient will locate the source of this sensation in the portion of the external world lying opposite to this region of the retina, and will therefore believe always that what he sees is on his nasal side. Hence the mere statement that a light is seen is not sufficient proof of the possession of functional capacity by all parts of the retina. For this it is requisite that the situation of the light be told correctly every time.

A patient may not be able to keep his eye still; but if when the light is exposed he directs his eyes each time quickly towards it, without making searching movements, projection is presumably correct.

266. Tangent Plane.—We get at the field of vision more exactly by means of a blackboard or a curtain (Bjerrum's curtain), forming a *tangent plane*. We place the patient before this and take care that during the examination the distance between the eye and the plane remains always the same. Directly opposite the patient's eye we make a mark on the plane, and direct him to fix his gaze on this mark during the examination. The test-object is now gradually brought from the edge of the plane to its centre, and the patient is to tell at what moment he first sees it. By marking on the plane the limits of the visual field in every direction and connecting the points thus determined, we fix the extent of the field of vision. The size of the latter is, of course, in direct proportion to the distance at which it is taken.

A convenient form of Bjerrum's curtain is shown in Fig. 118. This is used at two distances, viz. 30 inches (0.75 metre) and 60 inches (1.50 metres). The former distance is used in determining limitations of the field lying anywhere within 50° from the centre

(especially sector-like and hemiopic limitations), in determining limitations of the color-field, in mapping the field of fixation (§ 306) and in plotting double images (§ 307). The 60-inch distance is used when we wish to delimit central and paracentral scotomata (§ 271) and enlargements of the blind spot (§ 270). Diagrams for plotting defects at these two distances are shown in Figs. 123 and 148.

Another very useful form of the tangent plane is Peter's *campimeter*. For use at the bedside or in a patient's home Peter has a hand campimeter.

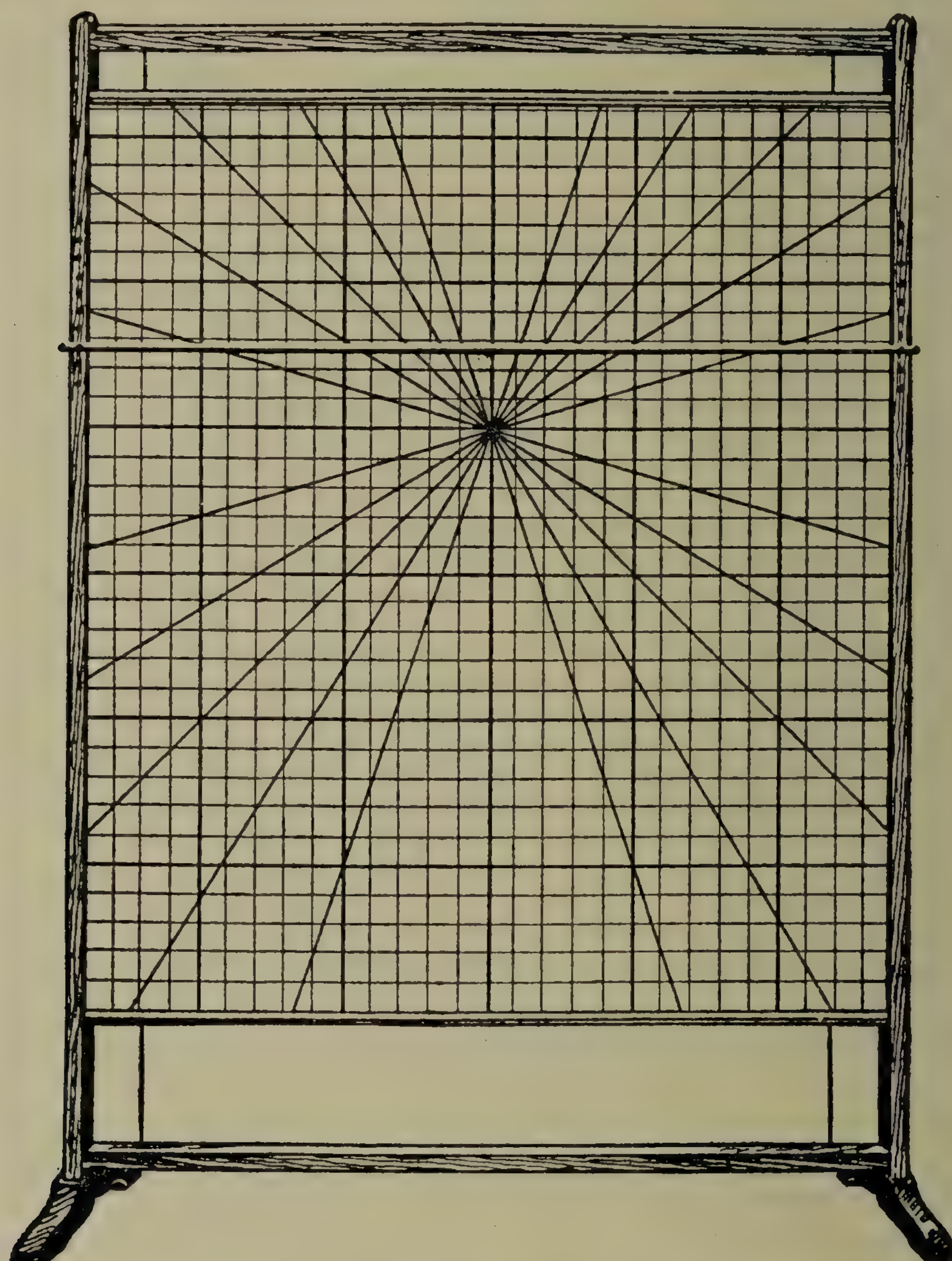

Fig. 118.—Tangent Plane for Plotting Central and Paracentral Scotomata, the Field of Fixation, and the Double Images in Paralysis.

The figure shows the reverse, or white side of the curtain forming the tangent plane, the other side, turned toward the patient, being a dead black and without markings except for a white-headed pin right at its centre used as the object of fixation. The small squares of the checkerboard in the figure denote two-inch (5 cm.) intervals, the larger squares intervals of 10 inches (25 cm.). Pins are thrust into the black side of the curtain, to show the outlines of scotomata, the limits of the field of fixation, the site of double images, etc. The plot outlined by these pins on the reverse side of the curtain may be transferred at once to a chart (Fig. 123) which has printed on it a diagram like that shown in the figure.

267. Perimeter.—Even this method is not entirely free from drawbacks which spring from the difficulty of projecting a hollow sphere like the retina upon a plane.

One difficulty is that unequal distances in the field of vision correspond to equal distances on the retina. Thus, in Fig. 119, the distances *m a* and *b c* upon the retina are equal, each corresponding to an angle of ten degrees. In the field of vision projected on the board, *T T*, however, the section (from 70° to 80°) that corresponds to the second region of the retina is many times greater than that (from 0° to 10°) which corresponds to the first. Hence, a spot upon the retina of definite size that has become insensitive

to light would in such a visual field appear as a gap, the size of which would be quite different according as it is nearer to or farther from the centre. This discrepancy is really of very little moment, since in the case of a central defect it is not large enough to be important and in the case of a peripheral defect we are concerned not so much with its actual size as with its effect in limiting the patient's peripheral vision; and, as the patient habitually projects objects on a plane, a peripheral defect of 10° really does look much larger to him than a defect of 10° near the centre and the representation made by the tangent plane consequently gives a truer idea of his subjective limitation than does that made by the perimeter. Moreover, if desired, the defect plotted on the tangent plane can be put on a chart in which the degrees at the periphery and the centre are of the same size.

Another more important objection to the tangent plane is that the whole of a normal visual field does not find a place on it be it ever so large. The normal field of vision, that is, extends outward to 90° and more. Therefore, as is evident from Fig. 119, the temporal limit of the visual field can never be projected on the tangent plane.

In order, then, to represent the entire visual field, it is projected upon a hollow sphere (Aubert). Upon this principle different *perimeters* are constructed. The perimeter consists, not of a complete hemisphere, but of a metallic semicircle (Fig. 119, *P*; see also Fig. 120) which represents, as it were, one meridian of the hemisphere. The semicircle is capable of being revolved so as to take the direction of each meridian in succession. The patient supports his head on a chin-rest which is so placed in front of the semicircle that the eye to be examined is situated in the centre of curvature of the latter. In the examination, the eye must be fixed upon the middle point of the semicircular arc, while the mark that serves for the test is carried to and fro along the latter. A scale of degrees marked upon the semicircular arc enables us to read off directly the situation of the boundary of the visual field and the result obtained is transferred to a diagram (Fig. 121).

In Holth's portable *chord perimeter*, the metallic semicircle is replaced by a right-angled rule.

The perimeter or the confrontation method will show gross alterations in the field, and must be employed in any event when the field is of nearly normal extent. For very contracted fields and particularly for mapping out central or paracentral scotomata and the blind spot the perimeter has too small a radius.

For these purposes, as well as for accurately mapping hemianopic defects and indeed, for most purposes some form of the tangent plane is preferable (see also § 272).

It is important that the field should be taken thoroughly.

As Elliot points out, it is essential, in order to get a really accurate plot of the field that we should carry the test object in a series of concentric circles round the fixation point, noting in each circle all the defective points. Particular care must be taken to see if a defect stops short at the vertical meridian (hemianopsia; see § 275) or the horizontal meridian (Rönne's step, § 828).

268. Extent of the Field of Vision.—The normal field of vision, as a glance at the appended diagram (Fig. 121) shows, does not by any means extend equally far in all directions. It stretches farthest toward the external (temporal) side, where it has an extent of over 90°. Accordingly, we can still see objects on the temporal side, although they lie in, or even somewhat behind a plane passing through the pupil (for example, the point *O*

in Fig. 119). This is rendered possible by the fact that the rays from such a point undergo such strong refraction at the surface of the cornea that they can still enter the pupil. The field of vision is much less extensive in other directions, especially in directions inward and upward. One reason for this is the fact that the nose and the eyebrows project into the field of view and limit it. This obstacle can be partially overcome by directing the gaze downward and outward while the field is taken. Then a field somewhat larger than ordinary is delimited (*true* field—Mauthner). But even this is not as extensive on the nasal side as it is on the temporal. The cause of

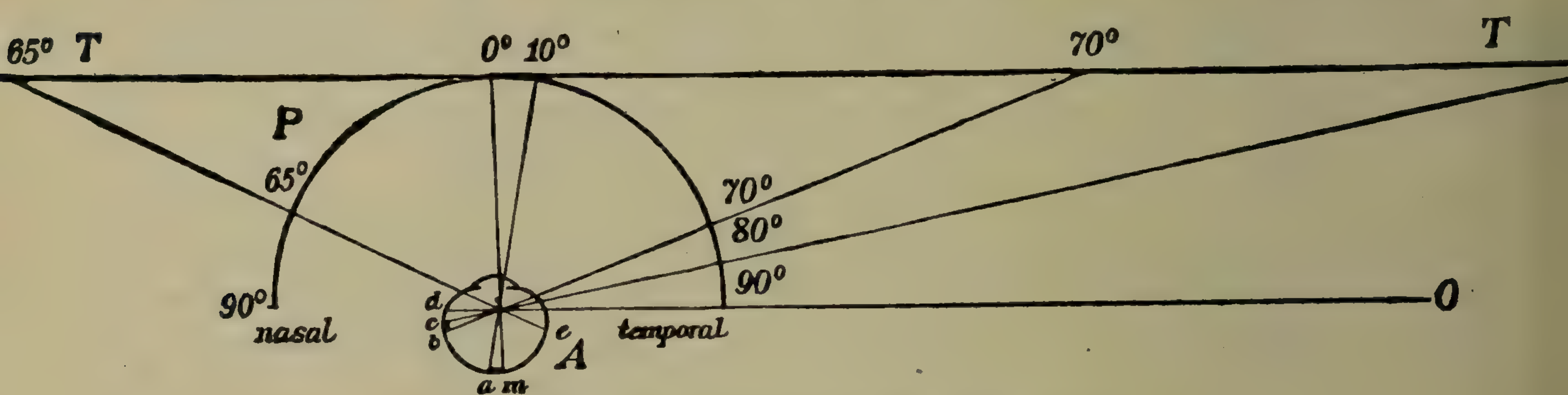

Fig. 119.—Projection of the Visual Field.

The field of vision of the eye, *A*, projected in the semicircle of the perimeter, *P*, extends from 65° on the nasal side to 90° on the temporal side, corresponding to the points, *e* and *d*, of the retina. These mark the anterior border of the sensitive portion of the retina, which extends farther forward on the nasal side than on the temporal. On a flat surface, *T T*, the field of vision cannot be represented as far as its temporal limit, since its projection, *O*, falls outside of the surface.

this is that the margin of the percipient layers of the retina does not extend as far forward on the temporal side as on the nasal (Fig. 119, *d* and *e*).

As Elliot points out, the blind spot is about in the centre of the visual field, indicating that the nerve head is at about the centre of the percipient part of the retina.

The field that we obtain in any given case, will vary according to the size and conspicuousness of the test-object, the background against which the object is projected, the character and intensity of the illumination, the adaptation of the patient's eye, and other circumstances. This is especially true of pathological cases, in which we shall often find that the field shows considerable variations from day to day without there being any actual change in their condition itself. In fact, the comparison between two perimetric records either of the same case or of different cases may be quite misleading unless we are sure that the conditions under which the two records were taken were identical. Hence a perimetric record is not complete unless the attendant circumstances and particularly the kind and size of the test-object used and its distance from the eye are noted. This notation is best made in the form of a fraction, the numerator of which indicates the size of the test-object, and the denominator its distance in mm.(Elliot and others). Thus $\frac{3}{330}$ would indicate a 3 mm. test-object used at the ordinary perimetric distance (330 mm.).

The extent of the field given by different-sized test-objects is shown in Figs. 121 and 122.

For purposes of description the field may be divided from within outward into (1) the *central area*, comprising, the fixation point and 1° or 2° about it; beyond this in succession, (2) the *paracentral zone* extending out to about 8° on the nasal and 12° in the temporal side, (3) the *cœcal zone* extending out to 25°, (4) the *intermediate zone* between 25° and 60°, and (5) the *peripheral zone* (beyond 60°). As the illustrations show, areas 1, 2, and 3 are delimited (and alterations in them made out) with the smallest sized test-

objects. On the other hand, for determining extreme peripheric limitations and the limits of the color fields (see § 269) the test-object must be large (5 to 10 mm. for the perimeter and 10 to 20 mm. for the tangent plane at 0.75 metre)—the size varying with the illumination and the patient's vision. For mapping hemianopsic defects and the form or color fields within 50° of the fixation point, the object may be 3 mm. for the perimeter and 9 mm. with the tangent plane at either 0.75 or 1.50 metres. Within 40° of the fixation point a better delimitation of the field and especially of relative scotomata (§ 271) and also a better delimitation of the blind spot can usually be had with smaller

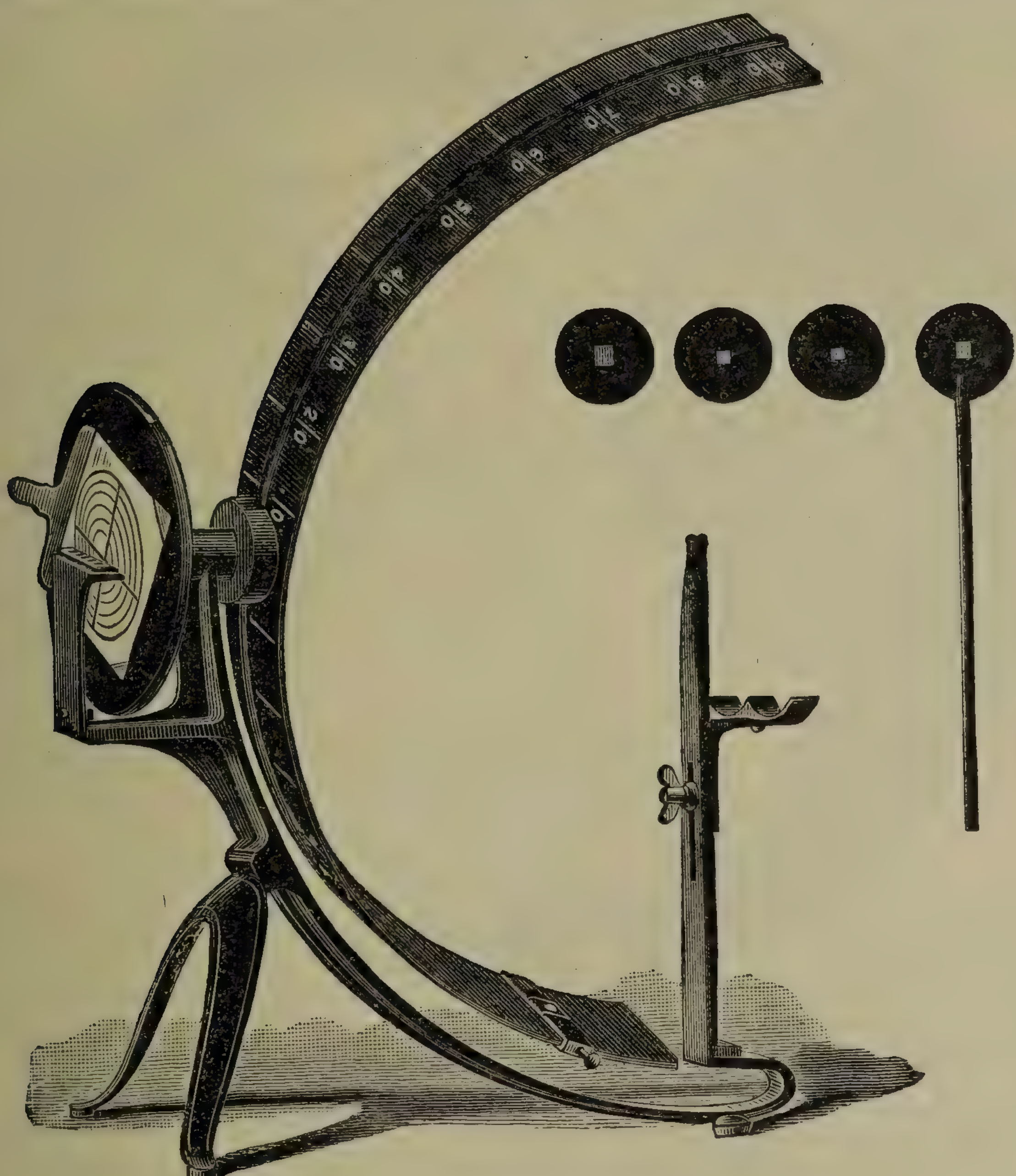

Fig. 120.—A Perimeter.

The perimeter consists essentially of a graduated arc along which the test-object is carried. This arc is capable of rotation through all meridians, the meridian which it occupies and the position of the test-object on the arc being marked upon the diagram, which, as shown on the left side of the cut, is attached to the instrument. The patient sits with his chin in the chin-rest sliding in the upright, shown on the right-hand side of the cut.

objects (1 or 2 mm. for the perimeter and 3 mm. for the tangent plane at 1.50 metres). These smaller objects, which should be supplemented by tests with the larger ones, will reveal defects which the others do not show and thus afford the same sort of information that is given by tests with colors (see § 269) and often in a more trustworthy fashion (Walker).

269. Color Fields.—Peripheral color perception is tested on a tangent plane or perimeter in the same way as the form field but with colored marks for test-objects. The bigger, more vivid, and better illuminated the colored surfaces used, the further toward the periphery will their color be distinguished, and they may even be distinguished up to the extreme limits

of the field. But when the examination is made with the ordinary test-objects used with the perimeter (colored squares of paper 3 to 10 mm. in diameter) the most peripheral portions of the retina are found to be color-

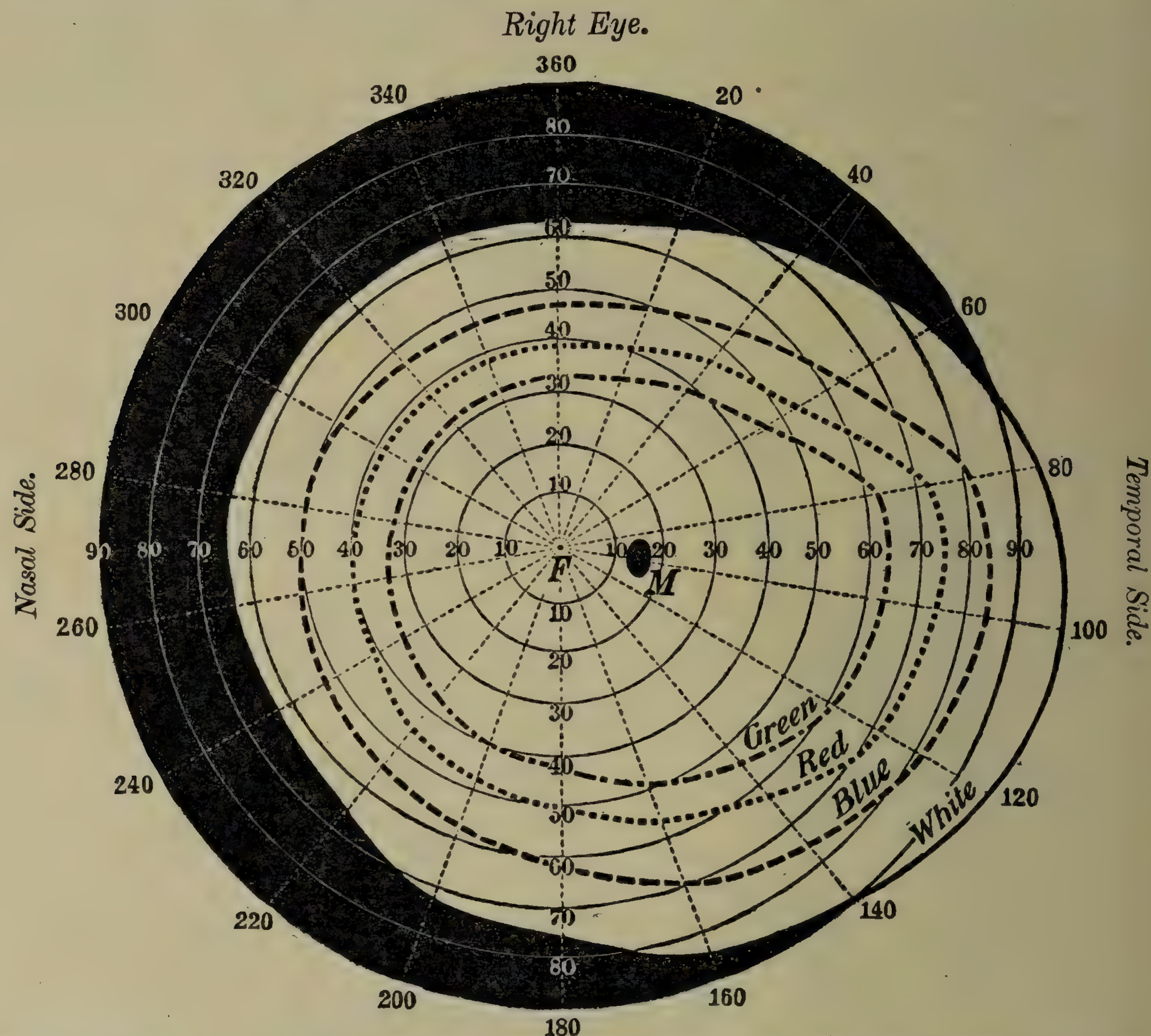

Fig. 121.—Field of Vision of the Right Eye for White, Blue, Red, and Green, for a Test object 20 mm. Square. (After Baas).

In normal eyes, the field for white is essentially the same with smaller (even 2 or 3 mm.) test-objects. The field for colors diminishes as the size of the test-object is reduced, and in any event varies so considerably with the tint of color used, the illumination, the adaptation of the eye, and other circumstances, and also varies so considerably in different subjects, that no standard limits can be indicated. The limits given in the illustration must be regarded as maximal, being in many cases 15° or 20° less. Particularly it is the case that the field for red is uncertain, owing to the fact that toward the periphery red changes to orange and then yellow. This zone of uncertain coloration is some 10° in width, and hence the patient's estimates of the limits of the field for red may vary by this amount. Again it not infrequently happens that the field for green equals or exceeds that for red, especially when spectral colors are used. These facts render tests of the color fields doubtful in any case and worthless unless the precise conditions of the test are noted. The field for any color, however, should in general be roughly concentric with the field for white, and any marked variation in this regard would be abnormal. With sufficiently large objects and sufficiently intense illumination, the limits of the color fields except for green are practically co-extensive with those for white.

blind. If such a colored square is pushed from the periphery of the visual field toward the centre, the person examined at first recognizes only the presence of a moving object. It is not until the square approaches nearer the centre of the visual field that its color is correctly given. The moment when this occurs is not the same for all colors, some being recognized farther out from the centre than others. Under ordinary conditions the visual field for green is the smallest, that for red somewhat larger, that for yellow still larger, that for blue the largest (see Fig. 121).

The limits of the color fields vary greatly with the illumination. The field for green is regularly the smallest, but according to circumstances the fields for red and blue may interlace, may be interchanged, or may even be equal to the field for white (Ferree and Rand.)

The examination with colors is a more delicate test than that with white; it betrays a diminution of the visual power before it has advanced so far that a white object can no longer be recognized. If, therefore, we take two cases in which the field for white is equally large, but the field for colors is unequal, that case in which the field for colors is smaller affords the worse prognosis, since here a still further diminution of the field for form is to be expected. Rapid diminution of color perception is pre-eminently associated with the progressive lesions of the optic nerve that lead to blindness. Tests with colors are also requisite for the recognition of central scotomata, so long as they are not absolute.

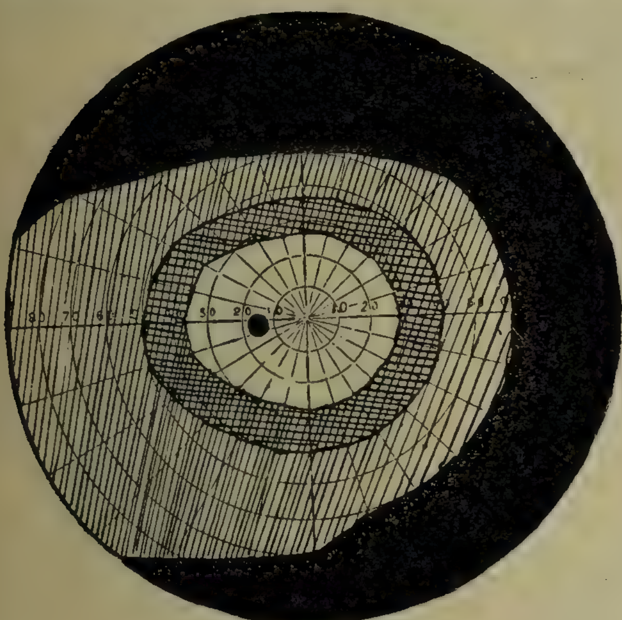

Fig. 122 A.

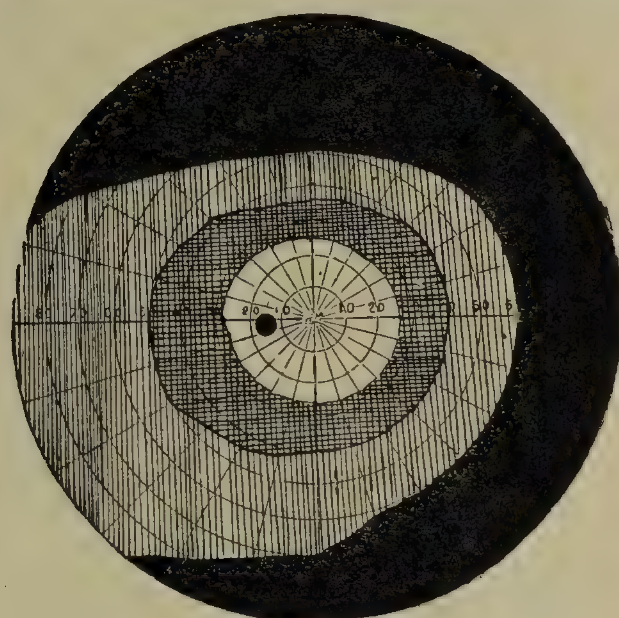

Fig. 122 B.

Visual Fields Obtained with Different-sized Test-Objects.

A (Elliot after Sinclair). From within outward fields obtained with $\frac{1}{2000}$, $\frac{6}{2000}$, and $\frac{10}{300}$ test-objects.
B (Elliot after Bjerrum). Fields obtained with $\frac{3}{2000}$, $\frac{6}{2000}$, and $\frac{10}{300}$ test-objects.

Furthermore, the way in which the color sense is diminished gives us a clew as to the site of the morbid changes. Thus diminution in the perception of blue corresponds to a lesion of the percipient elements (rods and cones) of the retina, such as occurs in chorioiditis, retinitis, detachment of the retina and hemeralopia; diminution in the perception of red and green to a lesion of the conducting elements, as in affections in the optic nerve. If, however, the vision is impaired simply by opacities in the media, the perception of colors is normal. (Cf. analogous differences in light sense tests, § 260).

It must be admitted that, owing to the difficulty of standardizing the color tests, the results obtained by them are often unsatisfactory and that better information as to the presence of relative scotomata or a relative obscuration at the periphery is afforded by minute white objects (see § 268 and remarks under Fig. 121).

270. Scotomata; Normal Blind Spot.—Island-like gaps in the visual field are called *scotomata.* [14] One of these exists in the healthy eye at that point in the visual field which corresponds to the entrance of the optic nerve, and is known as Mariotte's blind spot. In the field of vision it lies 15° to 16° to the outside of the point of fixation (see Fig. 121 *M*).

It varies in size and shape, but has usually the form of an erect oval, between 5° to 6° broad and 7° to 8° high (thus corresponding to the dimensions of the optic disc; see § 403 and Fig. 123). This area of complete invisibility is surrounded by a rim ¼°—¾° wide, of relative obscuration (penumbra) in which large objects are still well seen, but small objects and colored objects are made out dimly or not at all (Haycraft, Van der Hoeve). Any marked extension of this penumbra or any notable enlargement of the

[14] From σκότος, darkness.

blind spot indicates disease and particularly chronic glaucoma or degenerative or inflammatory changes in the optic nerve. This enlargement may be the only or at least the determining diagnostic sign present in these conditions. Hence in suspected cases the blind spot should be carefully plotted (see § 272).

271. Pathological Scotomata.—The scotomata which occur as the result of disease have a very different significance for vision according to their

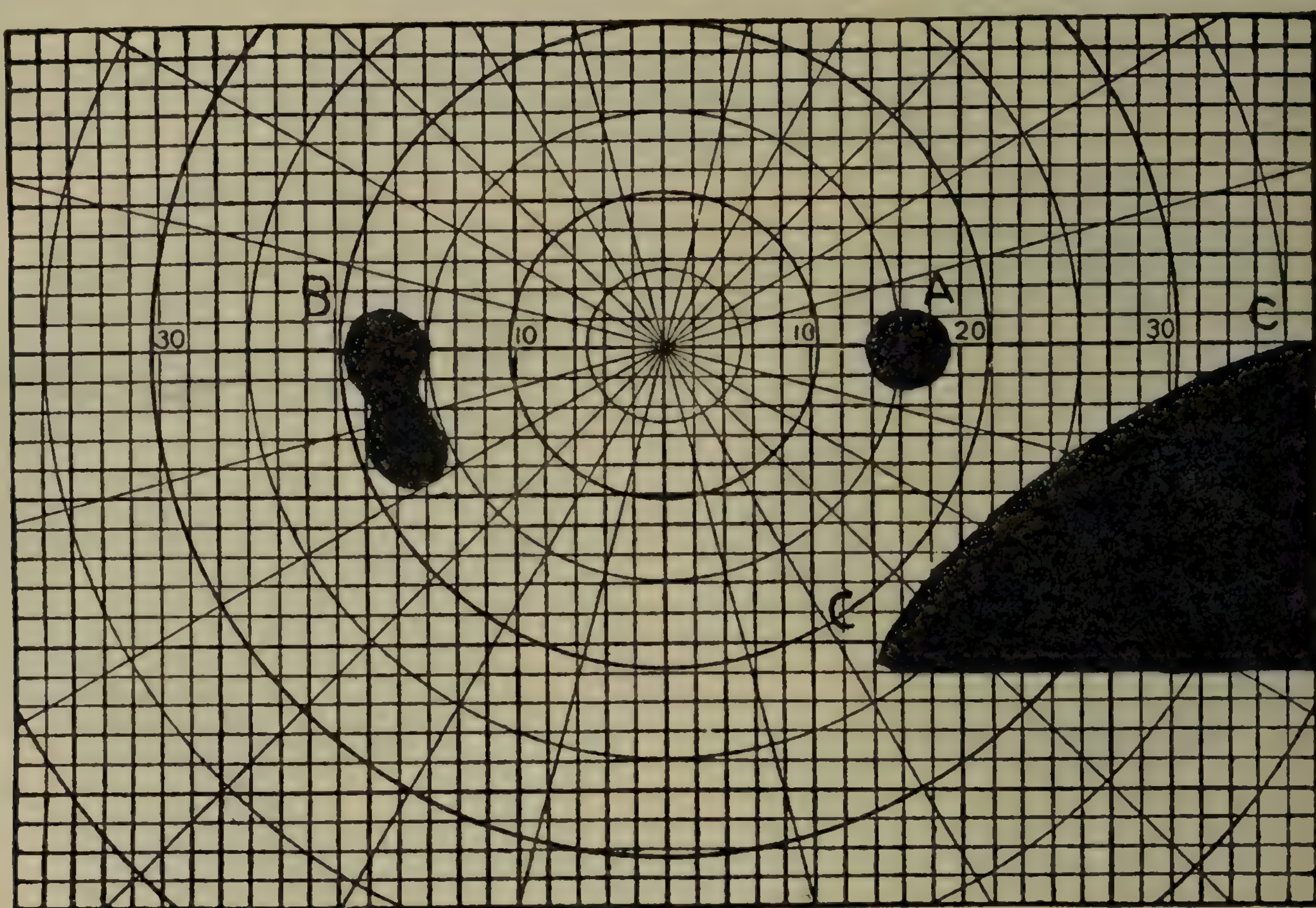

Fig. 123.—The Blind Spot.

A, blind spot (of normal size) of right eye. It differs from the usual form in being horizontally instead of vertically oval. B, blind spot (enlarged) of left eye. C, C, area of limitation of nasal field in left eye. Taken from a case of glaucoma in the translator's practice. Field plotted on Bjerrum curtain. Figure shows card used for making plot when patient is placed 1.50 metres from curtain.

situation; and, according to the latter, we distinguish them into central and peripheral. A *central* scotoma is one which involves the point of fixation (cf. Fig. 368). In this case direct vision is either greatly diminished or is abrogated altogether. The patient can no longer do any fine work, although his power of orientation remains intact. *Peripheral* scotomata cause little disturbance of sight, especially if they lie far from the point of fixation, in which case they may not come to the patient's knowledge until his visual field is being examined.

A special variety of scotoma is the *annular*, which encircles the point of fixation like a ring (which is not always completely closed), but leaves intact the point of fixation itself.

Scotomata are most frequently met with in diseases of the fundus with focal lesions; especially, therefore, in chorioiditis disseminata, in which, as a rule, the gaps in the visual field correspond to the separate spots visible with the ophthalmoscope. So long as these gaps affect the periphery only of the visual field, they cause little disturbance of sight. If they are very numerous, the visual field acquires a sieve-like character.

Isolated *central scotomata* occur in diseases of the retina and chorioid at the posterior pole of the eye, especially as a result of syphilis, or myopia of a high degree, or of senile changes. In all these cases there corresponds to the scotoma a change in the region

of the macula lutea, visible with the ophthalmoscope. In another series of cases, on the contrary, a central scotoma exists, but the macula on ophthalmoscopic examination looks normal; the cause of the scotoma is then to be looked for in the optic nerve. In the latter it is just those fibres which supply the region of the macula lutea that are the most favorite seat of disease (in retrobulbar neuritis and in toxic amblyopia, glaucoma simplex, etc.). Normal eyes will develop a relative central scotoma if placed in a sufficiently dark room. If the room is darkened still more, the scotoma becomes absolute—under-sensitiveness of the fovea to light in eyes that are adapted to the dark (Hess).

A central scotoma, even when so minute as to be demonstrable with difficulty, usually causes much trouble in near work. A person with such a scotoma may have comparatively good vision (20/40) for distance, and yet may be utterly unable to read even large type; while a person with similar vision but no central scotoma can read quite well. A person with central scotoma reads letters in a characteristically hesitating way, picking out the letters slowly and often missing a letter altogether.

Scotomata in the *cæcal zone* (§ 268) are characteristic of glaucoma (Bjerrum's and Seidel's signs, § 828) and are also found in diseases of the accessory nasal sinuses. An annular scotoma in this zone is found in the earlier stages of retinitis pigmentosa (§ 792).

Defects of the *intermediate zone* in the nasal field are found often in glaucoma. Such defects often end in a sharp edge right in the horizontal meridian (§ 828).

Scotomata are divided into positive and negative (Förster). A *positive scotoma* is a dark spot which the patient perceives in his visual field—projects, that is, upon some portion of his field. Scotomata of this kind are best brought to light by making the patient fix his gaze upon a uniformly bright surface (e. g., a sheet of white paper). They are often more readily perceived if the illumination is at the same time diminished. A positive scotoma may originate in a lesion, e. g., an exudate or a hæmorrhage, in the retina. Such lesions throw a shadow on the rods and cones, as these are the furthest back of all the layers of the retina, and this shadow is perceived and is projected exteriorly. Opacities in the refracting media also throw their shadow upon the retina, and are therefore visible as dark spots. If the opacities lie in the vitreous they are motile (muscæ volitantes) and the scotomata caused by them are characterized as motile scotomata. (It is better not to use the expression scotoma for cases of this sort.)

A *negative scotoma* is a hiatus in the visual field, an isolated spot within the confines of which the patient fails to perceive external objects. Such a scotoma is usually not discovered until the visual field is examined.

Scotomata are also divided into *absolute* and *relative* according as the object used in examining the field of vision becomes altogether invisible or simply indistinct in the confines of the scotoma. Hence an absolute scotoma for white is present if a white object disappears entirely at one spot, but there is a relative scotoma for white which at that point simply appears of a less pure white, or indistinct in outline. But even when a white object shows no change whatever, this does not prove that there is no change in the field. We must then make the examination with colored objects also. For with a certain diminution of the visual power the ability to distinguish colors accurately disappears, while the objects themselves, owing to differences in their luminosity, can still be recognized For example, in a recent case of nicotine poisoning the field, measured with a white test object, may seem quite normal; but if a small red paper disc is used there is a small region in the centre in which the disc is either not recognized as red at all (absolute scotoma for red) or is less vivid there (relative scotoma for red).

272. Mapping of Scotomata.—Scotomata (including the blind-spot) are best mapped out on a tangent plane, preferably at a distance of 1, 1.5, or 2 metres

In Elliot's *scotometer* the blind spot is mapped on a large curved surface.

Scotomata, especially central scotomata, are often hard to delimit, because the patient, on account of his poor central vision, fails to see distinctly the spot which he is asked to fixate, and hence lets his eye wander about, so as to project his scotoma on successive portions of the plane. To obviate this, we employ some device by which the

other eye, presumably sound, maintains fixation and yet does not see the test-object. This may be effected by a special stereoscope (*stereo-campimeter*) with special stereoscopic charts (Haitz) or a graduated slate (Bissell, Lloyd). This instrument is also excellent for mapping the blind spot. Another good means of mapping either the blind spot or a unilateral central scotoma is the *extinction test*. In this, if we are examining the right eye we place a red glass before the left and direct the patient to look with both eyes at a black pin in the centre of the white side of the Bjerrum curtain (Fig. 118). As a test-object, a red disc is used. To the left eye, viewing it through the red glass this appears white and therefore invisible against the white background. The right eye sees it as a red spot until it enters the limits of the scotoma when it suddenly fades or disappears.

When the fellow eye cannot be used for maintaining fixation, this may be secured by placing a sufficiently large white ring round the fixation point and directing the patient to look at its centre (Marx). Or, the patient may place his finger on the fixation point and be directed to look at it, his muscle sense enabling him to maintain direction (Peter).

An accurate method of delimiting scotomata, and especially the blind spot is to use *two test-objects* which are carried from opposite sides towards the limits of the scotoma, then brought within the latter, and then separated until both are just simultaneously visible. This fixes one diameter of the scotoma, and by repeating the test in different diameters, a fairly accurate plot may be made. The result may be checked by putting in a series of white pins round the periphery of the scotoma as found. If when the eye is properly directed these all disappear at once, the plot may be regarded as accurate.

273. Peripheral Contractions.—The peripheral field may be narrowed from all points of its circumference at once. This is called a concentric contraction. When this is considerable, it causes, as we have seen, an incapacity for orientation even if central vision is still quite good. In other cases, the contraction extends from only one side of the periphery into the field. If it has the shape of a triangle whose base corresponds to the periphery of the field, it is called a sector-shaped contraction.

Concentric contraction of the field, associated with retention of good central vision, we meet with especially in retinitis pigmentosa, and sometimes also in glaucoma. In other diseases which are frequently accompanied by concentric contraction of the field, as, for instance, in atrophy of the optic nerve or of the retina and diffuse chorio-retinitis, central vision is also simultaneously and markedly affected. Concentric contraction, often of high degree is found in hysteria and neurasthenia (see § 274).

We find the *sector-shaped deficiencies* especially in atrophy of the optic nerve; also in occlusion of one of the larger retinal arteries, when the sector-shaped district of the retina supplied by such an artery has its function abrogated. We observe more extensive, although not triangular, contractions in detachment of the retina, and these most often extending in an upward direction, since the detachment, if of long standing, generally involves the lowermost part of the eye. In glaucoma a contraction of the field toward the nasal side, often sector-like or annular and continuous with the blind spot, is relatively frequent (see § 828). The contraction here regularly begins not at the periphery but at the blind spot.

274. The Field in Hysteria and Neurasthenia.—In hysteria and neurasthenia, the field sometimes shows a concentric contraction (Fig. 125) and in both, but in neurasthenia particularly, it often gets smaller and smaller, the longer the patient is tested (exhaustion field). The contraction may be associated with amblyopia (§ 245).

In the case of an exhaustion field, if we test each meridian of the field in succession and repeat the examination on reaching the meridian first tested, the progressive shrink-

ing of the limits in each successive meridian may make the field assume a spiral form (Fig. 124). Other peculiar modifications of the field found in hysteria, beside the contracted field mentioned above are: (*a*) The *tubular* field. The field is greatly contracted and remains of the same absolute size, no matter how far the patient is removed from the point of fixation. (*b*) The *oscillating* field, in which there are a series of scotomata in each meridian (see Fig. 126). This seems very rare. (*c*) The *reversed* or *interlacing* field, in which the field for green or red is larger than that for blue or crosses it (see Fig. 125).

275. Hemianopsia.[15]—Hemianopsia is the condition in which one half of the field in each eye is abolished. It is denoted according to the situation

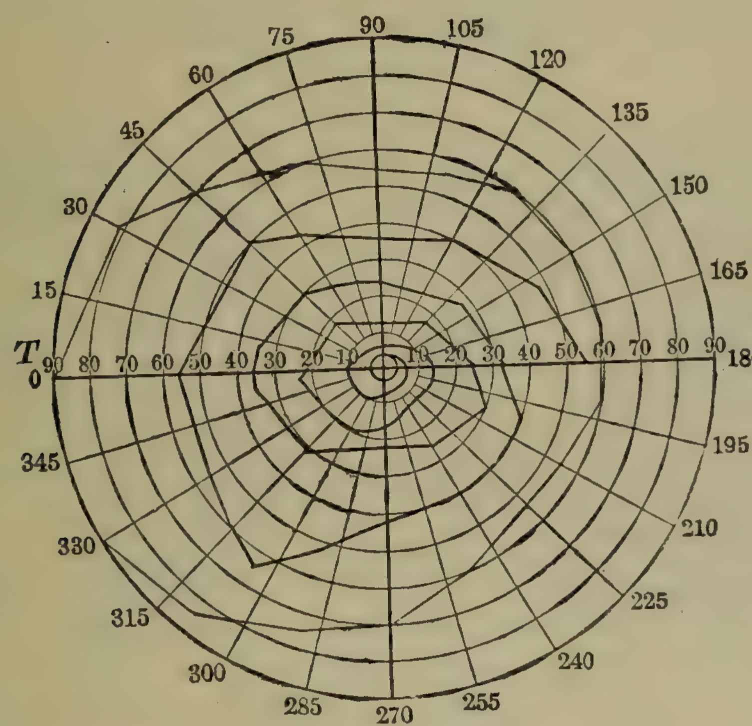

FIG. 124.—SPIRAL FATIGUE FIELD OF VON REUSS. (After Weeks.)

of the defect in the field. Thus the defect may occupy the right or left half of the field in each eye (*homonymous* hemianopsia, Fig. 127); the temporal half in each eye (*bitemporal* hemianopsia, Fig. 128); the nasal half in each eye (*binasal* hemianopsia); or the upper or lower half in each eye (*altitudinal* hemianopsia). Homonymous hemianopsia is further designated as right-sided or left-sided, according as it is the right or left half of each field that is abolished.

Hemianopsia in the more extended sense of the term exists not only when an entire half of each visual field is wanting, but also when there is a deficiency which, though smaller, occupies a symmetrical position in the visual fields of the two eyes (*incomplete hemianopsia*, Wilbrand). Sometimes the deficiency is limited to a symmetrical *quadrant*, e. g., the left upper quadrant, in each eye. In homonymous hemianopsia it is the rule that the field of vision is not divided exactly in half, the vertical border of the field bending out a little at the site of the point of fixation (Fig. 127), so that the portion of the field of vision corresponding to the macula lutea is preserved intact (*exemption of the macula*). Hence, if, as is very rarely the case, a bilateral hemianopsia is produced by a bilateral central lesion, the combination of the visual defects on the two sides does not give complete blindness, but usually leaves intact right in the centre a very small central visual field, representing the fovea centralis.

Hemianopsia may be limited to a loss of color perception in one half the field (*hemiachromatopsia*).

[15] From ἥμισυς, half, ἀ, privative, and ὤψ, sight. Also called *hemianopia and hemiopia*. The latter meaning "half sight" seems less applicable than hemianopia ("half blindness"), since the term is always applied to the blind half ot the field of vision.

276. Diagnostic Significance of Bitemporal Hemianopsia; Pituitary Disease.—The facts regarding the course of the optic-nerve fibres (§ 233 and Fig. 110) enable us to determine the site of the lesion causing a visual disturbance which ophthalmoscopic examination shows is not due to disease in the eye itself. Objects in the right visual field (G, Fig. 110) are perceived by the left half (l, l_1,) and objects in the left field (G_1) are perceived by the right half (r, r_1,) of the retina in each eye. Hence in *bitemporal hemianopsia*, in which G is lost for the right eye and G_1 for the left, the nasal half

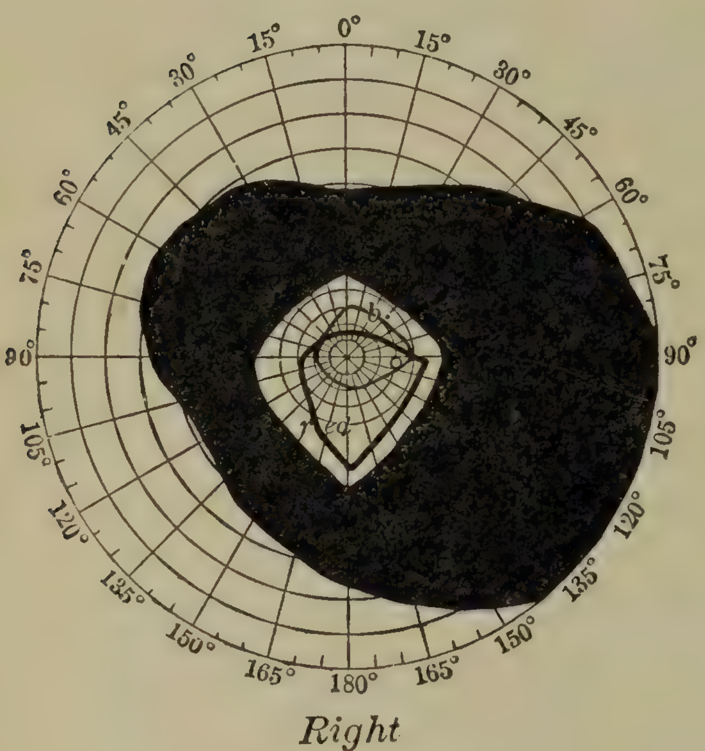

FIG. 125.

FIG. 126.

THE VISUAL FIELD IN HYSTERIA. (After De Schweinitz in Posey and Spiller.)
FIG. 125.—Contracted field (in this case a tubular field) with partial reversal of color fields. *b*, field for blue. *r*, field for red.
FIG. 126.—Contracted and oscillating field (Wilbrand). The dark lines denote scotomata.

of each retina (l_1, r) is out of function. If the retina itself is intact so that the lesion lies back of the eye it must lie in the decussating bundles, which supply l_1, and r. Such a lesion, if single, must occupy the middle of the chiasm or its anterior or posterior angle (at s s, Fig. 110).

Such a lesion is usually the result of an enlargement of the pituitary body (hypophysis), which, therefore, is often first discovered by the ophthalmologist. The symptoms vary, depending on whether the degeneration affects the glandular or the nervous portion of the hypophysis, whether it causes increase or diminution of function, and finally whether it begins in the growing period or not till later in life. When it occurs during the growing period, there develop disturbances of growth, viz., either gigantism or more rarely dwarfing of growth, general adiposity and defective development of the sexual parts or of the hair (beard, pubic hair). When the disease develops later in life, the disturbance of growth manifests itself by an enlargement of the skeleton and the soft parts of the face, hands and feet (acromegaly), impotence and in women amenorrhœa, chilliness, somnolence, and polyuria. The eye symptoms are slow reduction of central vision, a progressive, gradual contraction of the field usually on the temporal side, and a peculiar waxy pallor of the disc, unassociated with alteration of the large vessels or with excavation. This pallor sets in some three or four months after the visual symptoms and is not an evidence of degeneration of the nerve—true atrophy not occurring until later (Benedict). Optic neuritis rarely occurs. Occasionally homonymous hemianop-

sia is found or less extensive, though still hemianopic defects in the field. The x-ray shows dilatation of the sella by the enlarged hypophysis. Treatment consists in operation or the use of glandular extracts, supplemented advantageously with mercury and the iodides (De Schweinitz). A temporary bitemporal hemianopsia, often only partial, seems to occur frequently in pregnancy (Finlay) and is attributable to the physiological enlargement of the pituitary body regularly found in this condition.

277. Conditions Causing Homonymous Hemianopsia.—Homonymous hemianopsia may in rare cases be due to chiasm lesion (pituitary disease—see § 276). But usually it argues a lesion above the chiasm. Thus if there is a right-sided hemianopsia so that the visual half-field *G* (Fig. 110) is cut off for both eyes, the left halves of the retinæ, *l*, l_1, are both out of func-

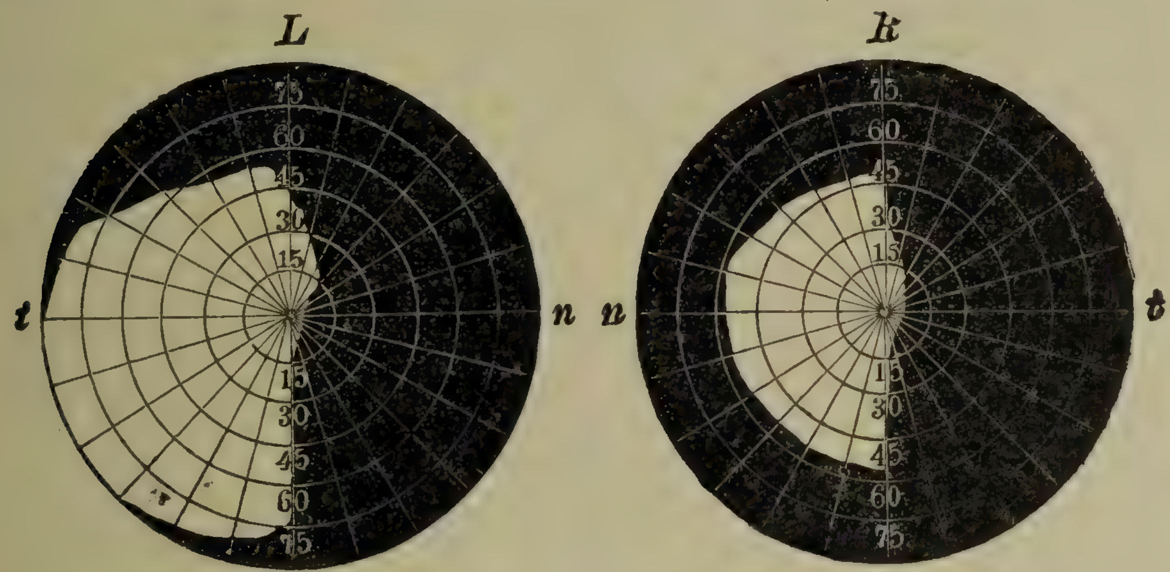

FIG. 127.—HOMONYMOUS HEMIANOPSIA. (After Schweigger.)
The areas which have been left white correspond to the left halves of the visual fields, *R* and *L*, of the right eye and left eye, which are still intact; *t*, temporal; *n*, nasal side.

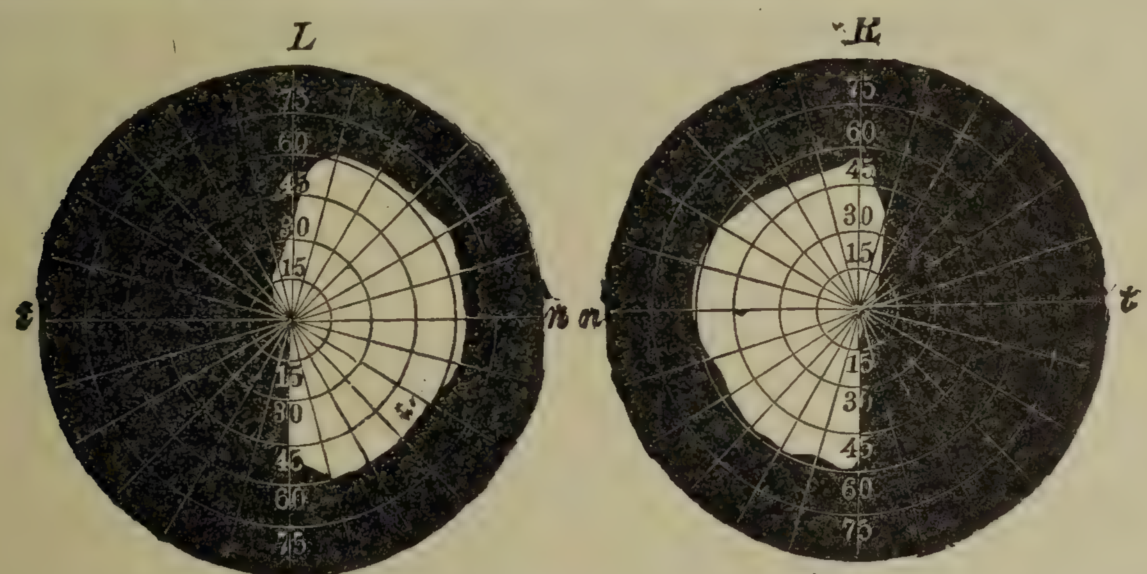

FIG. 128.—TEMPORAL HEMIANOPSIA. (After Schweigger.)
The areas left white correspond to the nasal halves of the visual fields, *R* and *L*, of the right and left eye, which are still intact; *t*, temporal; *n*, nasal side.

tion, in which case the lesion, if not in the retina itself, must affect the non-decussating bundle of the left eye and the decussating bundle, of the right. Such a lesion in general will be caused by a lesion of the left optic tract (e. g. at *gg*) or in the fibres ascending from this to the left cortex (at *ee*) or in the left cortex itself near the calcarine fissure (*B*)

In the case of *partial* hemianopsic defects the lesion has the same situation but instead of affecting all, affects only a part of the fibres running from the chiasm to the cortex on one side or a very limited part of the visual cortex itself. Such partial defects

may be limited to a central or paracentral scotoma, symmetrically placed in the visual field of each eye; and such defects may be central—even cortical—in origin (Wilbrand). A condition causing an homonymous quadrant defect (see § 275), if cortical in site is limited to one lip of the calcarine fissure (in the upper lip if a pair of the lower quadrants, and in the lower lip if a pair of the upper quadrants of the field are obliterated).

Since the pupillary fibres (*m*) in the left tract do not leave the latter until just before the visual fibres ascend to the cortex, theoretically a tract lesion causing hemianopsia (hemianopsia anterior) should be associated with inaction of the pupils when the left half of either pupil receives the light stimulus (Wernicks's hemianopic pupillary reaction or inaction, hemikinesis). If the lesion causing hemianopsia is above the tract (hemianopsia posterior), hemikinesis should not occur. As a matter of fact hemikinesis has been observed in posterior hemianopsia and may be absent in anterior hemianopsia. Moreover, as it is hard to demonstrate in any case on account of the difficulty of limiting the light to the affected area of the retina and as it may be produced in normal eyes by lowering the adaptation of the retina, it has very little diagnostic significance.

278 **Lesions Corresponding to Binasal Hemianopsia and Asymmetrical Defects.**—The lesions causing conditions other than bitemporal and homonymous hemianopsia are as follows:

Binasal Hemianopsia. This very rare condition is due to lesions affecting both lateral aspects of the chiasm.

Unilateral and Asymmetrical Defects. In all cases in which the defect is restricted to one visual field or in which, while there are defects in both visual fields they are not symmetrically situated, the lesion must be in the *optic nerve* itself or in the retina, that is, in front of the chiasm—since all interruptions back of the chiasm result in the production of symmetrical defects in the fields. For the same reason complete blindness of one eye, with retention of good sight in the other, must be referred to an affection in front of the chiasm. And finally it must be remembered that even symmetrical visual defects are not necessarily due to lesions behind the chiasm, but may be caused by symmetrical lesions in the two optic nerves or in both retinæ. *Central scotomata* correspond to an affection of the papillo-macular bundle. Central scotomata are sometimes caused by a lesion in the region of the macula so inconspicuous as to escape ophthalmoscopic observation. In such cases metamorphopsia (see § 238) may be present; a fact which would be conclusive evidence that the trouble was seated in the retina or chorioid and not in the optic nerve.

CHAPTER XVII.

MOTOR ANOMALIES OF THE EYE.[1]

I. Fixation and its Derangements.

279. Monocular Fixation.—When we direct one of our eyes towards an object so as to see it as distinctly as possible we are said to *fixate* or *fix* that object.

The fovea centralis is the most sensitive portion of the retina. Hence, in performing fixation we normally direct the eye so that the image of the object looked at falls upon the fovea. This is called *central fixation*. It may happen, however, that owing to disease of the retina or to other reasons vision with the fovea is less acute than with some outlying portion of the retina, and in that case the eye, in monocular fixation at least, will be directed so that the image shall fall upon this outlying portion (*eccentric fixation*).

Cases have been reported in which the outlying portion used to fix with so surpassed the remaining portions of the retina in sensitivity as to entitle it to be called an *eccentric fovea*. Such cases, are certainly extremely rare. Generally in eccentric fixation, the vision is very poor and no single spot of the retina is uniformly employed to fix with, so that fixation is not only eccentric, but uncertain and wandering.

280. Monocular Projection.—When we are looking with one eye at an object and that object forms its image on the retina, we not only see the object, but get an idea that it occupies a certain definite position in space. This process by which we infer the position of an object in space from the visual impression that the object produces we call *projection*.

An object towards which the eye is pointing forms its image on the fovea. Conversely, an object which thus forms its image on the fovea we judge to be situated in that part of space towards which we think the eye is pointing. If our judgments are correct as to the direction of our eye, we project—i. e., locate—objects correctly; but if, as in paralysis, our notions of the positions of the eye are erroneous, our projection will be faulty and we will see objects in the wrong place.

Not only do we get an idea of the location of an object, A (Fig. 129), towards which the eye is pointing, but also a notion, more or less precise, of the location of all objects surrounding A. Thus, an object, B, that forms its image below the fovea, we regard as situated in space correspondingly far above A, as, objects that are actually above the point we are looking at do regularly, in our experience, form their image below the fovea. So also the object, C (Fig. 130), which forms its image to the left of the fovea, we judge to be situated correspondingly far to the right of A.

[1] This chapter has been entirely rewritten by the translator, who therefore is responsible for the statements contained in it.

We thus arrive at the *law of projection,* which may be stated thus: *An object which forms its image on any point in the retina is projected to—i. e., mentally located in—the point in space directly opposite.*

By virtue of projection we see the objects in the outer world arranged side by side just as their images are upon our retina, only in reverse order; whatever forms an image to the right of the point of fixation, is seen upon the left of the latter, etc. We are thus informed with certainty in regard to the

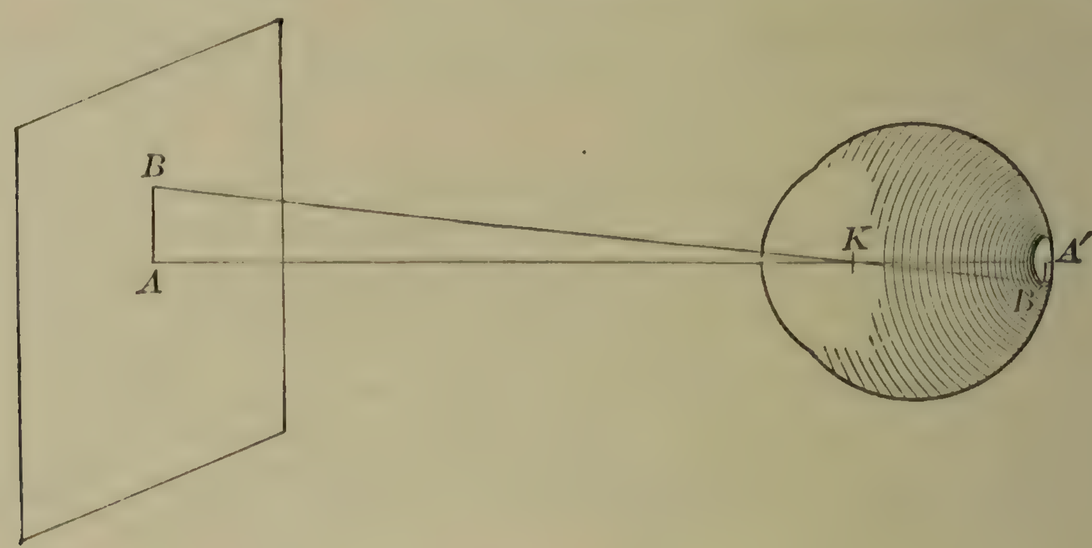

FIG. 129.—(After Posey and Spiller).

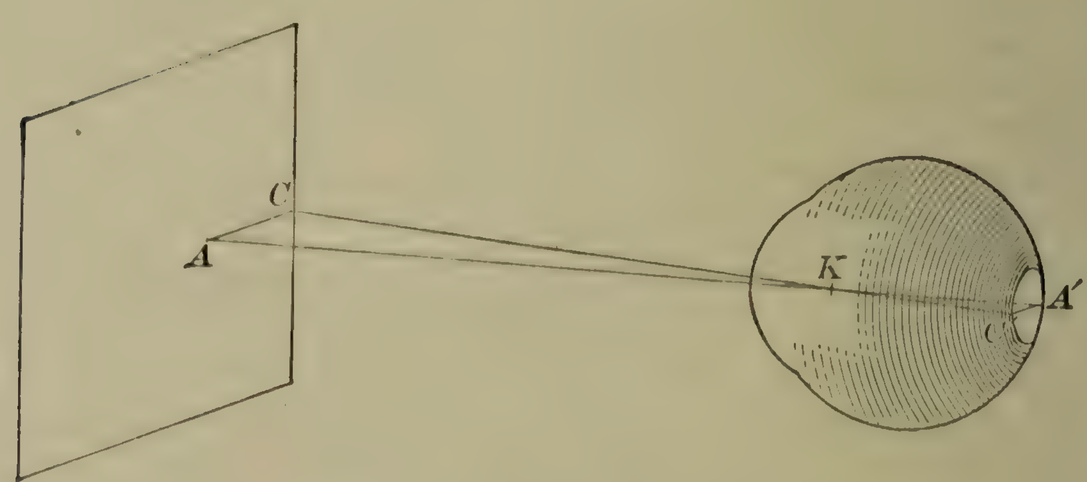

FIG. 130.—(After Posey and Spiller).

position of objects relative to each other (*objective orientation*). But for perfect orientation it is further requisite that we should assign to its correct situation in space the whole mosaic of images that we project from our retina into the outside world, and which is already properly constructed as far as the relation of its own parts to each other is concerned. Not till we do this can we have any conception, corresponding to the real state of things; a perception, that is, of the position of objects not only with reference to each other but also with reference to our own body (*subjective orientation*). Such subjective orientation depends upon our having a knowledge of the position of our own body in space, and of the position which the eyes occupy in our body. The former is accomplished by virtue of the sense of equilibrium, the latter by the muscular sensations which originate in the ocular muscles, and which inform us how our eyes are directed with relation to our

body. By means of subjective and objective orientation together, we are able to recognize correctly the absolute position in space of any object that we see.

As above indicated projection is a complex process. An object on our right forms an image which falls on the left side of the retina in either eye (really on the left brain-terminal; see § 234 and Fig. 112), and from there is conveyed to the left occipital cortex where it probably stimulates some very circumscribed area close to the calcarine fissure. This excitation produces in consciousness the idea of a body situated in some definite situation on the right. That is, the retinal image is projected to a certain definite area of the brain, and from here is re-projected into space to the site of the object that originated it. Our notions, however, of the absolute and relative position in space of this object are defined as above stated, not only by the position of the retinal image but by several other factors, notably, the *muscular sense*. The importance of this is shown in the following experiment (Sherrington). If we look at three vertically placed dots straight ahead, we get an idea that (1) they are of a certain size and shape, (2) are a certain distance apart, (3) are in a straight line, which is (4) vertical and (5) straight ahead. Of these five sensations, 1, 2, and 3, are undoubtedly attributable to the position occupied on the retina by the images of the dots, which images are re-projected into space according to the law of projection above given. But it is otherwise with 4 and 5. If we shift the gaze to a similar series of dots placed up and to the right these dots will still appear vertical although it can be proved that the eye in the meantime has rotated on its antero-posterior axis (see § 295) so that the images of the new line of dots now lie more or less athwart the vertical meridian of the retina and, according to the law of projection, the line itself should appear tilted. That nevertheless it appears vertical must be due to some second impression correcting the impression derived from the retinal images. This second impression, as Sherrington points out, must originate in the muscular sensations that are produced by turning the eyes. This second impression also accounts for the fact that the new object, although it forms its image on the fovea is no longer regarded as straight ahead but as situated up and to the right. These muscular sensations are conveyed by what is called the *proprioceptive* nerves of the orbit. When instead of moving the eye itself, we move the head or body to direct the eye at an object, notions as to the latter's position are afforded by impressions derived from the proprioceptive nerves of the body muscles and especially from the *labyrinth*.

281. Binocular Fixation.—In the vast majority of cases, if one eye fixates an object with its fovea the other will do so too, (binocular fixation). In fact, the whole extremely complicated mechanism of muscles and nerves with which the two eyes are supplied is designed primarily to accomplish this single end—i. e., to so move the eyes that they shall both be directed accurately to the object we wish to see and each eye shall receive the image of that object precisely upon the fovea. The movements of the two eyes are hence almost invariably so co-ordinated as to secure binocular fixation under all conditions.

282. Derangements of Binocular Fixation; Deviations of the Eye.—In the majority of cases both eyes will fixate the same object—i. e., both will be "straight" not only when both eyes are open, but even when one eye is covered. This condition is called *orthophoria*[2], and must be regarded as the ideal or natural condition.

In other instances both eyes will look straight at the same object when both are uncovered, but either eye as soon as it is covered will deviate—

[2] From, ὄρθος, straight, and φορεῖν, to bear constantly.

turning out, in, up, or down. This condition is called *heterophoria*[3], or less properly *insufficiency*.

In a third class of cases, comparatively few in number, only one eye is straight at a time, the other deviating even when both eyes are open. This condition is called *squint, strabismus*[4] or *heterotropia*[5].

A deviation, whether squint or heterophoria is called—

1. *Constant*, if present all the time; *intermittent*, if under the same conditions of vision it is sometimes present, sometimes not.

2. *Continuous*, if present to about an equal degree in both distant and near vision; *periodic*, if very much greater for near objects than for distance or vice versa. (In the former case it is said to be *directly*, in the latter *inversely* periodic.

3. *Comitant* (or *concomitant*) if the amount of deviation remains the same, and *non-comitant* if it changes notably, as the eyes are carried in different directions.

A non-comitant deviation is usually of paralytic origin (*paralytic squint* or *heterophoria*); sometimes is spastic. A comitant deviation is not usually due to paralysis, at least paralysis of the individual muscles, although it may develop out of such a paralysis.

Deviations are also named according to the *direction* of the deviating eye. Thus—

A. With both eyes open. Right or left eye deviates—

In.	Esotropia (Convergent squint, Strabismus convergens).
Out.	Exotropia (Divergent squint, Strabismus divergens).
Up.	Hypertropia (Strabismus sursum vergens).
Down.	Hypotropia (Strabismus deorsum vergens).
Not at all.	Heterophoria.

B. If one eye is covered, covered eye deviates—

	Heterophoria	Heterotropia
In.	Esophoria	Esotropia.
Out.	Exophoria	Exotropia.
Right up, left down[5].	Right hyperphoria.	Right hypertropia, left hypotropia.
Right down, left up[6].	Left hyperphoria	Left hypertropia, right hypotropia.

Esophoria, exophoria, esotropia, and exotropia are also known as *lateral* deviations (heterophoria, squint); hyperphoria, hypertropia, and hypotropia as *vertical* deviations.

A condition in which the vertical meridian of one eye deviates from the vertical position is called a *cyclophoria*, if the deviation occurs only when the eye is screened and a *cyclotropia* if it is present when *both* eyes are open.

Mixed conditions are common. Common combinations are (a) lateral deviation associated with a vertical; (b) a squint for near with orthophoria or heterophoria for distance, or vice versa; (c) a squint, vertical or lateral, in one direction of the gaze, with no deviation or any entirely different kind of deviation in another.

Finally, a squint is called *monocular* if, with both eyes open, it always

[3] From, ἕτερος, other, and φορεῖν, to bear constantly.
[4] From στρέφειν, to turn.
[5] From ἕτερος, other, and τρόπος, a turning.
[5-6] In some rare cases both eyes deviate down or both up behind the screen (double hypotropia or hypertropia).

affects the same eye, and is then called right or left, according to the eye affected; and it is called *alternating*, if with both eyes open, sometimes one eye, sometimes the other deviates.

Of *alternating squint* three varieties are distinguished. In the first, both eyes have good vision but unequal refraction. For instance, one eye is far-sighted (hyperopic or emmetropic) and the other near-sighted, so that the former sees clearly at a distance, the other near by, but both eyes can never see distinctly at once. In this case the far-sighted eye will fixate in looking at a distance, the near-sighted eye when looking near by (periodic alternation), and in either event the eye not used squints; the image in this eye not being distinct, its suppression is readily accomplished. Alternating strabismus of this sort is usually divergent, rarely convergent. Another kind of alternating squint is found exclusively in esotropia. It consists in each eye's being used for that half of the common field of fixation which is on the opposite side from it. An object which is carried from the patient's left to his right in front of the eyes is first fixed by the right eye, while the left eye squints past the object to the right, but as soon as the object has passed to the right of the middle line, the left eye, by a movement of both eyes back to the left, takes up the work of following the object the rest of the way. Thus each eye avoids the necessity of making a strong movement of abduction. The third and commonest kind of alternating squint is that in which the patient, sighting an object straight ahead of him and either far or near, fixes with either eye indifferently and sometimes with either at will. In this case the two eyes are usually, though not always, equal in refraction and vision. This kind of alternation occurs both in esotropia and exotropia, and it is probable that in their beginning very many cases of squint are alternating. In all types of alternating strabismus since the two eyes are used alternately, the sight of both remains good, though it may be unequal; in *monocular* squint, the squinting eye is often amblyopic (see § 337).

II. Binocular Vision.

283. Binocular Single Vision: Fusion.—As we have seen, in all but a small minority of cases binocular fixation is present whenever we look with both eyes. The image of the object looked at then is formed on each fovea (see Fig. 131).

Yet, though there are the two images, we see, not two objects, but one (binocular single vision). For the mind by a peculiar process which we denominate *fusion* combines the two retinal images into a single visual impression. This binocular visual impression differs somewhat in character from the impression gained when we look with either eye by itself—the image being rather more distinct and also more solid-looking. In fact, vision with both eyes differs from vision with one eye in that with the former we get clear, and with the latter but imperfect, ideas of distance, depth, and relief. In other words, when we look with both eyes we have solid or *stereoscopic vision* (see § 286).

The mental faculty by which fusion is effected is called the *fusion faculty*. It acts, presumably, by means of a cerebral nerve-centre (*fusion centre*), the site of which is undetermined.

Not only does the mind fuse the two retinal images of any object looked at directly, so that that object appears single, but it fuses the images of a good many other objects as well. Thus, if we are fixing an object O (Fig 131) with both eyes, O, which forms an image on each fovea, will appear single and will look to be straight in front of the eyes. Another object, O_1, situated

to the left of O, will form its retinal image a certain distance, say 10° to the right of the fovea in the right eye (at b). If now it is so situated as to form its retinal image 10° to the right of the fovea in the left eye too (at b_1) it will appear single, the two outlying retinal images being fused in this case just as the two foveal images were. In general, any object whose retinal images are formed equally far and in the same direction from the fovea in each eye—are formed as we say, on the *corresponding points* of the two retinæ—will appear single.

284. Binocular Projection.—Just as in the case of the monocular visual impression, only more accurately and with greater certainty, the visual impression produced by fusion of the two retinal images is mentally projected, or referred to a definite portion of space outside of the body. The point to which we thus refer it is the point of intersection of our two visual lines. Thus, when we look at an object one foot off the eyes turn until their visual lines are both directed at the object, and hence intersect at the point where it is situated. Then each foveal image is projected out to this point of intersection—i. e., to the place that the object itself actually occupies.

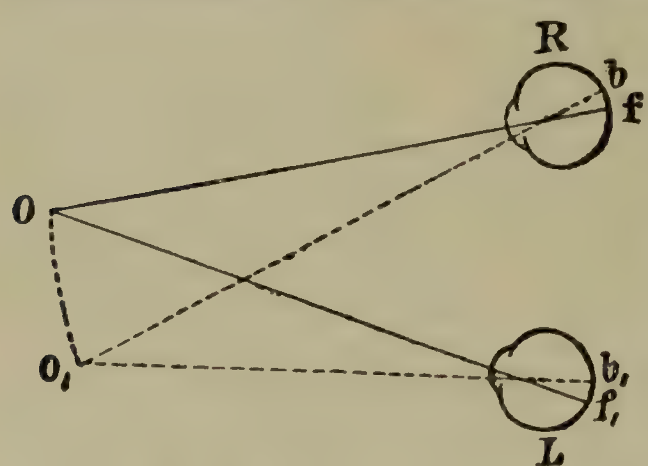

FIG. 131.—BINOCULAR SINGLE VISION. The object o, forming its image on the fovea of each eye (f and f_1) is seen single. So is the object O_1, which forms its image on the corresponding points, b and b_1.

Projection, when performed by both eyes may be conceived of as taking place from a single *cyclopean eye* situated in the median line and about at the root of the nose, but it is not necessarily true that projection takes place thus.

285. Double Vision; Physiological Diplopia.—As we have just seen, when the images of the same object fall upon corresponding points, the object appears single. It is otherwise when the images fall upon points not corresponding—e. g., on the fovea in one eye and on any outlying portion of the retina in the other. Then, in general, we have double vision (diplopia), the mind receiving two visual impressions of the same object. In this case each eye projects the image differently—i. e., locates it in a different point in space.

In many cases this diplopia is *physiological.* Thus, in Fig. 132, the object A, at which both eyes are looking and which, therefore, forms its retinal images on the two foveæ M and M' respectively, appears single, being projected by both eyes to its true place, A; but B, an object more remote than A and situated in the prolongation of the line MA, will appear double, for while it forms its image in the right eye on the fovea, M, it will form its image in the left eye on a point, N, to the right of the fovea. To the right eye it will appear directly in line with A (behind it); to the left eye it will, according to the law of projection, appear to be to the left of A, and the more so the further N is to the right of M. We shall have thus a condition of *homonymous* diplopia—i. e., a condition in which the image seen by the left eye is to the left of that seen by the right eye.

The object, C, lying nearer to the eyes than A and in the line, AM will, to the right eye, seem to be in line with A (just in front of it) and to the left eye will seem to lie to the right of A, since C forms its image in the left eye at O to the left of the fovea. Such diplopia, in which the image seen by the left eye appears to the right of that seen by the right eye, is said to be *heteronymous* or *crossed.* This physiological diplopia, crossed for objects nearer than the object looked at and homonymous for more distant objects, although readily demonstrable, rarely, if ever, appears spontaneously and hardly ever occasions any confusion.

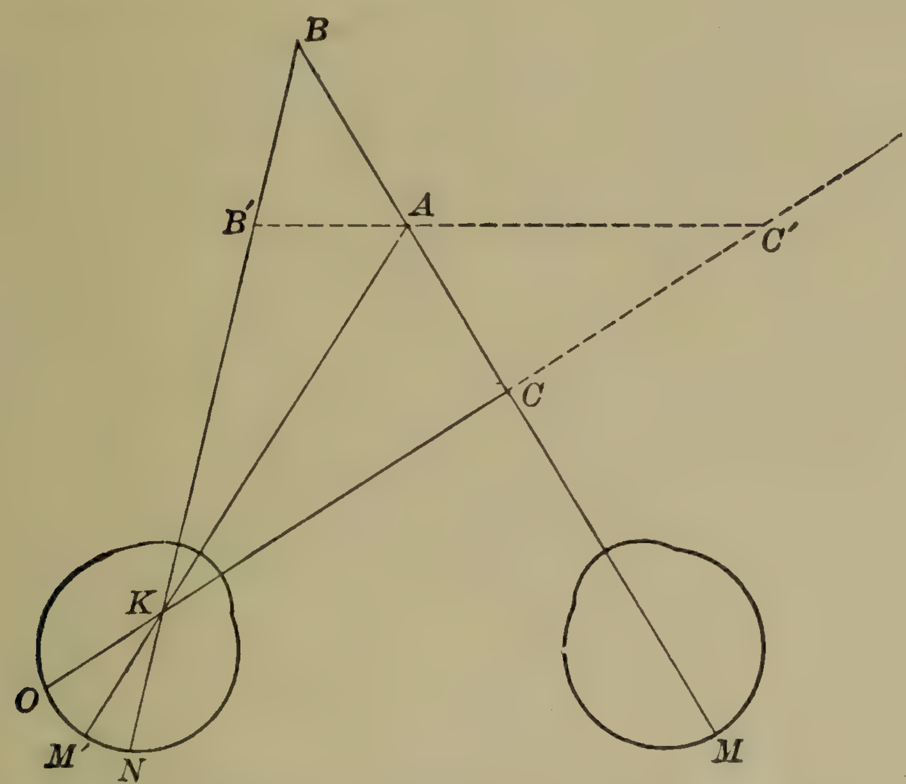

FIG. 132.—PHYSIOLOGICAL DIPLOPIA, (after Posey and Spiller).

In physiological diplopia both images are indistinct, and if one image happens to fall on the fovea in one eye it often, perhaps usually, appears less distinct than the non-foveal image formed on the other eye. In pathological diplopia the opposite conditions obtain (§ 289).

286. Stereopsis.—The visual impression that we get with both eyes together differs from that gained when we look with either eye by itself in being more distinct (see § 187) and also more solid-looking. In the former case we get clear, in the latter but imperfect ideas of distance, depth and relief. In other words when we look with both eyes we get solid or stereoscopic vision (stereopsis).

We owe our ability to gauge depth and distance largely to the subconscious impression afforded by physiological double images. In the case shown in Fig. 132, we regard B as further off and C as nearer than A, because although we are not consciously aware of it B and C look double; the double images of B being crossed, and those of C being homonymous. Another factor that aids in binocular stereopsis is the varying amount of muscular effort that we make in converging and in accommodating on objects differing in remoteness. In monocular vision the perceptions of depth are mainly dependent on effects of contrast (light and shade) and distinctness. Furthermore, when moving (as in a railway train), we can gauge the distance of objects we are passing by the parallactic displacement of the nearer on the more remote.

287. Artificial Diplopia produced by Prisms.—Diplopia may also be produced by prisms. Thus, suppose both eyes are looking at an object, A (Fig. 133) so that the image of that object falls on the fovea in each eye, and then a prism with the *base directed inwards* (towards the nose) is placed before the left eye. The prism will deflect the rays of light towards its base, so that the retinal image in the left eye will no longer fall on the fovea, M', but to the right of it, N. The eye, according to the law of projection, will regard this image as situated to the left of A,—i. e., the right eye fixing A with its fovea will see A in its true place, while the left eye will see a similar

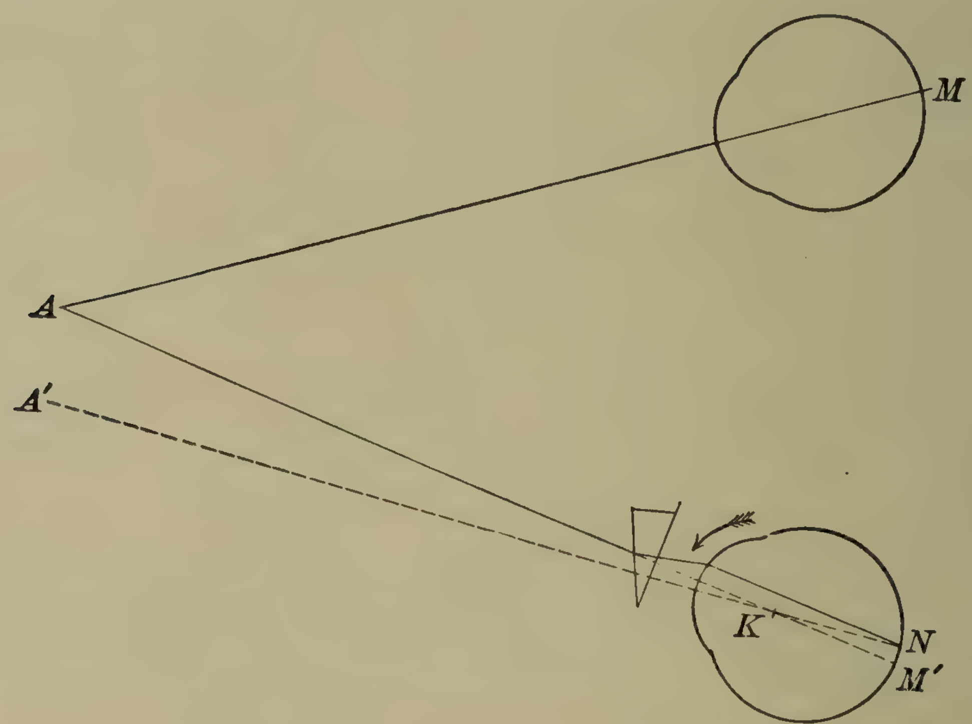

FIG. 133.—DIPLOPIA PRODUCED BY PRISMS. (After Duane in Posey and Spiller).

image, but dislocated to the left (at A'). There will then be a condition of homonymous diplopia.

So also, a prism placed *base out* before one eye ill wproduce crossed diplopia. A prism placed *base up* or *base down* will make the corresponding image appear below or above the other— i. e., will cause a vertical diplopia.

288. Pathological Diplopia.—Diplopia is also caused by a deviation of the eyes. That is, if the right eye (R, Fig. 134) is fixing the object A, but the left eye, instead of fixing it, is *turned too far inwards*, the retinal image of A in this eye will fall not on the fovea M′, but to the inner or right side of it (N). Now, the mind really does not know how the left eye is pointing; in fact, it seems to regard both eyes as looking in the same direction; hence, the object whose image is on the fovea of the left eye will be projected out to A, the direction in which the mind conceives this eye to be pointing (see § 280). An object whose retinal image is at N, to the right of the fovea, will be regarded as located correspondingly far to the left of A. Such a patient, therefore, sees two images of A—one seen by the right eye, situated in its true place straight ahead; the other seen by the left eye, situated to the left (A')—i. e., there is here a homonymous diplopia. If the left eye had been *turned out* there would have been a crossed diplopia; if *up* or *down*, a vertical diplopia.

Vertical diplopia is called *right* or *left* according as the image seen by the right or the left eye is the lower.

In conditions causing vertical diplopia, the image that should be higher may seem on a level with the other, or back of it, or even lower. This occurs particularly when the eyes are directed down, and is due partly to the character of the background upon which the images are projected, partly to other factors (M. Sachs, Frölich). Sometimes in lateral diplopia one image appears back of the other.

Double images may not only be placed side by side (lateral diplopia) or one over the other. but may also be *inclined* in such a way that their upper

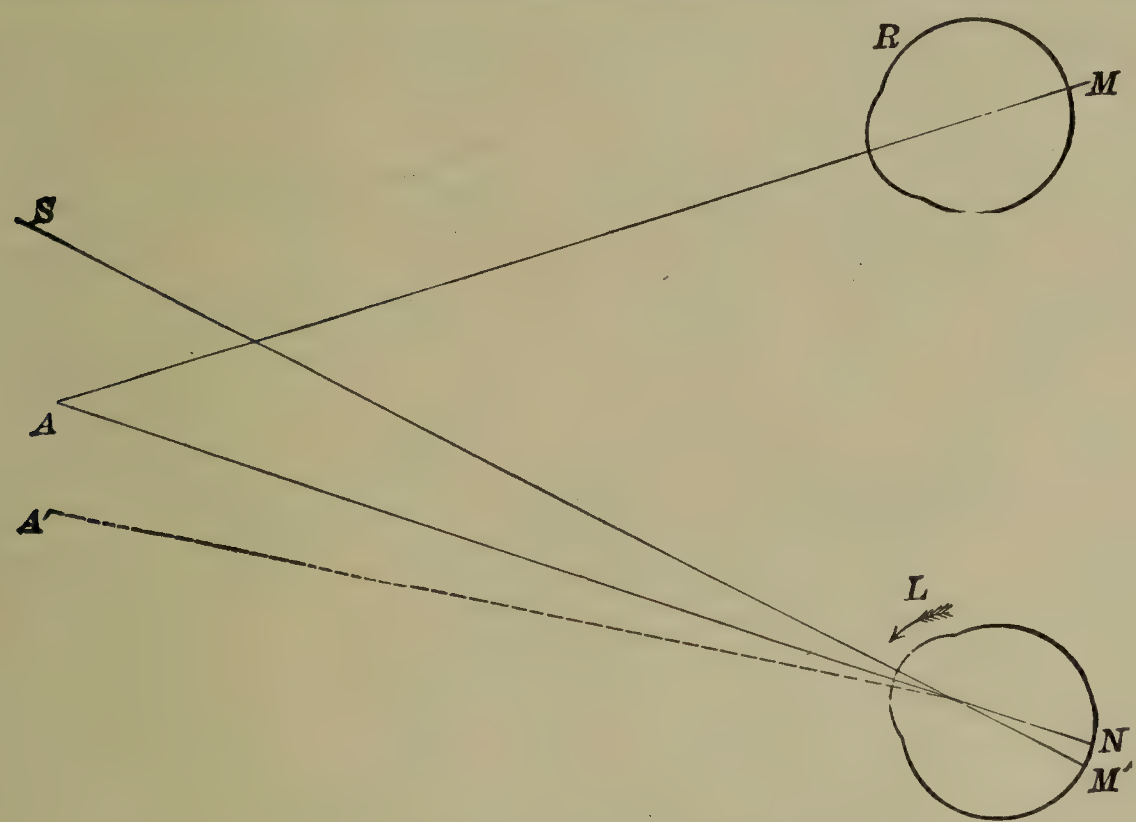

FIG. 134.—PATHOLOGICAL DIPLOPIA, (after Duane in Posey and Spiller).

or lower extremities are approximated. This is the case when one of the two eyes has undergone a rotation about its sagittal axis and the other has not.

In Fig. 135 A, *R* and *L* represent the posterior halves of the two eyes seen from behind and imagined to be transparent, so that the inverted image of an arrow is seen as it is situated upon the retina. In the right eye the vertical meridian of the retina, *v v*, really does stand vertical, but in the left eye (v_1 v_1) it is supposed to be inclined. The image of a vertically directed arrow is also vertical in the two retinæ; hence, in the right eye it coincides with the vertical meridian, but in the left eye it forms an angle with the vertical meridian, because this is inclined. Since, now, the left eye has been previously accustomed to consider as vertical only those objects the images of which coincide with the vertical meridian, it will consider the arrow as having an oblique position[7]. Hence, two images of the arrow are seen (Fig. 135 B, *w* and *s*), of which that belonging to the left eye stands obliquely. Necessarily, according to the laws of projection, the image, *s*, seen by the left eye, appears tilted the opposite way to that in which the eye itself is tilted. For the lower end, p_1, of the retinal image is formed on the outer as well as the lower part of the retina. Consequently it is projected not only up, but also in (to the right), so that the point of the arrow appears located in space above and to the right of

[7] This applies only to a pathological tilting in which the vertical meridian of one eye is no longer parallel with that of the other. The physiological tilting which occurs in the oblique positions of the gaze (see §§ 280, 295, 296), and in which the vertical meridians of the two eyes, although tilted, remain parallel to each other, is not associated with apparent tilting of the object looked at. Nor do we observe any tilting of a vertical line of light if while looking at it we tip the head to one shoulder. The retinal image of the line in this case sweeps successively over different meridians of the retina and yet always appears vertical, because our muscular sensations which tell us how our head is directed correct the impressions derived from the retina (cf. Sherrington's observation, § 280).

the arrow's centre. So too, the upper end, p, of the retinal image, which falls on the inner part of the retina, is projected out (to the left) as well as down, and appears in space, therefore, to the left of the arrow's centre. Hence, while the eye itself is tilted to the left, the image projected by it is tilted to the right.

Thus it will be seen that in all varieties of diplopia *the image formed by the deviating eye is always deflected in a direction opposite to that in which the eye is deviated* (to the right if the eye is deflected to the left, up if the eye is deflected down, tilted to the right if the eye is tilted to the left, etc.). This is a necessary consequence of the law of projection.

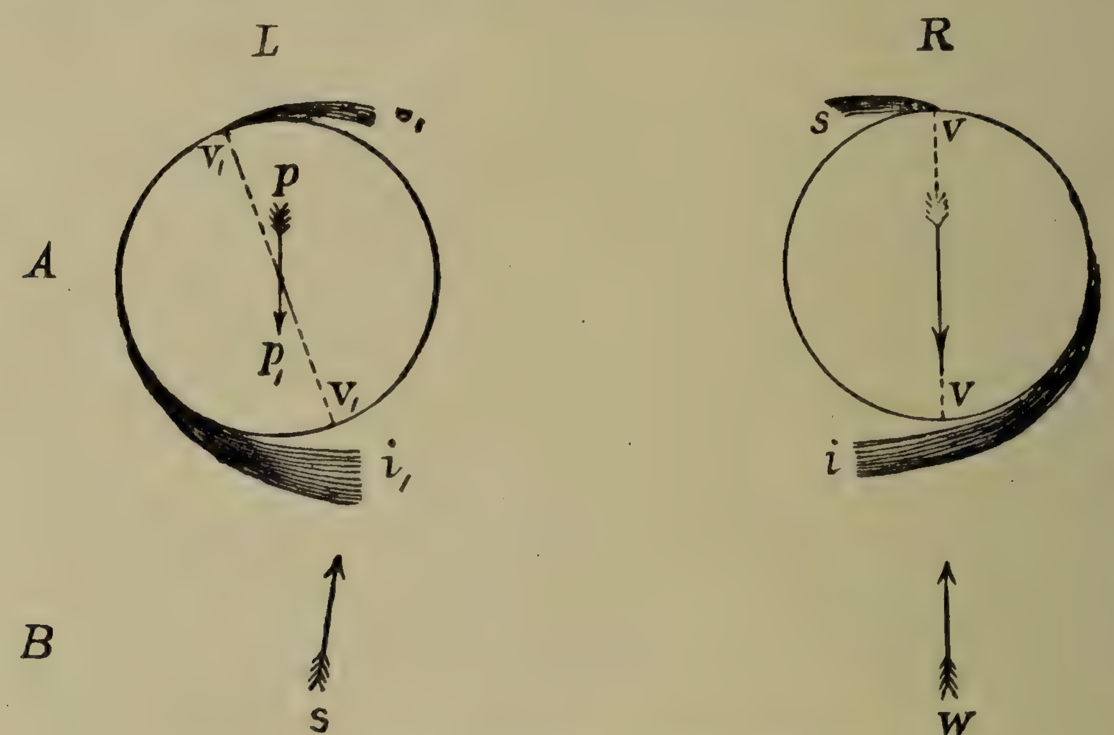

FIG. 135.—DOUBLE IMAGES WITH OBLIQUITY.

Binocular single vision is converted into *binocular double vision* whenever one of the two eyes leaves the correct position of fixation. This takes place most frequently in consequence of disturbances in the motor apparatus of the eye. But the eye can also be forced mechanically into an incorrect position—e. g., by tumors in the orbit, etc. Binocular double vision can readily be produced experimentally by pushing one eye a little to one side by pressure with the finger. Finally, double vision ensues whenever the excursions of one eye are limited, as compared with those of the other by mechanical obstacles, as, for example, by symblepharon or by pterygium.

289. Characters of Double Images.—In pathological diplopia and also in diplopia produced by a prism, the image formed in the deviating eye, being extra-foveal will be less distinct than the foveal image formed in the fixing eye. It is, hence, called the *false* image.

In diplopia the *distance between the double images* measured in degrees of arc corresponds precisely to the deflection of the deviating eye from the correct position. The linear distance between the double images, on the contrary, depends not only upon the degree of deviation, but also upon the distance to which the double images are projected. The greater this distance is, the farther apart the double images appear to be. When the double images stand very close to each other they overlap in part, so that only their outlines appear double. In this case the patient is often not aware that he sees double, but only complains of seeing indistinctly or complains that every object has a shadow.

290. Overcoming Diplopia.—Diplopia produced by prisms, or diplopia produced by pathological deviation, may be overcome if the *eye is turned* so that the image in either case falls upon the fovea. Thus, the homonymous diplopia shown in Fig. 133 and in Fig. 134 will be abolished if the left eye

turns out as shown by the arrow, for, turning the eye out will turn the fovea M' in, and when M' has gone in far enough the retinal image of A will fall upon it; then the retinal image will fall on the fovea in each eye, and there will be single vision.

The ability to overcome diplopia depends partly on the readiness with which the special movement required for the purpose can be executed (see § 299), partly on the development of the fusion faculty (see § 291).

Diplopia may also be overcome by *prisms*. In the case shown in Fig. 134, in which the left eye is deviated in (to the right) and the image of A therefore falls on N to the inner (right) side of the fovea, M^1, we may get it on M^1, without turning the eye by placing before the latter a prism with its apex directed to the right. This prism, deflecting the rays of light away from its apex will swing the ray AN to the left, and, if sufficiently strong, will swing it so as to take the direction SM^1, the image of A then falling on M^1.

Applying the same principle to other kinds of double vision we see that to correct and therefore to measure diplopia of any sort by a prism we must always put the apex of the prism in the direction in which we wish the projected image A to go, i. e., opposite to the way in which it is deflected. As the image A is always deflected opposite to the way in which the eye itself is deviated, we say that *to correct the diplopia or the malposition of the image caused by any deviation of an eye, we must place the prism before that eye with its apex pointing in the direction in which the eye itself is deviated.*

291. Congruous and Incongruous Diplopia; Defects of Fusion.—The disturbances of binocular vision comprise binocular diplopia and defects of fusion.

(a) **Binocular diplopia** may be *congruous*, when, as in the conditions so far considered, it corresponds in amount and character with the deviation causing it; and in the reverse case is *incongruous*.

Incongruous diplopia, also called *perverse* or *paradoxical* diplopia, is due to some unusual sort of binocular projection and occurs occasionally in squint or cured squint. In a case of this sort we may find, for example, an esotropia with crossed diplopia, or, if a homonymous diplopia is present, it is much less than the deviation in amount (see § 337).

(b) **Defective or Absent Fusion.**—In some cases the fusion faculty may be so poorly developed that binocular vision is absent altogether. In this case two things happen. First, if one eye is deviating there is *suppression* of the double image that should be appreciated by the deflected eye, so that no diplopia is present spontaneously nor usually can it be evoked by prisms or any other artifice. Second, if either spontaneously or by operation or by prisms the two eyes are made to fix the same object, the latter is perceived only by one eye so that stereoscopic vision is not obtained. This condition is common in squint, even when the sight in the squinting eye is normal. But it may be produced artificially in any eye, especially by conditions which make the sight of one eye different from that of the other.

Suppression is a *psychic process.* The eye affected by it really does see, but the visual perceptions set up by it do not excite attention. It may be produced voluntarily, e. g., by those who, looking through a microscope with one eye, leave the other open and yet do not see with it. The ultimate consequence of continued suppression if confined to one eye is amblyopia (§ 241).

Suppression affects only objects in the field of vision *common to the two eyes* (see § 235). Hence, objects that are so far in the temporal field of the squinting eye that they cannot be seen by the other are recognized by the former. Accordingly, even in suppression a man's peripheral field (recognition of dangers to one side of him) is practically normal[8]. Moreover, the object which forms its image on the fovea of the deviating eye is not suppressed, but this object is not seen double since its image formed on the retina of the fixing eye is suppressed (*regional exclusion* or *bifoveal exclusion*).

In suppression the vision may *alternate* from one eye to the other, this occuring for example, in alternating squint or in artificial suppression. If the vision of the two eyes is quite different, confusion may be caused by the sudden change in the character of the images when first one eye and then the other is used.

In many cases, especially in squint, the fusion faculty is not absent but simply more or less *deficient.* Thus the patient may be able to fuse images but does not get stereopsis; or he may fuse images of one kind and not others. Again, he may get fusion under ordinary conditions, but cannot do so, if the least obstacle is interposed. In such cases, which occur especially in neurasthenia, hysteria, and organic brain disease, diplopia is easily produced and when produced is difficult to overcome. In such cases the mere act of placing a red glass or the weakest prism before one eye suffices to evoke an insuperable diplopia.

A peculiar and very rare perversion is found in some who never by any adjustment of the eyes or with any prism get fusion of the double images—in whom, for example a prism of 3 *d*, base out, produces a crossed, and a prism of 3.5 *d* a homonymous diplopia, but no prism produces a single vision. This condition of so-called *antipathy to single vision* occurs in squint, particularly after operation, and is probably attributable to non-development of the fusion faculty.

292. Binocular Vision and Binocular Fixation Compared.—Derangements of binocular fixation cause the various deviations of the eyes (squint, heterophoria). Direct evidence of the existence of these derangements can be had only by the *objective tests* which show whether binocular fixation exists or not and whether the eyes move in harmony or not.

Derangements of binocular vision, while forming an important evidence of an existing deviation, are indirect evidence only, since the tests that demonstrate them are *subjective tests* which may be inaccurately reported by the patient. Such tests, moreover, are dependent on the production of a symptom (diplopia) which may be present when the deviation is absent (e. g. if prisms are used or in the case of incongruity) and may be absent when the deviation is present (suppression). The subjective tests are therefore sometimes inapplicable, as in most cases of squint, and are occasionally misleading.

In the great majority of cases binocular fixation and binocular vision are strictly correspondent, and the objective and subjective tests are completely in accord.

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[8] As a matter of fact it is somewhat smaller in strabismus convergens, because, owing to the convergent position of the eyes, the visual fields overlap more than usual; and for an analogous reason in strabismus divergens it is larger than usual.

III. Movements of the Eyes.

293. Character and Starting Point of Eye Movements; Main and Subsidiary Actions of Muscles.—The movements of each eye separately comprise abduction, adduction, elevation, and depression. The movement in each case starts from the position of equilibrium (*primary position*). This in general is the position assumed by eye when directed straight ahead or a little down. When both eyes are in the primary position, their visual lines are parallel, and the vertical meridians of the two corneæ are also both vertical and parallel. The maintenance of the eye in the primary position is effected by a certain contraction (tonus) of all the muscles acting in unison. When the eye leaves the primary position for any other, from one to three of the muscles relax this contraction and the remaining muscles contract still more, so as to pull the eye in the given direction.

The relaxation that takes place in the opponents to the contracting muscles is not to be considered as a passive process but as due to the *active inhibition* which always affects an innervation of one sort when an opposing innervation is set into action (Sherrington's law).

Except in the case of the internal and external rectus, none of the muscles has a simple action. Each of the other muscles—the elevators and depressors—has a main and certain subsidiary actions. The latter include not only adduction and abduction, but also torsion effects or tilting of the vertical meridian of the cornea either temporally (extorsion) or nasally (intorsion) and movements of the lids produced by fascial connections between the muscles and the latter (see § 66). The main action (elevation and depression) of the superior and inferior recti increases as the eye is abducted, and that of the obliques increases as the eye is adducted. The subsidiary effects (adduction, abduction, torsion) of these muscles in each case behave in just the reverse way. These facts are shown in the following table:

	Main Action	Subsidiary Action
Superior Rectus	Moves eye up; action increases as eye is turned out; becomes nil when eye is turned in.	Adducts eye and rotates vertical meridian inward; action increases as eye is turned in. Raises upper lid.
Inferior Oblique	Moves eye up; action increases as eye is turned in; becomes nil when eye is turned out.	Abducts eye and rotates vertical meridian outward; action increases as eye is turned out.
Inferior Rectus	Moves eye down; action increases as eye is turned out; becomes nil when eye is turned in.	Adducts eye and rotates vertical meridian out; action increases as eye is turned in. Pulls down lower lid.
Superior Oblique	Moves eye down; action increases as eye is turned in; becomes nil when eye is turned out.	Abducts eye and rotates vertical meridian in; action increases as eye is turned out.

The actions of the eye muscles may be deduced from a consideration of the *anatomical relations*. We may take the case of the superior rectus. This runs from within outward so as to cut obliquely across the antero-posterior axis of the eye when the eye is in the primary position (Figs. 47 and 136 A). Hence in this position only part of its energy is expended in pulling the eye up, part in adducting it and rotating the vertical meridian inward. When the eye is abducted (Fig. 136 B) the superior rectus is in line with the antero-posterior axis of the eye ($G\ G_1$) and acts as a pure elevator, pulling the eye up therefore very strongly. On the other hand, if the eye is in extreme adduction (Fig. 136 C) the muscle is almost at right angles to $G\ G_1$ and hence acts only to adduct

and intort the eye. Like considerations, mutatis mutandis, obtain for the inferior rectus and for the obliques, whose tendons of insertion all cross the sagittal axis of the eye obliquely (see Fig. 47). The action of the obliques is exerted on the posterior portion of the eye, which the superior oblique elevates (thereby depressing the cornea) and the inferior oblique depresses.

These anatomical facts supplemented by physiological experiments and the results obtained in cases of paralysis show that for the *obliques* elevation and depression, respectively, are the predominant functions; and that the torsion effects and the abducting or adducting action of each in the primary position and also in simple elevation and depression of the eye just balance the opposing actions of the superior and inferior recti

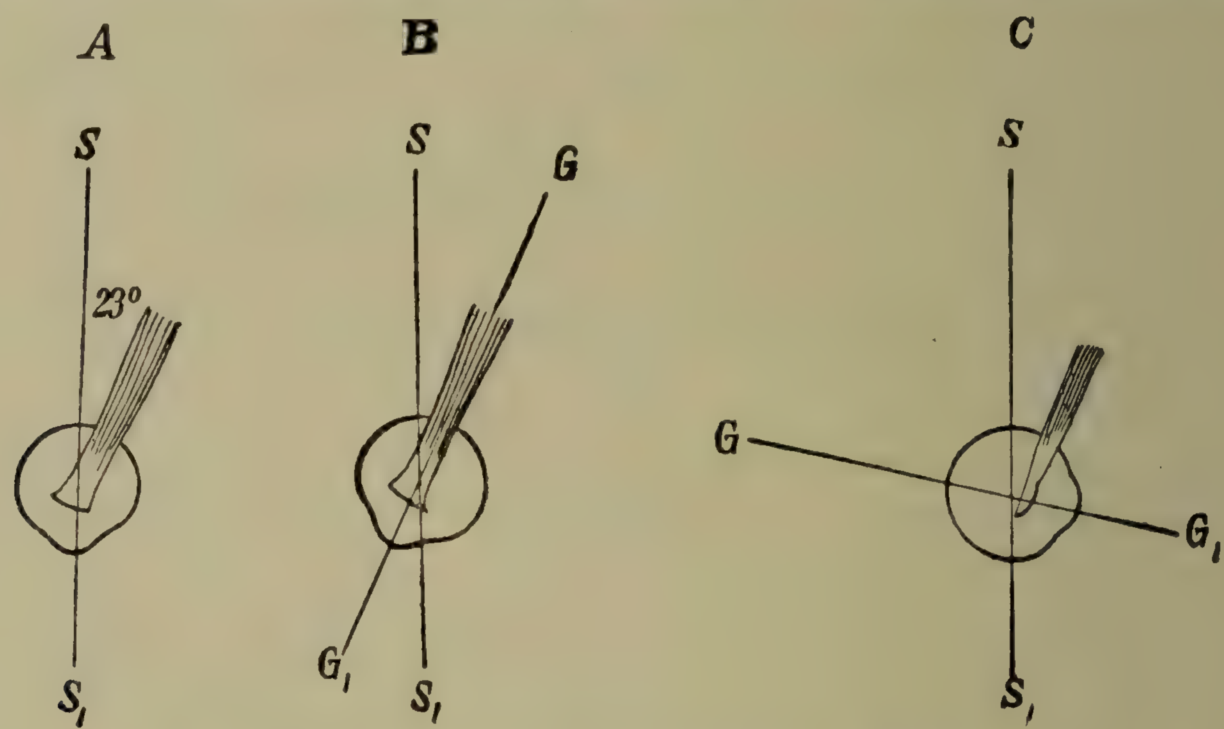

FIG. 136.—METHOD OF ACTION OF THE SUPERIOR RECTUS.
A, in looking straight forward; B, in abduction; C, in adduction; S S_1, sagittal axis of motion; G G_1 visual axis.

respectively. The elevating action of the inferior oblique and the depressing action of the superior oblique are considerably less in the primary position than the corresponding actions of the superior and inferior recti, but as the eye is adducted the obliques soon equal and then progressively surpass the recti in vertical effect.

The actions of the ocular muscles are considerably modified by the system of *fascial bands* in the orbit and particularly by the *check ligaments* (§ 54). The latter have two functions. They prevent any extreme rotation of the eye in any direction, and they help to support the eye and keep it from leaving its place. The two check ligaments of the superior oblique and the other fascial bands above together with the strong fascial band connecting the inferior oblique and inferior rectus and stretched beneath the eye (suspensory ligament of the eye) help to keep the eye from sagging down; and the lateral check ligaments help to keep it from moving too far laterally. The tension of the four recti keeps the eye from pushing too far forward, and the counter-tension of the obliques, the check ligaments, and the palpebral connections of the muscles keeps the eye from retracting. By all these agencies assisted by the pressure of the orbital fat, the eye is slung in the orbit and kept in place, so that when the muscles act on it the eye rotates more or less as in a *socket*. The true socket of the eye is held to be not Tenon's capsule, but the orbital fat surrounding it (Motais).

294. Antagonists.—The internal and external recti are direct antagonists. The superior and inferior recti are direct antagonists in their vertical and their torsion actions, but both co-operate to adduct the eye. The like is true of the obliques, which antagonistic in other regards, both abduct the eye.

The superior rectus and inferior oblique oppose each other in lateral and torsion effects, but both act to elevate the eye. The like is true of the superior oblique and inferior rectus, which otherwise antagonistic, both depress the eye.

Opposing muscles act not only to check, reverse, or modify the movement initiated by the antagonist, but also by their counteraction make the movement of a moving eye *steadier* (see §§ 295, 298) and furthermore maintain the *balance* of the eye when at rest.

295. Monocular Movements.—As the following scheme shows, in every movement of the eye, including simple adduction and abduction, from three to five muscles take part, some acting directly, others serving to steady the eye in its course.

ABDUCTION

The eye is carried straight *out* by the external rectus assisted especially toward the end of its course, by the two obliques.[9] The latter nullify each others' vertical and torsion action, so that the eye moves straight out, and its vertical meridian remains vertical. The external rectus pulls the outer canthus outward.

ADDUCTION

The eye is carried straight *in* by the internal rectus assisted, especially toward the end of its course by the superior and inferior recti.[9] The latter nullify each others' vertical and torsion action, so that the eye moves straight inward, and its vertical meridian remains vertical. The inner canthus is pulled back and inward.

ELEVATION

The eye is carried *straight up* by the superior rectus and the inferior oblique, the external and internal recti probably acting to steady it in its course. The two elevating muscles counteract each other so far as regards lateral action and torsion, so that the eye moves straight up and the vertical meridian remains vertical. The superior rectus helps to lift the upper lid.

In moving *up and out*, the eye is carried up mainly by the superior rectus, out by the external rectus assisted by the inferior oblique. The vertical meridian is tilted outward.[10]

In moving *up and in*, the eye is carried up mainly by the inferior oblique, in by the internal rectus assisted by the superior rectus. The vertical meridian is tilted inward.[10]

DEPRESSION

The eye is carried *straight down* by the inferior rectus and the superior oblique, the external and internal recti probably acting to steady it in its descent. The lateral and the torsion actions of the two depressors nullify each other, so that the eye moves straight down and its vertical meridian remains vertical. The inferior rectus assists in depressing the lower lid.

In moving *down and out*, the eye is carried down mainly by the inferior rectus, out by the external rectus assisted by the superior oblique. The vertical meridian is tilted inward.[10]

In moving *down and in*, the eye is carried down mainly by the superior oblique, in by the internal rectus aided by the inferior rectus. The vertical meridian is tilted out.

[9] When the eye is abducted the superior and inferior recti also act, but as they have little or no adducting power when the eye is turned out, and as their torsion and vertical actions nullify each other, all they can do is to steady the eye in its course by their mutually counteracting pull. The same is true of the two obliques when the eye is adducted.

[10] When the eye is abducted the lateral and torsion action of the superior rectus is slight and that of the inferior oblique marked; on the other hand, the elevating power of the latter is nil and that of the superior rectus considerable. Hence if both muscles act, the eye is carried up almost exclusively by the superior rectus while the preponderating lateral action of the oblique carries it still further out, and tilts its meridian outward. Similar reasoning applies to the statements made regarding the lateral movements and the position of the vertical meridian in the other directions of the gaze.

Torsion

In the primary position and also in looking up, down, in, and out there is no torsion. In looking up and in and down and out there is intorsion; in looking up and out and down and in there is extorsion.[11]

296. Field of Fixation.—The limits of the excursions of the eye in all directions of the gaze bound the field of fixation. It represents that portion of space that we can see by central fixation without moving the head.

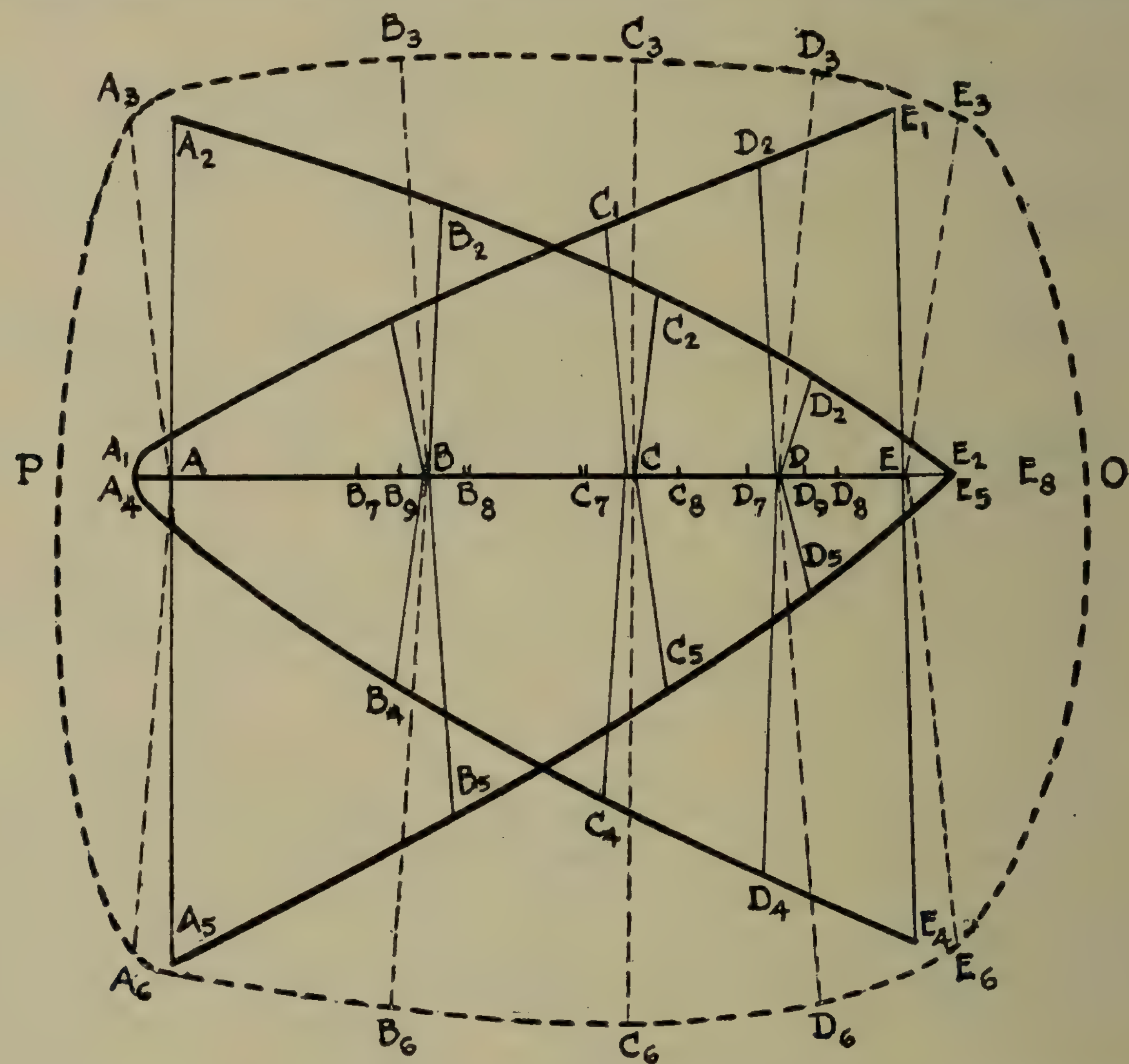

Fig. 137.—Projection of Field of Fixation of Eye and of Field of Action of Each of the Ocular Muscles. (After Duane.)

The limits of the field of fixation are variously given by different observers and probably vary somewhat widely even in normal subjects. According to Landolt, with whom the translator is in substantial agreement, the field of fixation extends not less than 47° in all directions, except above, where it is generally somewhat less. Outward and inward it is usually somewhat more than this (about 50°), and, below it often extends to 55°. Permanent restriction within 47° in any direction except above must be regarded as probably pathological (Landolt). Quite certainly pathological would be a rotation upward of less than 40°, one downward of less than 50°, and one in or out of less than 45°.

Fig. 137 represents a projection of the field of fixation. In this, C is the projection of extremity of line of sight (point of fixation), when eye is in primary position; D, E, O, its projection when eye is abducted 18°, 25°, and 45°, respectively; B, A, P, projection when eye is adducted 20°, 45°, and 50°, respectively. The distance CO, represents the maximum degree of excursion of the eye outward. Most of this movement is effected by the *external rectus*, but a certain portion especially towards the outer end of the excursion is accomplished by the united action of the two obliques (see infra). The distance CP represents the maximum range of excursion of the eye inward. This inward movement is effected mainly by the *internal rectus*, assisted especially towards the end of the excursion by the superior and inferior recti (see infra).

[11] These facts can be proved by physiological experiments with after images.

The lines AA_1, BB_1, CC_1, DD_1, EE_1, represent the amount and direction of the movement produced by the *superior rectus* when the eye is respectively adducted 45° (A); adducted 20° (B); in the primary position (C); abducted 18° (D); and abducted 25° (E). The lines AA_2, BB_2, CC_2, DD_2, EE_2, represent the amount and direction of the movement effected by the *inferior oblique* and the dotted lines AA_3, BB_3, CC_3, DD_3, EE_3, the movement effected by the inferior oblique and the superior rectus acting together. Similarly, the lines AA_4, BB_4, CC_4, DD_4, EE_4, represent the lines of action of the *inferior rectus*; AA_5, BB_5, CC_5, DD_5, EE_5, those of the *superior oblique*; and AA_6, BB_6, CC_6, DD_6, EE_6, those of the two latter muscles combined. The heavy lines $A_1B_1C_1D_1E_1$ and $A_4B_4C_4D_4E_4$ represent the limits of the *fields of action* of the superior and the inferior recti, respectively; and the heavy lines, $A_2B_2C_2D_2E_2$ and $A_5B_5C_5D_5E_5$ the limits of the fields of action of the inferior and the superior obliques. The heavy dotted line A_6PA_3 B_3C_3, etc., represents the limit of the *field of fixation*.

It will be seen from the diagram how the *vertical* (elevating and depressing) action of the superior and inferior recti increases, and how the vertical action of the obliques decreases progressively as the eye is carried from a position of marked adduction (A) to one of moderate abduction (E). It will also be apparent how the *lateral* action of each of these muscles diminishes as its vertical action increases; so that the adductive power of the superior and inferior recti shows a progressive diminution, and the abductive power of the two obliques a progressive increase as the eye passes from A to E. That is, at A (i. e., when the eye is adducted 45°) the superior and inferior recti have no vertical action at all, but simply adduct the eye through a comparatively large extent, while the two obliques have no lateral action at all, but simply elevate and depress the eye. At E, on the other hand (when the eye is abducted 25°), the two recti no longer act as adductors at all, but simply elevate and depress the eye; and the two obliques no longer exert any vertical effect, but combine to carry the eye outwards, their abductive action here being particularly great.

At A the *superior rectus and the inferior oblique acting together* to their full extent will carry the eye up and noticeably inwards (to A_3); since here the adductive action of the rectus is at its maximum and besides is not balanced by any opposing abductive action on the part of the oblique. Similarly at E the two muscles acting together will carry the eye upwards and noticeably outwards. In intermediate positions, as at B and D, the lateral action of one muscle will partially counteract that of the other, so that the net lateral effect will be less. E. g., at B the adductive action of the superior rectus is less than it was at A and moreover is now opposed by a moderate abductive action on the part of the inferior oblique, so that the net adductive effect is but slight. The eye, therefore, here is carried up by the inferior oblique, assisted somewhat by the superior rectus, and is also carried slightly inwards (to B_3). At D, on the contrary, it is carried upwards mainly by the superior rectus, assisted somewhat by the inferior oblique, and is also carried slightly outwards (to D_3), by the preponderating lateral (abductive) action of the latter muscle. At C (the primary position) the adductive action of the superior rectus balances the abductive action of the inferior oblique, and hence the effect of the two elevators acting together will be to carry the eye straight upwards.

In a similar way the *inferior rectus and the superior oblique acting together* will carry the eye down and in (AA_6, BB_6), down and out (DD_6, EE_6), or straight down (CC_6), according as the eye is already adducted, abducted, or in the primary position.

The *superior and inferior rectus acting together* with just sufficient force to neutralize each other's vertical action, will combine to adduct the eye, the adducent effect being forcible at A ($=AA_1+AA_4$), and diminishing gradually to E where it is zero. At E, therefore, i. e., when the eye is abducted 25°, the inferior and superior recti acting together will produce no movement and hence in this position are direct antagonists.

Similarly the *superior and inferior obliques acting together* neutralize each others' vertical action, but combine to abduct the eye. The abducent effect is marked at E ($=EE_2+EE_5=EE_8$), and diminishes progressively to A, where it is zero. In the latter position, therefore, i. e., when the eye is adducted 45°, the two obliques, acting simply to elevate and depress the eye respectively, are direct antagonists.

The four muscles, *superior and inferior recti and superior and inferior obliques*

acting together with the force required to neutralize each other's vertical action, will produce a lateral effect varying with the amount by which the eye is already abducted or adducted. Thus, if by the action of the internal rectus the eye is already adducted 45° (to A), a position in which the obliques exert no lateral action at all, the total effect of the four muscles will be to carry the eye quite a little distance further inward (nearly to P. If the eye is adducted only 20° (B) the resultant action of the four muscles will be the difference between the adductive action of the two recti (BB_7), and the less marked abducent action of the two obliques (BB_8); i. e., the eye will be adducted slightly (to B_9). At C (the primary position), the abducent action of the obliques balances the adducent action of the superior and inferior recti, so that the contraction of the four muscles will cause the eye to remain stationary. At D, on the contrary, the abductive effect will preponderate somewhat, and the eye already abducted 18°, by the external rectus will be carried still further moderately outwards (to D_9). At E, i. e., when the eye is abducted 25°, the abducent effect is still more pronounced ($=EE_8$).

It is thus apparent that if all four muscles act together they will, if the eye is being adducted or abducted, *tend to carry it still further in the direction in which it is going.* They will, therefore, reinforce the external rectus, in abducting and the internal rectus in adducting the eye, and the amount of the reinforcement will increase in proportion as the eye is already abducted or adducted. So that the internal rectus, for example, when it begins to contract (i. e., is just leaving the primary position at C) will receive little or no aid from the contraction of the other four muscles, but, as it continues to act, (e. g. at B), will be more and more assisted by them, and finally when it reaches the limit of its contraction (at A) and is consequently working at a great mechanical disadvantage, will be strongly reinforced. In like manner the external rectus will, as its own efficiency diminishes with the increasing abduction of the eye, be assisted more and more by the simultaneous contraction of the other muscles. And it is altogether probable that it is in this way that the outward and inward excursion movements of the eye are rendered regular and uniform.

The diagram may be also used to illustrate the *torsion occurring in different directions of the gaze.* That is the line BB_1, may be taken to represent the fact that when the eye is adducted to B, the superior rectus tilts the vertical meridian quite strongly inward (as BB_1, is inclined). So BB_2 shows that in the same position the inferior oblique tilts the vertical meridian slightly outward and BB_3 shows that both elevators acting together tilt the meridian moderately in. Similarly the other facts regarding torsion in the oblique as well as in the vertical and lateral directions of the gaze, are indicated by the lines BB_6 CC_3, etc.

297. Varieties of Binocular Movements; Conjugate Movements.— When we look with both eyes, the muscles of one eye work with those of the other in such a way that the visual lines of the two eyes intersect in the object of fixation. To effect this the eyes either both move in the same direction (i. e., both to the right, both up, etc.)—parallel (or conjugate) movements—or both move so as to produce convergence or divergence.

The two eyes when performing *conjugate* movements move alike; i. e., the movement of one eye equals that of the other and the two eyes keep pace (*comitant* movement). The only exception is that in oblique movements of the gaze the inturning eye as soon as the object which it is following is cut off from it by the nose tends to rise somewhat higher in looking up and to sink a little lower in looking down than the out-turning (fixing) eye.

Extreme conjugate movements cause strain and are avoided by *movements of the head* in the same direction, regularly accompanying and supplementing the movements of the eyes.

The conjugate movements of the two eyes are performed by twelve muscles which may be grouped according to their main action as follows:

I. Lateral Rotators

Right Rotators	*Left Rotators*
(Carrying both eyes to right.)	(Carrying both eyes to left.)
R. external rectus	R. internal rectus
L. internal rectus	L. external rectus

II. Elevators

Right-hand Elevators	*Left-hand Elevators*
(Acting as elevators mainly when eyes are directed to right.)	(Acting as elevators mainly when eyes are directed to left.)
R. superior rectus	R. inferior oblique
L. inferior oblique	L. superior rectus

III. Depressors

Right-hand Depressors.	*Left-hand Depressors.*
(Acting as depressors mainly when eyes are directed to right.)	(Acting as depressors mainly when eyes are directed to left.)
R. inferior rectus	R. superior oblique
L. superior oblique	L. inferior rectus

The conjugate movements are called *versions*. *Lateriversion* is a movement of both eyes to one side including *dextroversion* (movement to the right) and *levoversion* (movement to the left). Similarly we may speak of the lateral rotators as lateriverters (dextroverters, levoverters).

The twelve muscles of the two eyes are thus divided into three groups of four each; and each group comprises two pairs of muscles, one muscle of each pair being in the right eye, the other in the left. The two muscles of each pair are called *associates*.

It will be seen that each pair of associates has its predominant *field of action* (i. e., direction of the gaze in which it is most active). In the case of the elevators and depressors, the field of action is a quadrant and in the case of the lateral rotators a double quadrant of the field of fixation. There are thus four fields of action, each containing a pair of laterally acting and a pair of vertically acting associates, thus:

Fields of Action

Up and Left	Up and Right
R. inf. obl. L. sup. rectus	R. sup. rectus L. inf. obl.
R. int. rectus L. ext. rectus	R. ext. rectus L. int. rectus
R. int. rectus L. ext. rectus	R. ext. rectus L. int. rectus
R. sup. obl. L. inf. rectus	R. inf. rectus L. sup. obl.
Down and left	Down and Right

The four oblique directions of the gaze (up and right, down and right, down and left, up and left) are called the *diagonal directions*. These with

the simple right and left movements are of prime importance in the diagnosis of paralysis (see § 319 et seq.), and constitute the six *diagnostic* directions.

In the four *primary* (or *cardinal*) directions (up, down, right, left) there is no tilting of the vertical meridian, while in each of the four diagonal positions there is a distinctive tilting, which, however, cannot be recognized by ordinary clinical tests. Thus, in looking up and to the right, both vertical meridians are tilted to the left. Yet a vertical object looked at does not appear tilted (see § 280 and § 288 Note 7), and it is only by the use of after images that we demonstrate that the meridians are not vertical.

The *field of action* of each of the elevators and depressors is shown in Fig. 137. Thus the line $A_1B_1C_1D_1E_1$ represents the boundary of the field of action of the superior rectus, $A_2B_2C_2D_2E_2$ that of the inferior oblique, $A_4B_4C_4D_4E_4$ that of the inferior rectus, and $A_5B_5C_5D_5E_5$ that of the superior oblique.

The same figure also illustrates the *action of the associates* and their common field. If, alongside of Fig. 137, which represents the field of fixation of the right eye, we place one representing the field of fixation of the left eye (which may be done by turning the figure end for end, so that O is on the left and P on the right of the figure), we shall see how the field of action ($A_2B_2C_2D_2E_2$), of the inferior oblique of the right eye agrees in all respects with the field of action of the superior rectus of the left eye. So also of the other associates (right superior rectus and left inferior oblique; right inferior rectus and left superior oblique; right superior oblique and left inferior rectus).

As we see from the figures each one of a pair of associates has an action *similar in all respects* to that of its fellow. Thus in the case of the right superior rectus and left inferior oblique, each elevates the eye, and the elevating action increases progressively as the eyes are carried to the right. Each again turns the eye to the left and tilts the vertical meridian to the left, and these two actions increase progressively as the eyes are carried to the left. In other words, each muscle of the pair moves the eye to which it is attached in just the same way and to the same extent as does its fellow.

298. Varieties of Conjugate Movements.—If in the summary of monocular movements given in § 295, we substitute for in and out, their equivalents, right and left, as the case may be, we have the following varieties of conjugate movements.

Lateral Movements

The eyes are carried to the *right* by the two right rotators, to the *left* by the two left rotators, the eyes being steadied in their course by the elevators and depressors. The vertical meridians remain vertical.

Elevation

The two eyes are carried *straight up* by the four elevators, being also steadied in their course by the external and internal recti. The vertical meridians remain vertical.

In moving *up and right*, the eyes are carried up mainly by the two right-hand elevators; to the right by the two right rotators assisted by the two left-hand elevators. The vertical meridians are both rotated to the right.

In moving *up and left*, the two eyes are carried up mainly by the two left-hand elevators; to the left by the two left rotators assisted by the two right-hand elevators. The vertical meridians are both rotated to the left.

In all movements of elevation the eyes tend to diverge somewhat.

Depression

The two eyes are carried *straight down* by the four depressors, being also steadied in their course by the external and internal recti. The vertical meridians remain vertical.

In moving *down and right*, the eyes are carried down mainly by the two right-hand depressors; to the right by the two right rotators assisted by the two left-hand depressors. The vertical meridians are both rotated to the left.

In moving *down and left,* the eyes are carried down mainly by the two left-hand depressors; to the left by the two left rotators assisted by the two right-hand depressors. The vertical meridians are both rotated to the right.

In all movements of depression the eyes tend to converge somewhat.

299. Disjunctive Movements of the Two Eyes.—These are—

1. *Convergence,* effected by an equal and simultaneous contraction of both internal recti. It may be combined with a parallel movement, the eyes being converged and also moved right, left, up, or down.

Owing to a combination of this sort, it may appear that one eye stands still while the other converges. Thus, if a prism is placed, base out, before the left eye, it will seem as if the latter alone turned inward in obedience to the converging impulse, while the right eye remained fixed. In reality, both eyes converge to an equal amount say 5°, then both rotate 5° to the right, the net result of the movement being that the right eye, although converging, looks straight forward, The same thing happens when an object which the eyes are following, is brought toward them, not in the median line, but in line with the right eye.

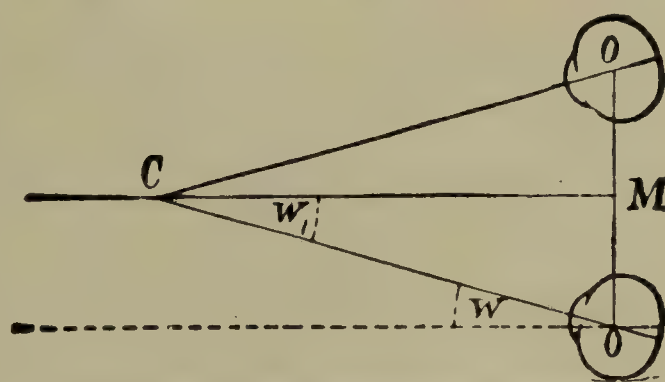

Fig. 138.—Representation of the Metre-angle

The nearest point on which the two eyes can be converged by the utmost effort is called the *convergence near-point,* and the furthest point to which they can be directed by the utmost relaxation of the converging muscles is the *convergence far-point.* The latter either lies at infinite distance, in case the visual lines are parallel, when the convergence is completely relaxed, or it lies within infinite distance (i. e., is positive), or beyond (i. e., is negative). The latter expression means that a certain degree of divergence is possible, which indeed is the rule for normal eyes. The difference between the maximum and minimum of convergence (far point and near point of convergence) is the *amplitude of convergence,* which, in cases where the eyes can be made to diverge beyond parallelism, is composed of a positive portion (convergence) and a negative portion (divergence; see infra). This way of regarding the convergence is like that which since Donder's time has obtained with respect to the accommodation (see § 179), and is intended to facilitate the comparison of these two functions which are so intimately related. With the same object in view Nagel has introduced the term *metre-angle.* Let *o o* (Fig. 138) be the base line, i. e., the line connecting the centres of rotation of the two eyes, and *M C* the median line. The angle of convergence is the angle through which the eye has to be deflected from the position of parallelism in order to be directed toward a point, *C.* That is, it is the angle w, or, what is the same thing, the angle w_1. Its magnitude is in inverse proportion to the distance of the object looked at (precisely as in the case of accommodation). The angle of convergence required in order to sight a point situated one metre in front of the eyes is called a metre-angle (mw or MA); and this constitutes the unit for the numerical denotation of the degree of convergence. When the object sighted is at 2 metres distance the convergence amounts to 0.5 MA; when the point is at 50 cm., the convergence is 2 MA, etc. This method of denoting the degree of convergence has the advantage that it parallels in all respects the expressions used to indicate the work done in accommodation. Thus, for a distance of 50 cm. a convergence of 2 MA and an accommodation of 2 D are required. The magnitude of the metre-angle, measured in degrees, varies in different persons, since it depends upon the length of the base line; on an average (with a base line of 64 mm.) it amounts to 1° 50′. The average normal convergence is 16 MA (convergence to a point 62 mm. from the base line, *o o*). The angle *oCM* or W_1, that each eye makes in converging on its fellow, is the *convergence-adduction.* Its usual maximum varies between 24° and 27°—rarely reaching 30°. It is, thus, not much over one-half the maximum *latero-adduction* or angle through

which the eye can be carried when turning inward, when making a parallel (conjugate) movement with its fellow (see § 296). For the ways of measuring the convergence see § 308.

2. *Divergence,* effected by a simultaneous and equal relaxation of both internal recti (passive divergence), assisted probably by a simultaneous and equal contraction of both external recti (active divergence).

The utmost amount by which each eye can diverge beyond parallelism (*divergence-abduction*) is not over 1.5°–2°. It is thus very much less than the *latero-abduction,* or the angle through which each eye can be turned out when moving in parallelism with its fellow (see § 296 and cf. §§ 308, 310).

3. *Divergence in a vertical plane,* effected by simultaneous contraction of the elevators of one eye and the depressors of the other.

In normal eyes this movement amounts to 1° at most.

300. Nerves and Nerve Centres for Ocular Movements.—The motor nerves of the eye are the oculo-motor, supplying the internal, superior, and inferior recti, inferior oblique, levator palpebræ superioris, sphincter pupillæ, and ciliary muscle; the trochlearis, supplying the superior oblique; the abducens supplying the external rectus; and the sympathetic, supplying the dilatator pupillæ. (see § 368). These nerves, except the last named, arise from three *nuclei* which preside over the actions of the individual muscles. These nuclei in turn are connected with various supranuclear centres, which effect the various co-ordinate binocular movements and are hence called *association centres.*

The nerve nuclei lie beneath the aqueduct and on the floor of the fourth ventricle on both sides of the rhaphe. The most anterior is the nucleus of the *oculo-motor nerve* (*III,* Fig. 139), which lies beneath the aqueduct in the region of the anterior corpora quadrigemina. It consists of several paired and one unpaired group of ganglion cells. And in a physiological sense it must be regarded as composed of a number of partial nuclei. But which separate group of ganglion cells belong to each muscle innervated by the oculo-motor nerve has not yet been precisely determined for man. For monkeys, in which the relations are probably similar, Bernheimer, on the basis of experimental investigations, has worked out the plan shown in Fig. 140. This confirms what clinical experience has already shown, that the nuclei of origin of muscles that are synergetic in action are in juxtaposition.

The fibres coming from the oculo-motor nucleus pass down through the crus cerebri, part of the fibres remaining on the same side, another part crossing to the other side (see Figs. 140 and 141). At the base of the brain the oculo-motor fibres become visible on the anterior border of the pons (*O,* Fig. 139).

The nucleus of the *trochlear nerve* (*IV,* Fig. 139) follows almost directly upon the posterior extremity of the oculo-motor nucleus, so that it might almost be regarded as the last partial nucleus of the latter (cf. Fig. 140). It lies beneath the posterior tubercles of the corpus quadrigeminum. The fibres which arise from it pass upward and backward, into the velum medullare anticum. In this they cross to the other side and then come out upon the base of the brain, winding about the crus cerebri and passing outward.

The nucleus of the *abducens* (*VI,* Fig. 139) lies pretty far behind the nuclei of the other two nerves, and in the immediate vicinity of the facial nucleus (*VII,* Fig. 139), a little in front of the striæ medullares. The nerve fibres arising from the nucleus pass downward between the bundles of the pyramidal tracts and become visible at the posterior border of the pons (*A,* Fig. 139).

All three nerves, after emerging at the base of the brain pass forward in the wall of the cavernous sinus (Fig. 142) and then enter the orbit through the superior orbital fissure.

The nuclei of the two sides are interconnected by transverse fibres, and the nuclei of the same side by the posterior longitudinal bundle, which also connects them with the facial nerve and through Deiters's nucleus with the labyrinth. They are, likewise, connected with the anterior corpora quadrigemina and either directly or indirectly by decussating paths with centres in the cortex. The situation of these centres and their interconnecting paths has not yet been certainly determined, although it seems likely that the middle frontal convolution is specially concerned in the production of conjugate

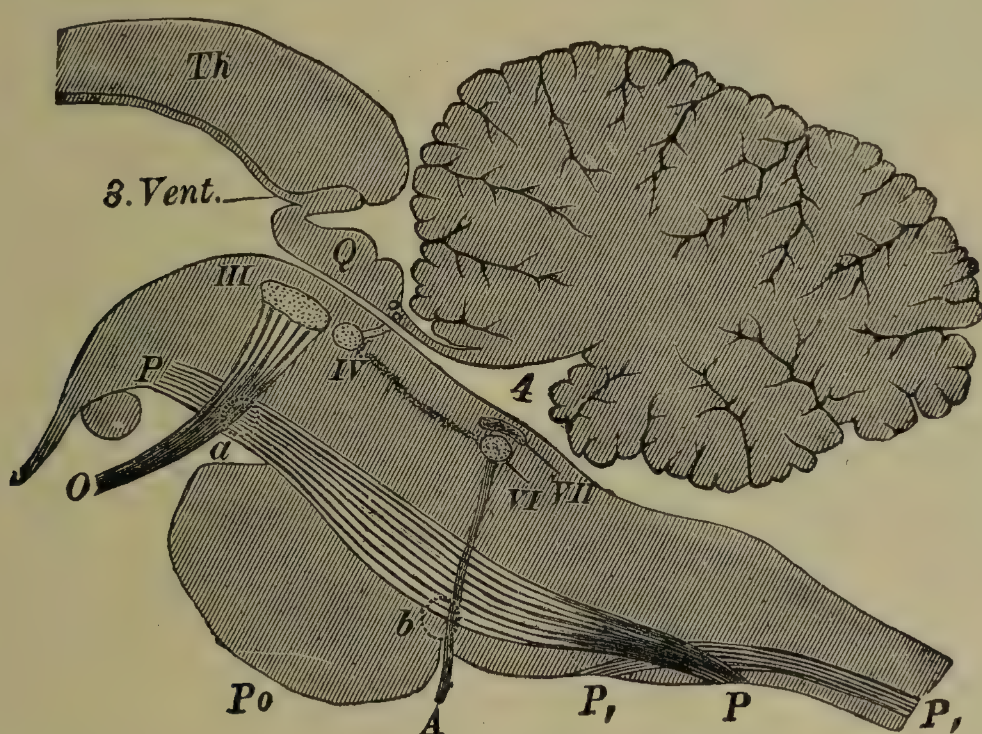

Fig. 139.—Nuclei of Origin of the Nerves of the Ocular Muscles. Schematic Sagittal Section through the Caudex Cerebri. Natural size.

The oculo-motor nucleus, *III*, lies beneath the anterior pair of tubercles of the corpus quadrigeminum, *Q*. The fibres coming from this nucleus run, converging downward, and emerge as a united nerve trunk, *O*, at the anterior border of the pons, *Po*. Directly behind the oculo-motor nucleus lies the nucleus, *IV*, of the trochlear nerve, from which the trunk of the nerve passes upward. The two points (drawn of light color in the figure) directly above it, and at the posterior margin of the corpus quadrigeminum, represent the cross sections of the trunks of the trochlear nerves as they decussate in the velum medullare anticum. The nucleus of the abducens, *VI*, lies upon the floor of the fourth ventricle, *4*, directly beneath the nucleus of the facial nerve, *VII*. The dotted band running from the nucleus of the abducens to the nucleus of the trochlear nerve represents the posterior longitudinal fasciculus connecting the nerve nuclei. The trunk of the abducens, *A*, emerges at the posterior border of the pons. *a* shows the site of a lesion which, through destruction of the oculo-motor nerve *O* and the pyramidal tract, *P P*, would result in a crossed paralysis of this nerve and of the extremities. In like fashion a lesion at *b* would produce crossed paralysis of the abducens and of the extremities. P_1 P_1, pyramidal tract of the other side; *Th*, optic thalamus.

eye movements. The frontal centre for eye movements is probably in close juxtaposition to centres for similarly directed movements of the head (§ 297). The angular gyrus which according to Bernheimer and others is a centre for ocular movements (lower visuo-motor centre), and other parts of the cortex which have been supposed to act in the same way are probably only way-stations in the path of centripetal (sensory) impulses going to the frontal centres (Wilbrand and Saenger). The subcortical centres for right and left rotation (*VI*, Fig. 141) are situated in the posterior longitudinal bundle close to the right and left abducens nuclei. The centre for convergence is thought to be close to the cell groups of the oculo-motor nucleus governing the action of the internal recti.

301. Production of Voluntary and Involuntary Movements.—The coordinate movements of the eyes are either voluntary or involuntary. The latter comprise movements produced by reflex stimuli, visual or labyrinthine, and those occurring in association with other motor mechanisms.

1. The *reflex movements due to visual impressions* comprise the movements by which the necessary adjustments to secure fusion are developed in infancy (*fusion movements*) and the altogether similar movements occurring later in life by which the visual impressions set up by an object forming its image on a peripheral part of the retina impel both eyes subconsciously to swing toward the object.

In *early infancy* the movements of the eyes are inco-ordinate, complete binocular fixation and association of movements not being secured until some time after birth. In the infant, light stimuli coming from the right of the eye and falling, therefore, on the left half of the retina cause a sensory impression which conveyed by reflex paths to the motor nuclei sets up an efferent impulse that automatically swings the eye to the right (see Fig. 141 and cf. Fig. 112). If the eye swings so far that it passes the fovea and falls on the right half of the retina, a counter-impulse is set up that swings the eye back to the left. At first probably the eye swings in pendulum-like fashion back and forth in this way until finally the image settles on the fovea. Its settling there will be determined very largely by the superior distinctness of the image when so placed.[12] This process of continual adjustment for foveal fixation, affecting necessarily both eyes, results in a condition of habitual binocular fixation. Similarly, if an object is close by, so that the eyes are looking past it, the stimulus affecting the temporal half of the retina in each eye will impel automatically both to turn nasally, i. e., to converge, until foveal fixation is secured in both. By continuous repetition of these acts the association of movement and the consequent fusion impulse become so established that they are not broken down even if one eye is covered or becomes blind.

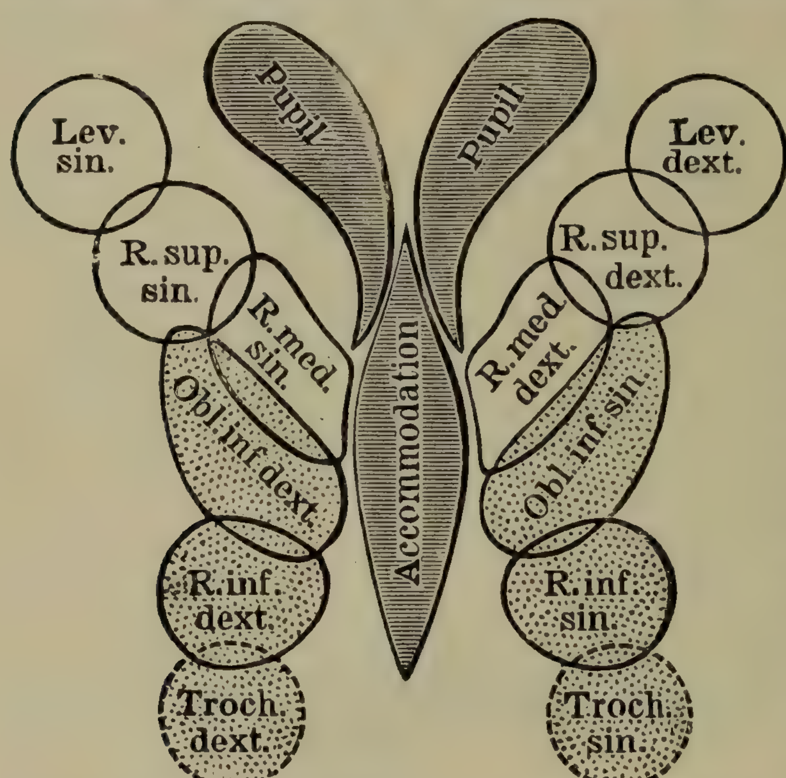

FIG. 140.—SCHEMATIC REPRESENTATION OF THE NUCLEAR REGION OF THE OCULO-MOTOR AND THE TROCHLEAR NERVES IN A MONKEY. Seen from above (After Bernheimer.)

The median nuclei are shaded; they are designed for the interior muscles of the eye, the two anterior (paired and composed of small cells) being designed for the pupil, the posterior (unpaired, large-celled median nucleus) for accommodation. The groups of ganglion cells situated on either side of the median nuclei form together the right and left main lateral nucleus. They comprise the region of origin of the nerves for the exterior ocular muscles, and immediately adjoining them behind is the nucleus of the trochlear nerve. All the fibres from the partial nuclei of the interior muscles, the levator and the superior rectus, most of those from the partial nucleus of the internal rectus, and a few of those from the partial nucleus for the inferior oblique, run to the eye of the same side; the remaining fibres from these latter two nuclei and all the fibres from the nuclei of the inferior rectus and the superior oblique run to the eye of the other side. See also Fig. 141.

When *binocular fixation is fully developed* quite similar movements take place in a reflex way, subconsciously directing first the attention, then the gaze to objects lying on the right or left (lateral rotation) or to objects nearer or further than the point one is looking at (movements of convergence and divergence). By many, a distinction is made between these subconscious movements of adjustment, which are quite certainly effected through the visual centres about the calcarine fissure, and the innate reflex movements, which develop and maintain fusion and which are attributed to impulses conveyed directly from the optic tract

[12] If we accept the bicerebral representation of the macula we should have still stronger reason why the swing stops when the fovea is reached. For when this occurs the image received on the fovea will, if the latter is connected with both hemispheres, send up a sensory impression to both, generating, thus, a bilateral opposing motor impulse. The eye receiving, thus, an equal impulse to go to the right and the left will come to a stop.

to the motor nuclei by way of the anterior corpora quadrigemina. But it would seem as if the relations indicated in Fig. 112, by which visual stimuli are transmitted successively to the visual centres about the calcarine fissure, the association centres and then the nerve nuclei, should serve to explain both classes of cases which are fundamentally alike.

In some cases when peripheral luminous stimuli are very intense, there is an involuntary reflex movement of the eye away from them (*avoidance* movement).

2. Reflex motor stimuli *emanating from the labyrinth* are conveyed to the nerve nuclei through Deiters's nucleus and the posterior longitudinal

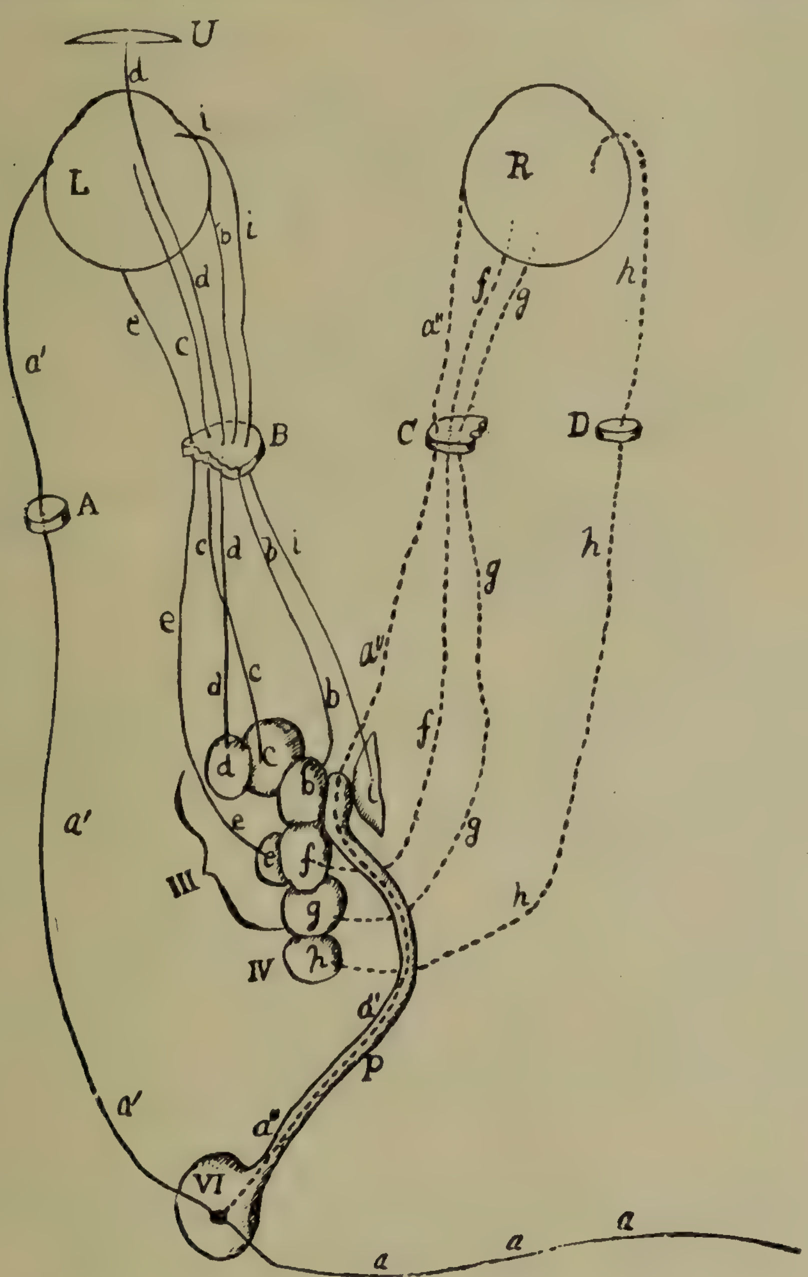

FIG. 141.—DISTRIBUTION OF THE NERVES RUNNING TO THE MOTOR APPARATUS OF THE EYE ACCORDING TO BERNHEIMER. Left Nucleus.

A, section of left 6th nerve; *B*, section of left 3d nerve, comprising only that portion which contains the direct fibres (from the left nucleus); *C*, section of right 3d nerve, comprising only that portion which contains the crossed fibres (from the left nucleus); *D*, section of right 4th nerve. *R*, right eye. *L*, left eye. *U*, left upper lid. *III*, left oculo-motor nucleus. *IV*, left trochlear nucleus. *VI*, association centre for left-hand rotation, connected with *III* by posterior longitudinal bundle, *P*. *a*, *a*, *a*, association fibres from right hemisphere to *VI*, dividing there into *a′*, *a′*, *a′*, fibres passing through left abducens nucleus to left external rectus and *a″*, *a″*, *a″*, fibres to right internal rectus. Latter fibres pass through *P* and *III* and thence through *C* to the muscle. These fibres subserve parallel movements only, i. e., rotation to the left in conjunction with the left external rectus. *b*, *b*, *b*, fibres to left internal rectus (subserving convergence only). *c*, *c*, *c*, fibres to left superior rectus. *d*, *d*, *d*, fibres to left levator palpebræ. *e*, *e*, *e*, fibres (few in number) to the left inferior oblique. *f*, *f*, *f*; fibres (many in number) to right inferior oblique. *g*, *g*, *g*, fibres to right inferior rectus. *h*, *h*, *h*, fibres to right superior oblique. *i*, *i*, *i*, fibres to left iris and ciliary muscle.

bundle. They apparently play an important part in maintaining fixation, especially when movements of the head are made.

3. A movement in *association with movements of the body muscles* is

chiefly seen in the uprolling of the eye that regularly takes place when the lids are closed (Bell's phenomenon). Cf. § 66.

4. *Willed movements* originate probably in the frontal cortex and descend through the internal capsule to the association centres and motor nuclei of the opposite side.

IV. Tests of Motor Anomalies

302. Tests.—The *object* of our tests is to determine how the eyes naturally stand with relation to each other in a given case.

Marlow and others believe that this is best determined when tests are made after *occlusion* of one eye for a number of days. But such prolonged occlusion, in the author's opinion, does not reproduce the actual motor relations as they exist in the given case

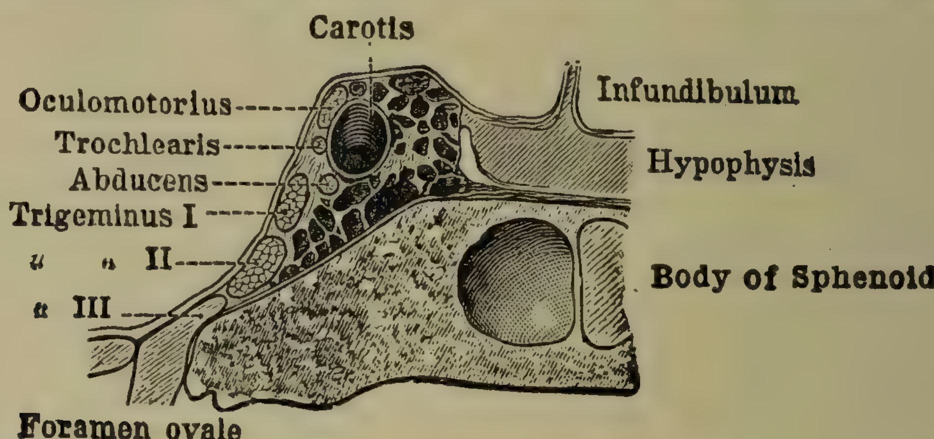

Fig. 142.—Frontal Section through the Sinus Cavernosus. (After Merkel.)
The sinus lies upon the lateral division of the body of the sphenoid bone. It is traversed by numerous septa which are attached also to the inner and lower wall of the carotid artery, which forming an S-shaped bend lies in the sinus. The outer and upper wall of the carotid artery is adherent to the wall of the sinus. Imbedded in this wall lie the three nerves supplying the eye mucles, and further below lie the first and second branches of the trigeminus. The third branch of the trigeminus leaves the cranial cavity through the foramen ovale.

but rather tends to make the eyes revert to their infantile state in which not being controlled by co-ordinate impulses they exhibit a varying imbalance.

Our main tests and what they show us are as follows:

I. Tests of monocular fixation, showing whether each eye by itself can perform fixation.

II. Tests of binocular fixation, showing whether the eyes always or usually perform binocular fixation or whether one deviates.

III. Tests of binocular vision, showing whether the patient has or has not simultaneous vision with the two eyes, and, if he has, whether he has binocular single vision, or else either diplopia or a tendency to diplopia, and how much tendency he has to fuse the double images.

IV. Tests of monocular movement, showing whether each eye by itself can move freely in all directions.

V. Tests of the co-ordinate movements of the two eyes and, particularly of:
(a) Parallel movements.
(b) Movements of convergence.
(c) Movements of divergence.

The best *test-object* is either a small flame on a dead-black background or a black spot on a large white surface. The spot should be 3 mm. wide for the distance tests and 1 mm. wide for the near tests.

In many of our tests we measure the deviation by correcting it with *prisms.* In so doing the apex of the prism is always placed in the direction in which the eye deviates, i. e., toward the temple in exophoria or divergent squint, toward the nose in esophoria or convergent squint, up before the right eye or down before the left in right hyperphoria, and in the opposite direction in left hyperphoria.

The prisms must be held in the proper position, i. e., with posterior surface perpendicular to the line drawn from the object of fixation to the eye (§ 145).

303. Test of Monocular Fixation.—*Fixation test.*—We make the patient cover one eye and then direct his other eye sharply at some well-defined object, like a pencil or a light, held straight in front of him. Inspection then will show, whether his fixation is central or eccentric, or whether, as in cases of a high grade amblyopia, he cannot fix at all.

304. Tests of Binocular Fixation.—1. *Inspection.*—We note by this any obvious deviation of the eyes, and whether this deviation is constant or intermittent, and monocular or alternating; also note any head-tilting.

A slight strabismus may escape notice. The diagnosis, however, will be made at once from the screen test.

On the other hand, it may appear that we are dealing with a strabismus when none exists. An *apparent strabismus* of this sort is most frequently simulated in the following way: The visual axis which joins the object of fixation to the fovea centralis does not in most eyes pass through the apex of the cornea, but the latter lies somewhat to the outside, or rarely to the inside of the axis (see § 163). If, then, the visual axes are parallel for distant vision, the corneal apices in the first case are divergent, in the second case convergent. If this deviation attains a pretty high degree, it becomes noticeable and simulates strabismus. Here, again, the screen test leads to the correct diagnosis; for when each eye is covered alternately it is apparent that the eye which is not covered remains steadfast in its position, and hence performs fixation properly.

2. *Screen Test.*—The patient is directed to look sharply at the test-object, and then a card is placed before the right eye and passed quickly to and fro from one eye to the other.

If there is *orthophoria,* neither eye will deviate when covered, and each consequently will remain steady when the screen is removed. If, however, there is *squint* or *heterophoria* of more than a degree or two, each eye when covered will deviate, and when uncovered will turn back into the fixing position. Thus, if there is either an esophoria or convergent squint each eye in turn will deviate in or towards the nose when covered, and will swing out again when uncovered.

In squint, whether non-comitant or comitant, the deviation of the squinting eye behind the screen is called the *primary* deviation; that of the non-squinting eye is the *secondary* deviation. Usually in paralytic squint the secondary deviation exceeds the primary (§ 312); in spastic squint it is less (§ 327), in comitant squint it equals it.

The *amount* of the deviation can be roughly determined by inspection, but is best measured accurately by placing a prism before the eye (apex in for esophoria and esotropia, apex out for exophoria and exotropia, apex up or down for hyperphoria), and increasing the strength of the prism until

the screen-deviation is first abolished and then converted into a movement in the opposite direction. The first prism that causes this over-correction, less 1*d* represents the amount of the deviation.

In paralysis and ordinary squint, the prism which abolishes the movement when placed before the deviating eye, measures the primary deviation; that which abolishes the movement when placed before the fixing eye measures the secondary deviation.

Having, thus, ascertained that a deviation is present and how much it is, we next inquire *if it is a squint or a heterophoria.*

To determine this, we make the patient look fixedly at the test-object with both eyes open, and then alternately cover and uncover one eye—say the right—leaving the other all the time uncovered (*method of binocular uncovering*). By thus covering the right eye we compel the left eye to fix, if it is not already doing so, and we allow the right eye to deviate, if it has any tendency to do so or if it is not already deviating.

In this case three conditions may obtain:

(*a*) There may be *heterophoria.* In this event, the left eye which is fixing already, will continue fixing when the right eye is covered, and the latter, which was fixing before being covered will now deviate. Then, when the cover is taken off again, the right eye will swing back into place and the left will still remain fixing—i. e., both on applying and removing the screen *but one eye* will move, and that the one that is being covered.

(*b*) There may be a *squint of the right eye.* In this case the left eye which was fixing before the right was covered will continue fixing afterward, and the right eye which was deviating before it was covered will remain deviating afterward. Then, when the right eye is uncovered again, the left eye will still remain fixing (because it is the eye that habitually fixes) and the right will still remain deviating (because it is the eye that habitually deviates). That is, both on covering and uncovering, *neither eye* moves, provided the cover is put over the squinting eye.

(*c*) There may be a *squint of the left eye.* Then the left eye, which was deviating before the right eye was covered, will now have to move into place in order to fix. As it moves into place, the right eye, which is behind the screen, will move out of place. Then, when the screen is removed, the right eye, which has thus become deviated, will move back into the fixing position, since it is the right eye which ordinarily fixes when both eyes are open, and the left eye will move out of the fixing position and back to its ordinary position of deviation. That is, both on covering and uncovering, *both eyes* move, provided in the case of a squint the screen is put before the fixing eye.

If repeated applications of the test show that sometimes the right eye squints, sometimes the left, we are dealing with an *alternating strabismus.*

3. *Corneal Reflex Tests.*—In *Hirschberg's test* the patient is made to look at a lighted candle placed one foot in front of his eyes. The observer stationing himself behind the candle notes the position of its reflection in the cornea of the squinting eye. If it is at the margin of the cornea, we assume that there is a squint of six mm. (i. e., about 45° of arc) if it is half way between the centre of the cornea and the margin it is three mm., etc.

In the *perimeter* test, the squinting eye (L, Fig. 143) is placed at the centre of a perimetric arc, and the good eye, R, is directed to look at a light, G, twenty feet distant and in line with the squinting eye and the zero point, o, of the arc. The observer carries another light, K, along the arc of the perimeter (keeping his own eye A, right behind the light), and watches the reflection of the latter as it sweeps across the cornea of the squinting eye. When the reflection occupies the centre of the cornea the position of the light on the perimetric arc indicates the angle of squint

Similar in principle is *Priestley Smith's tape method,* except that the good eye is made to follow an object along a tangent scale. The observer slips over one of his fingers a ring to which is attached two tapes. The patient holds one end of the first tape against his temple. The observer standing at such a distance that the first tape is taut, carries the second tape (which is graduated so as to form a tangent scale) out to one side with his other hand, directing the patient to follow the latter with his good eye until the reflection of an ophthalmoscopic mirror, through the peephole of which the observer is looking occupies the centre of the cornea of the squinting eye. This test has been advantageously modified by Holzer.

In all these methods allowance must be made for the amount of the angle gamma.[13]

No one of these methods at all approaches the screen test either in accuracy or in convenience. They do not show, or at least they show very imperfectly, the character of the squint (whether alternating or constant, periodic or continuous). Nor can they measure a vertical and a lateral deviation simultaneously, as the screen test does. Nor can they measure well, as it does, low degrees of squint, and they cannot measure heterophoria at all. Nor can they show, as it does, the varying amount of the deviation in different directions of the gaze.

Fig. 143.—Measurement of Strabismic Angle with a Perimeter.

305. Tests of Binocular Vision.—1. *Parallax Test.*—As we are passing the card from eye to eye in making the screen test, we ask the patient whether the object he is looking at seems to move or not. If it does, it means that he actually sees the object double, only instead of seeing the two images simultaneously he sees one after the other. Thus, if on uncovering the right eye the object seems to move to the right, it means that the right-eye image is to the right of the left—i. e., he has homonymous diplopia (escphoria). If, under the same conditions, the object had seemed to move to the left, he would really have crossed diplopia (exophoria). If it had moved down, he would have had a right hyperphoria; if up a left hyperphoria.

In order to be sure that the patient is actually fixing the object and not looking

[13] The angle γ is determined sufficiently well by placing the eye at the centre of a perimetric arc and making it fix the zero point of the perimetric scale. A light is then carried along the arc until its reflection occupies the centre of the cornea. The position of the light on the perimetric arc shows the amount of the angle κ (see § 163) which is nearly equivalent to the angle γ.

beyond it, so that he sees it projected against a background more remote, the test-object should be on the same plane as the objects immediately surrounding it.

The *amount* of the parallax is measured by the prism which, placed before the eyes, abolishes it. Thus, if a homonymous parallax is just abolished by a prism of 15 *d* base out, there is an esophoria of 15°.

The test is very precise, deflections of a quarter of a degree being measurable in this way. It is also accurate. It has further the advantage that vertical and lateral deflections, simultaneously present, can be simultaneously measured by a combined lateral and vertical prism. But its greatest value lies in the fact that it is a subjective test, which is made at the same time and under the same conditions as the objective screen test. If there is any real discrepancy between the subjective projection of images and the objective position of the eyes—in other words, if there is a faulty projection—it ought to show in a notable and constant difference between the findings of the two associated tests, the parallax and the screen. This, indeed, is occasionally the case.

2. *Red Glass.*—If there is much tendency to diplopia the mere interposition of a red glass before one eye will evoke it. In this case the test-object should be a light, of which, then, two images will be seen—one red, the other white. This proves that the patient has *binocular vision with diplopia.* If, with the red glass before one eye, the patient sees a single light which is white tinged with red, he surely has *no diplopia, and has binocular single vision.* If he sees a single light which is pure white or pure red he is suppressing the image in one eye (see § 291). In this case he has *no diplopia and has only monocular vision.*

The *amount* of deflection, and hence the amount of deviation, may be determined either by ascertaining the linear distance between the double images (allowance being made for the distance of the object from the patient), or by finding out what prism will be required to overcome the diplopia.

Placing a prism before one eye usually makes the other fix; hence, the prism which abolishes the diplopia measures the primary deviation if it is put before the deviating eye, and the secondary deviation if it is put before the other. Measurement of diplopia by prisms gives somewhat uncertain results, often affording an underestimate of the deviation. The reason for this is that when a prism is applied that corrects nearly all of the diplopia the patient may overcome the rest by involuntary effort.

3. *Maddox Rod.*—The Maddox rod consists of one or more little cylinders of glass set in a frame. (See Fig. 144.) If the instrument is held as shown in the figure and placed before the right eye, the patient, looking at the light, will see with this eye the light transformed into a luminous vertical streak. This streak is so different in appearance from the flame that the patient usually has no tendency to fuse it with the latter as he fuses ordinary double images. He hence allows a deviating eye to assume its natural position, as it would do behind the screen. If he has neither esophoria nor exophoria the streak will appear to run through the flame which in its unaltered form he sees with the left eye. If he has esophoria it will appear to the right of the flame; if he has exophoria, to the left of it.

To ascertain whether hyperphoria is present, the rod is turned at right

angles to the position shown in the cut and again placed before the right eye. If the patient has no hyperphoria, he will see a horizontal line bisecting the light. If, however, he has right hyperphoria the horizontal line will appear below, and if he has left hyperphoria will appear above the light.

The *amount of deviation* may be measured by the linear distance of the streak from the light measured on a tangent scale or by the amount of prism that will be required in order to make the streak coincide with the light.

The Maddox rod can also be used to indicate whether there is any *torsion* of the eye—i. e., any tilting of its vertical meridian to the right or left. If there is, the streak of light will appear tilted when the rod is set vertical, and the number of degrees through which rod has to be rotated to make the streak appear vertical will indicate the amount of the torsion. Thus, if set in the trial frame before the right eye it has to be rotated to 80° to make the streak appear vertical, there is an intorsion of 10°. Torsion can also be measured by Stevens's *clinometer*.

4. *Phorometer.*—The basis of this is *Von Graefe's equilibrium test.*

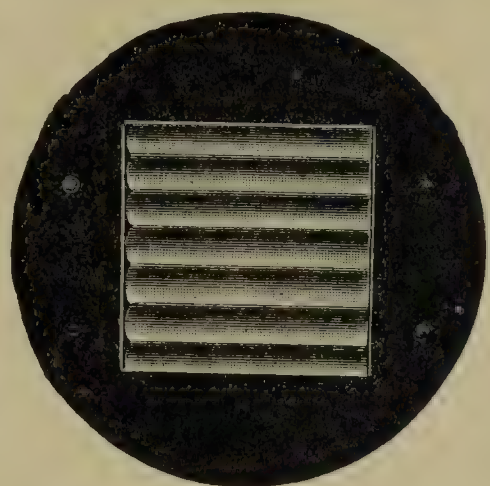

Fig. 144.—Maddox Rod.

Instead of a single thick glass rod, there is now used a series of thin rods usually made of red glass, placed side by side in a frame. If the rods are held horizontally the red line of light produced by them is vertical and vice versa.

This starts from the fact that a disturbance of equilibrium becomes manifest as soon as we render binocular single vision impossible. To accomplish this, we place before one eye, say the left, a prism with its base down and of such strength (4 d or more) that it cannot be overcome by any vertical divergence of the eyes (Fig. 145 A). The image seen by the left eye will then be above that of the right. If there is lateral orthophoria (i. e., no tendency to lateral separation of the eyes), this image will be straight over the other (Fig. 145 B). If, however, there is exophoria, the eye will diverge so that its image will be thrown inward (toward the right—Fig. 145 C). If there had been esophoria it would have been thrown to the left. The amount of deflection can be measured by the prism placed with apex in or out, that corrects it.

The equilibrium test can also be used to measure hyperphoria. In this case the prism is placed before one eye with its base toward the nose, thus separating the images laterally, and the difference of level, if any, is measured by a prism, base up or down.

The equilibrium test can be applied by putting a 6 *d* prism before one eye in a trial frame and *rotating* it until the images are vertical or horizontal as the case may be. Every 5° of rotation in this case denotes 0.5 *d* of deviation. Thus, if the prism, set before the right eye has to be rotated till its apex is at 80° to make the images vertical, there is an esophoria of 1 *d*; if the apex has to be placed at 15° to make the images level, there is a right hyperphoria of 1.5 *d*.

The phorometer (Fig. 146) consists essentially of two 3 *d* prisms, geared so that they can be rotated simultaneously but in opposite directions. The prisms are first set so that their bases both look inward; the patient then looking through the prisms will see two images of the test-object, that of the right eye being on the right side. If he has no hyperphoria the images will be on the same level. If he has right hyperphoria the right image will be lower; if he has left hyperphoria it will be higher. In case the images are

not on a level the prisms are rotated until they are; then the amount of hyperphoria can be read off from the index.

To measure for lateral deviations the prisms are rotated until the right hand one points down, the left up. Then, if there is no esophoria or exophoria, the images will appear directly over each other. If, however, there is esophoria the upper image will appear on the left; if there is exophoria it will appear on the right. In either of the latter events the prisms are rotated until the images do stand over each other, when the amount of the esophoria or exophoria, as the case may be, can be read off from the index.

5. *Bar-reading.*—If the patient while reading holds a pencil some three inches in front of the page, between it and his eyes, he will still, if he

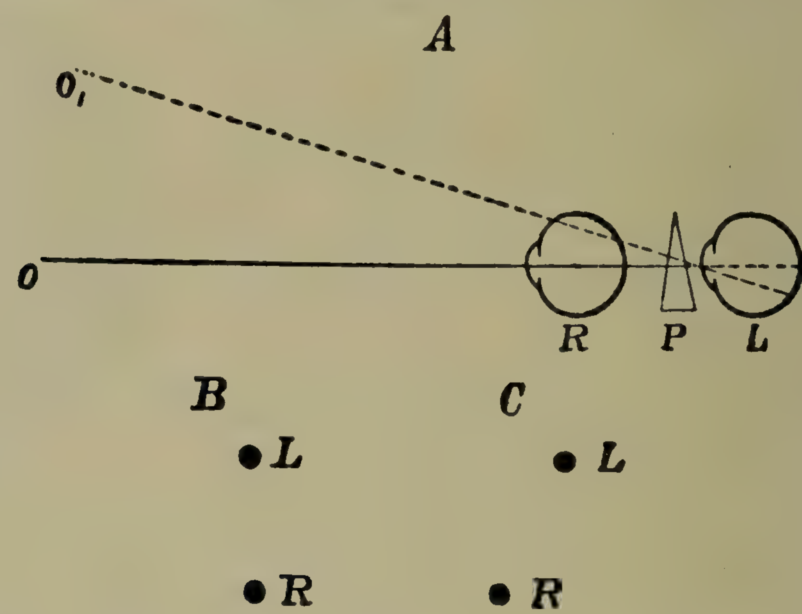

Fig. 145.—Von Graefe's Equilibrium Test.

has binocular vision, be able to read every word, because the letters hidden from the left eye will be seen by the right, and vice versa. If he has no binocular vision, the pencil obviously will cut out part of what he is reading. Hence, this test enables us to tell whether he is using one or both eyes (cf. § 246).

6. *Stereoscope and Amblyoscope.*—A patient who has binocular vision will be able to see two similar or slightly dissimilar images placed in the stereoscope and to fuse them into a single image. This is impossible for one who has only monocular vision. Different forms of stereoscope have been devised, both for determining the present or absence of binocular vision and also for training binocular vision and the fusion faculty. The same object is accomplished with the amblyoscope (Fig. 147).

The stereoscope and especially the amblyoscope will show both the patient's ability to perform fusion and to secure stereoscopic vision (stereopsis). In some cases, especially of long-standing squint, both fusion ability and stereopsis are entirely absent. In others they are present to a varying degree, and then can often be cultivated—the more so, the younger the patient.

7. *Hering's Test.*—In this the person tested looks through a tube at a vertical thread. Little balls are dropped alongside the thread either a little in front of it, or a little behind. A man having good binocular vision will tell promptly and correctly whether the balls fall in front of or behind the thread; he who has no stereoscopic vision

cannot do this. In Howard's test the patient looking through a window 20 feet distant judges whether a peg set close to another is behind or in front of it.

306. Tests of Monocular Movement.—These tests are made to determine whether either eye by itself can move properly—irrespective of whether it can keep pace with its fellow or not. By determining the limits of movement in every direction we delimit the *field of monocular fixation* (§ 296). The tests for this purpose are—

1. *Inspection.*—The eye is made to follow a well defined object and the observer notes when the eye begins to flag or waver. This, though a rough test, is sufficiently accurate in most cases. In moving outward the edge of

FIG. 146.—STEVENS' PHOROMETER.

the cornea should reach the outer and in moving inward should reach the inner canthus.

2. Measurement of the excursion on a *tangent curtain* or a *perimeter*. Here the patient is made to follow a sharply defined object (e. g., two closely set parallel lines on a bit of cardboard) which blurs the moment fixation wavers. The object is carried out from the centre, the eye following it. The exact point at which the object blurs is noted, and the corresponding amount of angular rotation read off from the scale of the perimeter or is plotted in the tangent-curtain card (§ 266).

3. Stevens's *tropometer*.

In measurements made by any of these methods it is absolutely essential that the head should be fixed and the eye alone move.

It is also important in most cases that measurements be taken in each one of the six diagnostic directions of the gaze.

4. *Pointing* (or *Projection*) *Test.*—In this the patient, closing one eye, looks at an object situated to one side of him with the other, then closes the latter and quickly points his finger at the place where he thinks the object to be. If in so doing he overshoots the mark, the muscle that moves the eye in that direction is too weak; if he undershoots it, the muscle is too strong (see §§ 313, 327). This test may also be made with the tested eye open, if the patient looking at a dot on a target, tries to touch with his finger the point on the back of the target that is precisely behind the dot (Landolt).

5. The *clinometer* or Maddox rod.—While these may be employed to test torsion in binocular vision (see § 305), they may also be employed to show deflections of the retinal

meridian in one eye even when the other is covered or blind. Occasionally in doubtful cases we can by thus proving the presence of torsion in one eye show that this eye is the one affected with paralysis or spasm (cf. § 327).

In general, the precise tests of monocular movement (tropometer, etc.) are of subordinate importance; first, because they do not show what we mainly wish to know, i. e., how the two eyes work together; and, second, because they are very variable and often fail to reveal an anomaly that is present. We repeatedly see cases in which one eye, when tested by itself, apparently moves well in all directions and yet shows a considerable and increasing deflection when the attempt is made to move it in conjunction with its fellow. Again, when we examine the eye in different days we may find wide variations in the range of its excursion, showing that the patient sometimes puts forth his full strength, some-

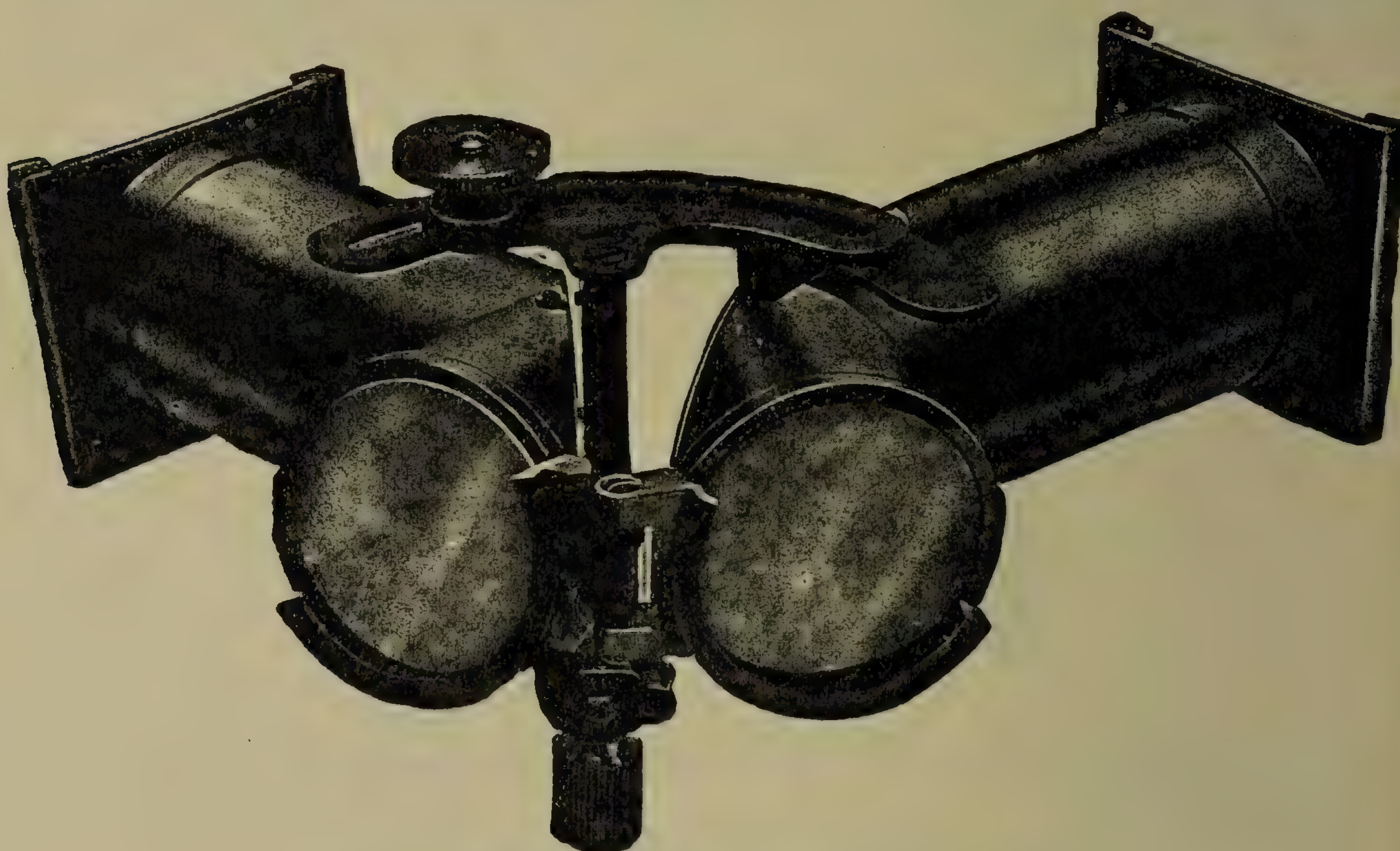

Fig. 147.—Worth's Amblyoscope, Modified by Black.

The amblyoscope consists of two tubes, bent and joined so as to admit of being converged and diverged through a considerable angle. In the Black modification here shown, one of the tubes can also be raised or lowered, an arrangement advantageous when the two eyes are not on the same level. A graduated arc may also be added, which shows the amount of the deviation or of the convergence and divergence employed. The further end of each tube carries a picture mounted on a translucent plate of celluloid. A mirror at the bent portion of the tube enables the patient looking through the eyepiece to see the picture with the corresponding eye. If he has binocular vision he will see two pictures, one with each eye, and by swinging the tubes in or out he will get these two pictures together and fuse them. By the use of simple devices the image formed by the better eye can be dimmed so that there is less tendency to suppression by the other.

times does not. In binocular movements whether he puts forth his full strength or not is of little importance, since if he fails with one eye he will equally fail with the other, and any disparity in movement between the two will still be apparent.

307. Tests of Binocular Movements; Tests of Conjugate Movements.—The tests of binocular movements comprise—

A. Tests of conjugate (parallel) movements.

B. Tests of convergence and divergence.

The former are the criteria for determining paralyses of the individual muscles (see §§ 319, 320, 321, 322). An essential requirement is that when they are applied the eyes shall be carried in each of the six diagnostic directions (§ 297). The tests of conjugate movements comprise—

1. *Excursion Test.*—In this we make the eyes follow a well-defined

object, e. g., a small white-headed pin, in each of the six directions, noting in each first, whether the fixing eye follows steadily or lags, and second, whether the other eye keeps pace with the first in all its movements or falls behind it or shoots ahead.

If in testing lateral movements we wish to avoid any possible error due to excessive convergence action, we either make the patient follow a moving object held not close to

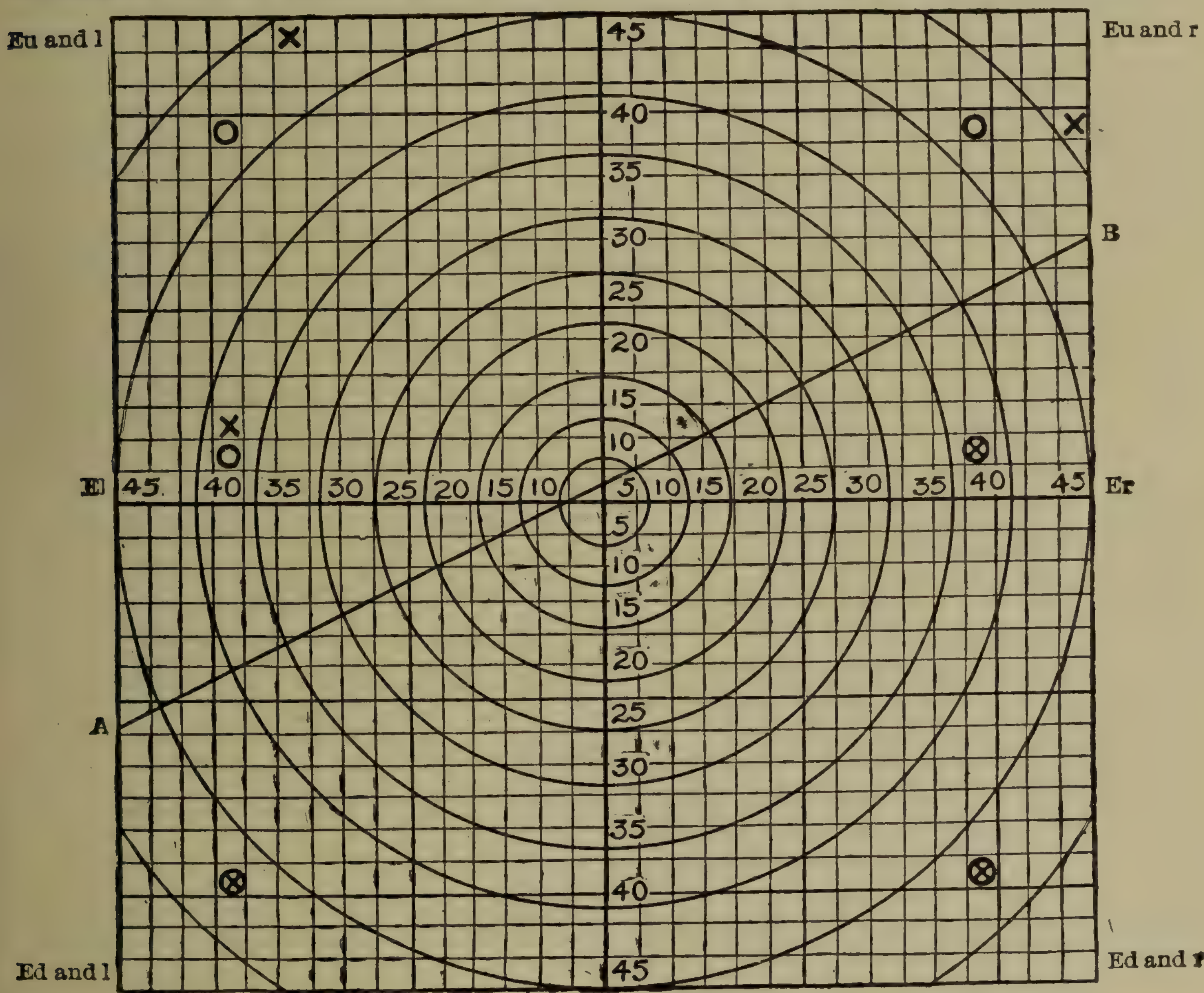

Fig. 148.—Double Images Plotted on Tangent Plane (from Posey and Spiller).

The reverse (white) side of the curtain tangent is here shown (seen from in front). (The other (black) side is turned toward the patient, who is placed 30 inches from it, with his eyes opposite the centre. A red glass is put before his right eye, and a small electric light is carried in the six diagnostic directions, "Eyes right" (Er), "Eyes up and right" (Eu and r), etc., the patient following it with his eyes but not moving his head. The position of the double images is marked by pins thrust into the curtain. In the case shown (paresis of the left superior rectus) the right eye (red) image is denoted by a circle, the left eye (white) image by a cross. There is a vertical (right) diplopia of 5° and a crossed diplopia of 2° when the eyes are directed two feet up and two feet to the left, and a simple crossed diplopia of 5° when they are directed up and to the right. Below the line AB there is single vision. It is found that the red image coincides with the actual position of the light on the screen; hence the right eye fixes, and the distance between the double images measures the primary deviation. (If the white image had coincided with the light the distance would have measured the secondary deviation.)

the eyes but a considerable distance (say 2 metres) away, or simply direct him to look to the right or left (Wilbrand and Saenger).

We may also note the excursions made by the eyes when the object looked at is fixed and the head is turned. In this case, however, we are testing not only the voluntary but also the reflex movements of the eyes (see § 301).

2. *Comitance Test.*—This is a variant of the preceding in which we slant a large card in front of the patient's nose in such a way as to hide the

moving object completely from one eye and yet not hide the eye from inspection. The patient is made to follow the moving object first with one eye, then with the other, while we observe in each case what the fixing and the covered eye do.

3. *Outlying Screen Deviation.*—In this we determine the screen deviation (primary and secondary) in the six directions.

These three tests are fixation tests determining the binocular excursions, the field of binocular fixation, and the deviation in different directions of the gaze. They are independent of binocular vision and can therefore be employed when there is suppression. The subjective counterpart of these objective tests, but which can be employed only when there is binocular vision, is the determination of

4. The *field of double vision* with the tangent curtain (§ 266). In applying this we place the patient 75 cm. from the curtain, facing the black side. A red glass is placed before the right eye, and the head is so adjusted that, when the eyes are directed at the centre pin, they are on a level with it and looking straight ahead. A small electric light is then carried over the curtain in the six directions, and the point where diplopia occurs in each meridian is noted by thrusting in a pin. The limits of the field of single vision are thus delimited. In the region in which diplopia occurs, the situation of the double images is similarly indicated. The patient sees two lights, a red and a white. The red image belongs to the right eye and the white image to the left. A black pin is thrust into the curtain at the site of the candle itself, and a light-colored pin at the site of the other image. This latter is evidently the false image and, if it is red, we know that the left eye is fixing; if it is white, we know that the right eye is fixing. The situation of the pins indicates the relation of the double images and their degree of separation.

In the great majority of cases the patient fixes with the eye not covered with the red glass, and, therefore, by shifting the red glass from one eye to the other, we can often get him to alternate fixation. We can then mark by a pair of pins the situation of the false image, when the right eye and the left eye respectively fix, and thus indicate the absolute and relative amount of the *primary* and *secondary deviation* for any given direction of the gaze. The plot which is formed by the pins on the curtain and which shows the limits of the field of single vision and the amount and kind of diplopia is transferred with the aid of the diagram on the back of the curtain to a similarly marked card—see Fig. 148. This forms a permanent record, which can be compared with a similar one taken at any future time.

308. Tests of Convergence and Divergence.—These tests are of special importance in the diagnosis of convergence and divergence anomalies (ordinary squint and heterophoria). They comprise—

1. A comparison of the *deviation for distance* and that for *near*, as determined by the screen, parallax, and phorometer. A deviation which increases notably in near vision, i. e., a directly periodic deviation, argues in general an anomaly of convergence; one which is much greater in distant vision, i. e., an inversely periodic deviation, argues an anomaly of divergence.

2. Ability to *overcome crossed diplopia*, however caused, or ability to

maintain fusion with the amblyoscope when the tubes are converged (test of converging power); or ability to *overcome homonymous diplopia* or to maintain fusion with the amblyoscope when the tubes are diverged (test of diverging power).

3. *Convergence near-point.*—This is determined by carrying a fine object up to the eyes and urging the patient to converge on it as much as possible. The distance of the nearest point at which he can do this is measured from a plane (spectacle frame) placed 11.5 mm. in front of the cornea. If we add 25 mm. to this we shall have the distance of the convergence near-point from the base line, i. e., the line connecting the centres of rotation of the two eyes.

Denoting the convergence near-point by the abbreviation *Pc* (Maddox) we may denote the distance from the base line by *PcB*. *PcB* is thus the distance *CM* (Fig. 138).

We may wish to determine the actual angle that the visual axes, *OC* (Fig. 138) make in converging on an object *C*, whether that is at the near-point of convergence or not. This is readily ascertained. If we determine by the method given in § 387, the distance *o o*, between the centres of rotation of the two eyes, and measure *CM* by the method given above, then for all distances of *C* from 5 to 22 cm. the relation

$$OCO \quad \frac{OO \times 50}{CM} + 3^{\circ}$$

will hold good (*OO* and *CM* being measured in mm.). Thus if we find *OO* to be 60 mm. and $CM = 120$ mm., then $OCO = \frac{60 \times 50}{120} + 3 = 28^{\circ}$. This is evidently double the angle W^1, by which each eye is converging, i. e., double the convergence-adduction of that eye (see § 299).

In certain cases of divergent squint in which the patient can never actually converge the eyes so as to secure binocular fixation, he will yet make the effort to converge and may do so down to a comparatively near point, the eyes visibly coming together, although not perfectly, until the object is brought to perhaps four or five inches from the eyes, when the latter suddenly give up the effort and diverge widely. The patient is then said to have a *relative* though not an absolute near-point of convergence.

4. *Prism-convergence* is determined by placing before the eyes successively stronger prisms, base out, until the patient can no longer unite the double images.

It is usually determined for distance only, although it may also be determined for near-point, especially when we are trying to ascertain the effect of prism exercise in relieving an exophoria.

The prism convergence can usually be run up to 30 *d* by practice. In the first trials the results vary greatly and the variations are evidently not due to any real variations in the muscular power. Hence, the test is of significance only when on repeated trials the patient cannot be made to overcome more than 6 *d* or at most 8 *d* and when even that small degree of convergence is made with effort and maintained with difficulty. In such cases, the convergence near-point pretty nearly always will be found to be remote.

5. *Prism-divergence* is the ability to overcome prisms, base in, while looking at a distant object. This ability, which varies between 1.5 and 4 *d* (usually between 2 and 3.5) *d* is a quantity, usually constant on repeated trials and seems to be a true measure of the diverging power. It is an important test.

309. How the Tests are made in Practice.—Usually not all the tests need be made, some being reserved for cases of special complexity. The following very complete routine suffices for all but the most unusual cases.

A. We measure the deviation for distance by

(1) The *screen* and (2) the *parallax* simultaneously. By the screen we also determine whether the deviation is a squint (monocular or alternating) or a heterophoria (see § 304).

(3) The *Maddox rod*, or (4) the *phorometer*.

B. We measure the *deviation for near points* (about 25 cm.) by

(5) the *screen* and (6) the *parallax* simultaneously and may supplement this with (7) the *phorometer*, though this is rarely necessary. In making the screen test, we ascertain by it whether the deviation is a squint or a heterophoria.

C. We determine:

(8) The *convergence near-point.*

(9) The *prism-divergence* (best done just after test 4).

(10) The *prism-convergence* (may be omitted if 8 is normal).

(11) The *excursion test.*

(12) The *field of binocular single vision.*

(13) The *comitance* test, supplemented if necessary by (14) the *screen deviation* in the six diagnostic directions of the gaze.

310. What the Tests Show in Normal Cases.—The motility may be regarded as normal if

(*a*) The screen test shows that there is no squint, and the other tests enumerated under A and B show that there is only a minimal amount of heterophoria (less than 1 *d* of esophoria or exophoria and less than 0.25 *d* of hyperphoria) for distance and not over $0-1.5$ *d* of exophoria for near.

(*b*) The convergence near-point is less than 8 cm.[14] from the base line connecting the centres of the two eyes.

(*c*) Prism-divergence is not less than 2 *d* and not over 4 *d*.

(*d*) Prism-convergence after a few trials can be run up to 7 to 10 *d* or more.

(*e*) The excursions of the eyes are normal in extent (see §§ 296, 306) and are comitant (§ 297) and the field of binocular single vision is normal —i. e., the patient having a red glass before one eye and following a light that is carried in all directions sees everywhere a single parti-colored light, each eye moves equally with its fellow without lagging, and there is no noticeable screen deviation anywhere.

In certain cases the diplopia tests (parallax, Maddox rod, phorometer) do not agree with the screen test, so that we have, for example, a well-marked divergent squint with an homonymous diplopia, etc. This is due to a peculiar kind of *false projection*, acquired usually in very early life. (See § 291.)

If there is suppression of the image in one eye, such as regularly takes place in long-established squint (see § 291), all the *tests for binocular vision* (parallax, phorometer, red glass, stereoscope, amblyoscope) will fail. Per

[14] In presbyopes it may be rather more than this; in children it should be not over 6 cm.

contra, the failure of these tests in any case argues the existence of suppression. In this event we must fall back on the screen test, and the determination of the field of monocular and binocular fixation and of the convergence near-point.

V. Etiological Classification of Deviations

311.—A deviation of the eyes may be caused by—

A. An anomaly of structure, insertion, or innervation of one or more of the *ocular muscles* (muscular paralysis and spasm, using these terms in their widest sense).

B. Anomalies of the *co-ordinate movements of the eyes* (the muscles per se being normal). These include—

1. Anomalies of *convergence* and *divergence*, either in the sense of excess or insufficiency.
2. Anomalies of *conjugate movements* comprising conjugate paralysis and spasm, nystagmus, and dissociated movements.

VI. Paralysis of the Ocular Muscles

312. Symptoms; Limitation of Movement; Deviation.—In paralysis of an ocular muscle the excursion of the eye in the field of action of the muscle that is paralyzed is diminished or entirely abrogated. If, for example, the external rectus of the right eye were completely paralyzed, the right eye could be brought only to the middle line and not beyond it to the right. When the paralysis is incomplete the deficiency in motility is, of course, less considerable, and often can be made out only by comparison with the other, or sound eye. In very slight paralyses the defective motility is not marked enough to be recognized at all with certainty. In these cases we must rely for our diagnosis upon the other symptoms which are, in fact, all dependent upon this limitation of motility. These symptoms are deviation of the paralyzed eye and secondary deviation of the other, false projection, diplopia, vertigo, and head-tilting.

The limitation of mobility is shown directly by a lagging of the eye when a conjugate movement is initiated within the field of action of the paralyzed muscle. Thus, if in paralysis of the right externus a point, o (Fig. 149), situated upon the right, should be the object of fixation, the left eye will be adjusted for it properly; but the right eye will not be turned sufficiently far to the right, and consequently its visual axis, g, will shoot off to the left of the object. The eye "squints" inward (*strabismus paralyticus*). This squinting takes place only when the eye is turned in the direction of the field of action of the paralyzed muscle, and becomes more pronounced the farther the eye is moved toward this side; but in all directions of the gaze in which the paralyzed muscle does not have to participate, the eyes stand in their proper position. By this fact paralytic squint is distinguished from ordinary or comitant squint which is present in all directions, of the gaze, and always to the same amount.

The measure of the deflection is determined by the angle s (Fig. 149), that the visual axis, g, makes with the line of direction, r, which passes

from the object to the retina through the nodal point of the eye, and which gives the place of the retinal image, *b*. This deflection of the squinting eye is known as the *primary deviation.* If now we make the right eye fix by putting a screen (*S*, Fig. 150) before the left eye, the latter turns strongly inward, so that the *secondary* deviation (s_1) of this eye is much greater than the primary deviation of the right.

This is accounted for as follows: When with both eyes uncovered the gaze was directed toward the right, the left internus and the right externus received the ordinary impulse for a movement to the right; but the right eye lagged behind the other in proportion as the right externus, owing to its impaired innervation, failed to answer to the

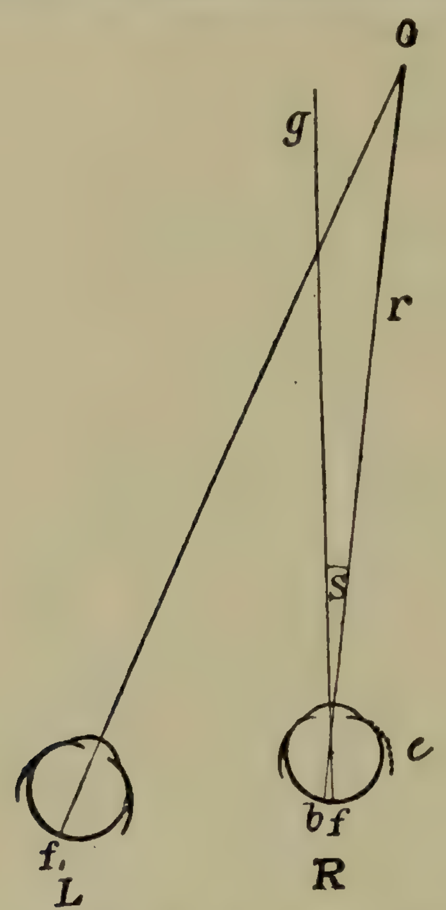

Fig. 149.—Primary Strabismic Deviation in Paralysis of the Right External Rectus.

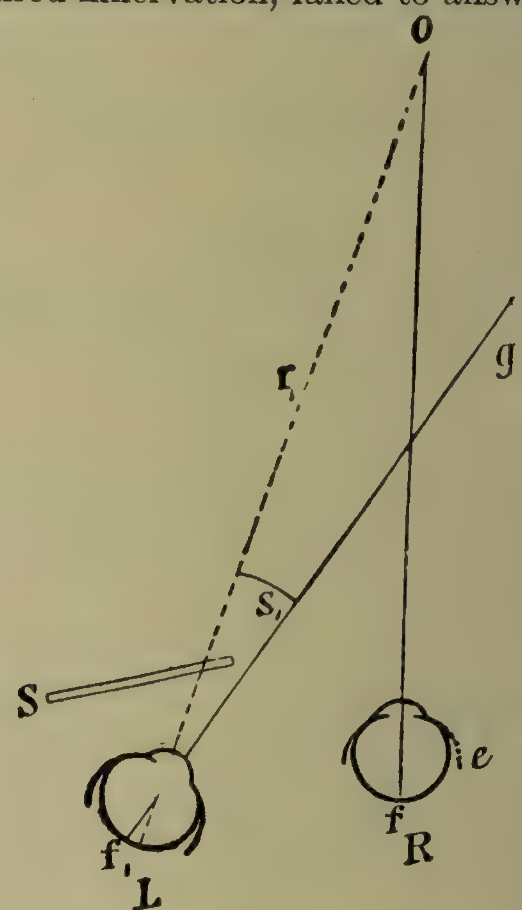

Fig. 150.—Secondary Strabismic Deviation in Paralysis of the Right External Rectus.

impulse. If now the left eye is covered, the patient is compelled to make fixation with the right. He tries now to turn this eye to the right, by sending into the right externus a very strong impulse of innervation, although in so doing he still obtains only a very slight effect. He cannot, however, innervate thus strongly the right externus alone, but can simply send out to both eyes a very energetic impulse for a lateral movement to the right. This impulse, therefore, also affects the left internus. But in the latter the impulse has its full effect, so that the left eye is drawn very strongly to the right (inward). Thus, while in primary deviation it is a mere question of lagging of the eye, secondary deviation is produced by a powerful muscular traction; therefore, the secondary deviation is greater than the primary. This point, is of importance, too, in distinguishing between paralytic and comitant squint, since in the latter the primary and secondary deviation are usually equal.

The secondary deviation of the sound eye shows itself not only when this is screened but also when for any other reason the *paretic eye takes up fixation.* This happens, for example, when the patient looks so far to one side that the view of the object is cut off from the sound eye by the nose. The sound eye in that case shows a sudden marked deviation (Fig. 153). Again it often happens, especially in congenital paralysis that the paretic eye is used habitually for fixation, so that the sound eye deviates in a spasmodic way, even when both eyes can see the object. Or, fixation may alternate between the two eyes, in which case the difference between the small primary and large secondary

deviation becomes at once apparent (see § 321). The amount of the primary and secondary deviation can be measured with prisms (§ 304) or by the double images on the tangent plane (§ 307). It must be admitted that in many cases there is much less difference between primary and secondary deviation than one would expect.

313. False Projection.—A patient seeking to locate solely with the aid of the paralyzed eye an object in the field of action of the paralyzed muscle will make wrong judgments as to its position. The object, in fact, seems displaced in the direction in which the paralyzed muscle if normal would move the eye. This leads the patient with the pointing test (§ 306) to shoot past the object which he is trying to touch. Again, if he tries to walk toward a point, using his paralyzed eye alone, he will go straight if the object is not in the field of action of the paralyzed muscle; otherwise, he takes a wavering and zigzag course, first bending his steps too far to the right, then recognizing his mistake and correcting it; then deviating anew to the right, and so on.

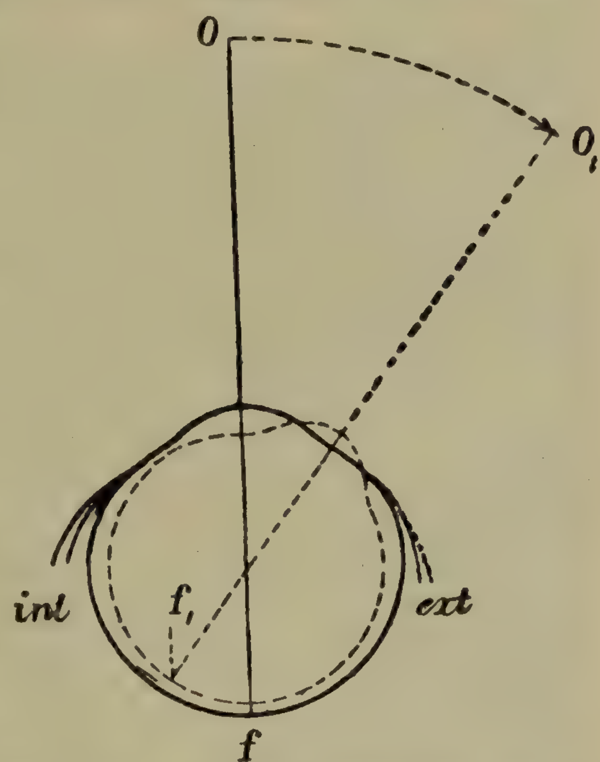

Fig. 151.—False Orientation in Paralysis of the Right Externus.

The explanation of this occurrence is similar to that which has been given for binocular diplopia (§ 288). The object is falsely localized because the patient is in error in regard to the position which his eye occupies. When (Fig. 151) the patient with his paralyzed right eye sights the object, *o*, which is placed somewhat to the right of him, i. e., looks at it in such a way that it forms an image upon the fovea centralis, *f*, he can accomplish this only by the strongest possible innervation of his paralyzed externus. Now, the ideas which we have with respect to the position of our eyes depend upon our sensations with regard to the innervation of the individual muscles. The patient, therefore, is compelled to believe that the right eye is standing in the most extreme position of right lateral rotation, like the eye represented by the dotted line in Fig. 151, because he has sent an impulse for a rotation to this amount into the right externus, and he cannot know that the latter, owing to the impairment of the conduction, only partially obeys this impulse. He hence proceeds upon the assumption that the right eye is turned very strongly to the right, and that consequently its fovea is at f_1; he is, therefore, also compelled to believe that the object whose image is formed at the fovea lies opposite f_1—i. e., at o_1—and he hence sees the object too far to the right.

314. Diplopia.—This occurs when vision is performed with both eyes simultaneously and the visual lines do not intersect in the point of fixation; it is the consequence of false projection of the paralyzed eye (see § 288).

The phenomena so far mentioned, such as restriction of motility, strabismus, false projection, and diplopia, make their appearance only when the eyes are moving in the field of action of the paralyzed muscle, and become more and more marked in proportion as the eyes are moved into this field. Thus, in complete paralysis of the right externus the double images and the strabismus make their appearance the moment the eyes pass to the right of the median line—in fact, often at a point some 15° or 20° to the

left of the median line. The more the gaze is turned in this direction, the farther apart are the double images and the more conspicuous is the strabismus. If we should have an incomplete paralysis (paresis) of the right externus before us, the double images and the strabismus would not show themselves until the eyes had been turned pretty far to the right, and in extreme cases would appear only at the very limit of the field of fixation.

315. Vertigo.—This may be excited by the diplopia, or may also occur when vision is performed with the paralyzed eye alone. The latter sees objects in their proper place so long as they occupy a direction of the gaze in which the paralyzed muscle is not called upon to exert itself. But as

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Fig. 152 A.

Fig. 152 B.

A. Head-tilting in case of congenital paralysis of the superior rectus with spasm of the inferior oblique. That the tilting was really due to the muscular deviation is shown by Fig. B. which indicates the result that immediately followed a complete tenotomy of the inferior oblique.

soon as the gaze is turned to the side representing the field of action of the paralyzed muscle, objects are located by the eye too far toward the same side, and the more so, the more the gaze is directed that way. Consequently, as the gaze passes from the region of correct to the region of false localization, objects appear to fly with constantly accelerated velocity in the direction in which the eye is moving. It is this apparent movement of the whole outside world that determines the development of vertigo. Vertigo, therefore, sets in whenever the patient feels himself called upon to move his eyes, and hence often even in walking upon a level floor, but still more in going up and down steps, in performing complicated manipulations, in doing work, etc. It makes the patient unsteady and timid, and even excites a tendency to vomit. This kind of vertigo is known as *visual* vertigo, and is distinguished from other kinds by the fact that it disappears at once when the paralyzed eye is covered. Most patients hit upon this fact themselves, and in walking keep the paralyzed eye either closed or covered. Another way of preserving themselves from visual vertigo lies in—

316. Head-Tilting.—A patient in whom, the right externus is paralyzed, keeps the head turned to the right. If he looks forward with his head in this position, both eyes are turned somewhat to the left, in which position the right externus does not come into play, and in which, therefore, paralysis of it does not make itself evident. The like is true of the other varieties of paralysis.

In paralysis of a right rotator, the patient turns his head to the right, so that in looking at objects ahead of him the eyes themselves shall be deflected to the left Similarly in paralysis of a left rotator he turns the head to the left. In paralysis of an elevator or depressor he usually tilts the head to one shoulder, as in this way he can more readily obviate the vertical diplopia (Fig. 152). In any case of combined vertical and lateral diplopia, tilting of the head toward the right shoulder will bring the right-hand image down. This is true whether the right-hand image belongs to the right eye (in case the diplopia is homonymous) or to the left eye (in case the diplopia is crossed). A vertical diplopia of 25° or more may be obviated in this way, the patient tipping the head toward the right or the left shoulder according as the right-hand or left-hand image is higher. The patient may combine this side-tipping with more or less lateral rotation and up or down tipping, but the side-tipping is the predominant and often the only abnormality of attitude. If with continuance of the paralysis such an attitude of the head becomes habitual, it may readily be confounded with wry-neck (particularly in congenital paralyses). If in such cases we set the head straight the up or down deflection of the paralyzed eye comes to light and enables us to make the correct diagnosis.

Through the muscular strain it produces and in other ways, the head-tilting may produce fatigue and discomfort.

317. Variation in Symptoms; Old Paralyses.—The symptoms of paralysis vary greatly in individual cases. In slight paralyses it often happens that little complaint is made of diplopia or vertigo, but, particularly if a depressor is affected, the patient has confusion and asthenopia in reading, book-keeping or other near work. Again, the characteristic symptoms of a paralysis are more unmixed and more pronounced the more recent it is. If the paralysis gets well comparatively soon, the symptoms that it produces disappear, and normal binocular vision is restored; if, on the other hand, the paralysis persists for a long time the symptomatic picture changes as follows: 1. The mistakes in orientation, particularly as they make their appearance in the pointing test, gradually cease; the patient learns by experience that the impulses of innervation for his paralyzed eye correspond to a much slighter action than those for the sound eye, and by taking account of this fact he once more forms a correct judgment of the situation of objects. 2. The diplopia may disappear through suppression, although such suppression is rarely complete and in many cases does not occur. 3. Contracture of the antagonists of the paralyzed muscle gradually sets in. Thus, in paralysis of the right externus, it is the right internus that becomes shortened; and so, while in a recent paralysis of the externus, the eye, when the gaze is directed straight forward, stands in the middle line, it afterward becomes drawn in more and more, and can no longer be brought up to the median position. The result of this contracture is that the deviation increases in amount and becomes apparent not only in the field of action of the paralyzed muscle, but also in the field of action of its antago-

nist and may even extend so far over the whole field of fixation that the paralytic strabismus gets to resemble an ordinary comitant squint.

Usually, however, even when this sort of extension has been carried quite far we can make out the characteristic increase of the deviation in some special quarter of the field and thus recognize its paralytic origin.

In some cases the contracture of the antagonist may persist after the paralysis itself is cured and may thus prevent the restoration of binocular vision.

318. Muscles Affected.—Paralysis may affect one or several muscles.

1. Paralysis of a *single muscle*, if acquired, affects most often the external rectus, less often the superior oblique or one of the muscles supplied by the oculo-motor nerve. In the latter case the superior or inferior rectus is most often involved. Isolated paralysis of the internal rectus is uncommon and that of the inferior oblique exceedingly rare. Congenital paralysis of the superior rectus is very common—so much so, indeed, that in all cases of paralysis seen in ordinary practice paralysis of the muscle is found nearly or quite as often as that of the external rectus. Slight pareses of the inferior rectus, probably congenital, are fairly frequent.

2. *Combined paralyses* comprise—

(a) *Oculo-motor paralysis*, complete or partial. In this regard we find all sorts of combinations. Complete *oculo-motor paralysis* presents a characteristic picture. The upper lid hangs loosely down (ptosis), and has to be drawn up with the finger to give a view of the eyeball, which is deflected strongly outward and somewhat down, because the two muscles not paralyzed—the external rectus and the superior oblique—draw it in this direction. The pupil is dilated and immobile (paralysis of the sphincter pupillae), and the eye is focused for the far point and cannot accommodate for near by (paralysis of the ciliary muscle). A slight degree of exophthalmus is present because three of the recti, which normally draw the eyeball backward into the orbit, have lost their tone. In *ophthalmoplegia interior* (or *interna*) only the interior muscles, i. e., the sphincter pupillæ and the ciliary muscle, are paralyzed—usually on both sides (cf. § 324).

(b) Paralyses of the muscles supplied by *two* or all *three* of the motor nerves of the eye. Of the manifold combinations seen, the two forms deserve special mention, namely *ophthalmoplegia totalis* and ophthalmoplegia exterior. In the former all the eye muscles in one or both eyes are paralyzed, so that the lids are drooping, the eyes are directed straight forward and are immobile, and there is dilatation of the pupil with abolition of the accommodation. In the more frequent *ophthalmoplegia exterior* the paralysis affects only the exterior eye muscles, the sphincter pupillæ and ciliary muscles being exempt (cf. §§ 324, 325).

319. Diagnosis.—The diagnosis of paralysis is based mainly on three points: The restriction of movement, the deviation produced, and the double vision. In this regard the cardinal fact to be kept in mind is that all these objective evidences of paralysis (and also the subjective symptoms of vertigo and confusion) increase in proportion as the eyes are carried into

the field of action of the paralyzed muscle—i. e. when they are carried into one particular direction of the gaze (§ 297). In order, therefore, to establish the diagnosis, it is essential to make the eyes move successively to the right, left, up and right, up and left, down and right, and down and left, and determine their behavior in each position.

This is obviously so, because when the eyes are carried into the field of action in which the paralyzed muscle is normally most effective, and both eyes attempt to proceed in that direction, both receive an equal nerve impulse (§ 312). The normal eye responds normally and proceeds on its way as usual; the paralyzed eye responds feebly or not at all, and therefore lags behind the other. In all other directions of the gaze the restriction in movement of the affected muscle is slight or affects only its subsidiary action (§ 293) and the lagging also is slight or nil. On the lagging depend the deviation and the diplopia, which consequently increase and decrease with it.

It is important to use the four *diagonal* and not the straight up and down directions of the gaze, since the latter do not afford sufficient diagnostic indications. In looking straight up, for example, four muscles are concerned in the elevation of the eyes, so that if the right eye lags down we can not be sure whether it is the right superior rectus or inferior oblique that is implicated. But, if the right lagging and the corresponding vertical diplopia increase in looking up and to the left, then we can be sure that there is a paralysis of the right inferior oblique A lateral deviation or diplopia increasing up and to the left, if considerable, must be due to a paralysis of a left lateral rotator (left external or right internal rectus). If slight it may be due to moderate paresis of a left rotator or to a paralysis of a right-hand elevator (right superior rectus, left inferior oblique) whose subsidiary (lateral) actions are most developed in the upper left quadrant of the field. Which is the case can be told at once by making the eyes move also up and to the right, when, if one of the elevators is concerned, a vertical diplopia should develop.

In making all the tests, it is essential, first, that the head shall be kept steady, so that the eyes alone move; and, second, that the patient be not allowed to tilt his head, as in this way we may obviate part or all of his deviation and diplopia (see § 316).

320. Determination of Deviation; Screen Test.—The deviation is determined by applying the screen test by alternate covering (§ 304), using for a test-object a dot on a card held first in the primary position, then carried in each of the six diagnostic directions. The diagnosis is made by this table given in § 322.

Because the paralyzed eye lags, it will deviate in a direction opposite to that in which the affected muscle would normally carry it—i. e., opposite to that in which the eye is moving (primary deviation). And because the non-paralyzed eye acts excessively when the paralyzed eye fixes, it will when screened deviate strongly in the direction in which the eyes are already moving (secondary deviation) Thus in paralysis of the right external rectus, when the eyes are carried into the field of action of this muscle, i. e., to the right, the right eye will lag, i. e., behind the screen it will deviate to the left, or lag inward. The left eye under the same conditions will when screened deviate to the right, i. e., will shoot inward. So in case of paralysis of the right internal rectus, both eyes will deviate out behind the screen, as soon as the eyes are carried to the left. On a similar principle, in paralysis of the right superior rectus, when the eyes are carried in to the field of action of this muscle, i. e., up and to the right, the right eye will drop and the left eye will shoot high up behind the screen. Moreover, since the right superior rectus is an adductor, the right eye when this muscle is paralyzed will not be adducted as well as formerly, and hence will tend to diverge. This divergence will be particularly marked when the eyes are directed up and to the left, because it is in this direction of the gaze that the right superior rectus is most effective as an adductor. This divergence, however, is compara-

tively slight, and may be masked or overcome by other conditions present. Thus, if there is a condition producing a constant convergence (esophoria), this may be great enough to neutralize in all parts of the field of fixation the divergence produced by the paralysis. In that case, instead of a moderate outward deviation increasing up and to the left we should find a moderate inward deviation diminishing,[15] in the same direction. The amount of the screen deviation can be *measured* by prisms (§ 304). A prism placed over the paralyzed eye measures the primary, and over the non-paralyzed, the secondary deviation.

321. Restriction of Movement; Excursion and Comitance Tests.— The restriction of movement is measured by the excursion and comitance tests (§ 307). With these the paretic eye will be seen to lag when it enters

A B

FIG. 153.—SECONDARY DEVIATION IN PARALYSIS. (After Posey.)

A, Paralysis of the left superior rectus with marked upshoot of the right eye, when the gaze is directed to the left. That the upshoot was due largely to the overaction of the right inferior oblique is demonstrated in B, which shows how the upshoot disappeared as a result of tenotomy of this muscle.

the field of action of the paralyzed muscle and will lag more and more the further it is carried into this field. Hence the following table[16] holds good:

	R. lags	Paralysis of	L. lags	Paralysis of
Er	In	R. external rectus	Out	L. internal rectus.
El	Out	R. internal rectus	In	L. external rectus.
Eu & R	Down	R. superior rectus	Down	L. inferior oblique.
Eu & l	Down	R. inferior oblique	Down	L. superior rectus.
Ed & r	Up	R. inferior rectus	Up	L. superior oblique.
Ed & l	Up	R. superior oblique	Up	L. inferior rectus.

We should be careful to observe not only the way the paralyzed eye lags, but the way in which the other eye behaves. For naturally the non-paralyzed eye follows the object and the other deviates, but when, either because the object is hidden from the non-paralyzed eye by the nose or for other reasons, the paralyzed eye takes up fixation, the non-paralyzed eye will be seen to make a sudden extreme movement (secondary deviation, spastic shoot) in the direction in which the eyes are moving (see § 312).

[15] Diminishing because it is there more and more neutralized by the increasing outward deviation produced by the paralysis of the superior rectus. Actually, the way in which such an anomalous inward deviation diminishes may be used to confirm the diagnosis.

[16] In this table, Er, El, Eu & r, Ed & r, etc., denote "when both eyes are directed to the right, left, up and right, down and right" etc.

The excessive movement is due mainly to overaction of the associate of the paralyzed muscle (see Fig. 153). For the same reason the diplopia increases when fixation is shifted from the non-paralyzed to the paralyzed eye (§ 312).

The results of the comitance test are best written in tabular form, thus (in a case indicative of paralysis of the right superior rectus):

	R. fixing	L. fixing
Eu & r	R. follows imperfectly; L. shoots higher	L. follows readily; R. lags. down.
Eu & l	R. normal; L. diverges slightly	L. normal; R. diverges very slightly.
Ed & r	R. and L. normal	R. and L. normal.
Ed & l	R. and L. normal	R. and L. normal.

We may also measure the restriction of movement produced by paralysis by taking the *field of fixation* with the perimeter or tropometer (see § 306) and noting how this field is diminished in any one of the six diagnostic directions. This is rarely necessary and moreover, does not afford as reliable indications as the other tests given. Nevertheless, whenever an eye apparently can not perform a certain movement if working with its fellow, it is well to see if it can perform it when the other eye is shut. In this way, cases of *false paralysis* of the abducens (really cases of convergence spasm) are detected (see § 330).

Another way to detect a false paralysis (e. g. one due to simulation or to inhibition of the will), is to place a strong prism before the eye with its apex in the direction of the defective movement and direct the patient to close the other eye and look at a light. If the eye really can move it will turn involuntarily toward the apex of the prism (A. Graefe.)

322. Measurement of Diplopia.—The diplopia being dependent on the deviation, has a character corresponding with the latter. Accordingly, paralytic deviations that produce an inward deviation are associated with an homonymous diplopia, those that produce an outward deviation are associated with a crossed diplopia, and those that produce a vertical deviation with a vertical diplopia (right or left diplopia, corresponding to a right or left hyperphoria). Moreover, the diplopia increases precisely as the deviation does. Hence all the remarks made with regard to the deviation are applicable to the diplopia as well.

The diplopia is best taken on a tangent curtain (§ 307).

The *types of diplopia* found in each variety of paralysis are shown in Figs. 154-165. The diagnosis by double images can be much better deduced, however, from the accompanying table, which also shows the types of screen deviation present.

The diagnosis of paralysis by the double images can also be readily and simply accomplished by an *analysis* which shows in succession, first, which group of muscles (lateral rotators, elevators, or depressors) is affected; next, which pair of associates in the group; and lastly, which muscle in the pair.

Thus, if we have a diplopia which is mainly lateral and which increases fast[17] in looking to the right or the left, the paralysis must affect a *lateral rotator*. If the diplopia increases to the right the paralysis affects a right rotator (right externus, left internus), if it increases to the left a left rotator (left externus, right internus). If the diplopia in either case is homonymous, it is the externus which is paralyzed, if the diplopia is crossed, it is the internus.

[17] See foot note p. 316.

If the diplopia is mainly vertical and this vertical diplopia increases fast in looking up, the paralysis affects an *elevator*. If then the vertical diplopia increases most in looking up and to the right, the muscle paralyzed must be a right-hand elevator (right superior rectus, left inferior oblique); if it increases up and to the left, the muscle paralyzed is a left-hand elevator (left superior rectus, right inferior oblique). In either case we can tell which eye and therefore which muscle is affected by remembering that the higher image belongs to the paralyzed eye (e. g., right-eye image is higher in paralysis of the right superior rectus and right inferior oblique).

Table of Diplopia and Deviation in Paralysis[18]

In the accompanying table DH, DX, DR, and DL denote respectively homonymous diplopia, crossed diplopia, right diplopia (vertical diplopia with the image of the right eye below), and left diplopia (vertical diplopia with the image of the left eye below). So also S, X, RH, and LH, denote a deviation in, out, right hyerphoria, and left hyperphoria. > > denotes "increasing progressively."

Paralysis of		Regularly (but not invariably) associated with
R. external rectus.......	DH and S >> greatly in Er.	
L. internal rectus........	DX and X >> " " Er.	
R. internal rectus........	DX and X >> " " El.	
L. external rectus.......	DH and S >> " " El.	
R. superior rectus.......	DL and LH >> " " Eu & r.	DX and X >> in Eu & l.
L. inferior oblique.......	DR and RH >> " " Eu & r.	DH and S >> in Eu & l.
R. inferior oblique.......	DL and LH >> " " Eu & l.	DH and S >> in Eu & r.
L. superior rectus.......	DR and RH >> " " Eu & l.	DX and X >> in Eu & r.
R. inferior rectus........	DR and RH >> " " Ed & r.	DX and X >> in Ed & l.
L. superior oblique......	DL and LH >> " " Ed & r.	DH and S >> in Ed & l.
R. superior oblique......	DR and RH >> " " Ed & l.	DH and S >> in Ed & r.
L. inferior rectus........	DL and LH >> " " Ed & l.	DX and X >> in Ed & r.

If the diplopia is mainly vertical and this vertical diplopia increases fast in looking down, the paralysis affects a *depressor*. If then the vertical diplopia increases most in looking down and to the right, the muscle paralyzed must be a right-hand depressor (right inferior rectus, left superior oblique); if it increases most in looking down and to the left, the muscle paralyzed is a left-hand depressor (left inferior rectus, right superior oblique). In either case we can tell the eye and therefore the muscle affected by remembering that the lower image belongs to the paralyzed eye.

In any case to tell the muscle that is paralyzed we have simply to remember that the *image formed by the paralyzed eye always lies on the side toward which the diplopia increases* (i. e., is the right-hand image when the diplopia increases to the right, the upper image when the diplopia increases upward, etc.).

And a rule to determine the kind of double vision produced by a given paralysis is —*the image belonging to the paralyzed eye is displaced in just that direction in which the affected muscle, if intact, would naturally move the eye.* Thus, in paralysis of the right superior rectus, which moves the right eye up and to the left and tilts the vertical meridian to the left, the image of the right eye is up (higher), deflected to the left (crossed, diplopia), and tilted to the left.

This rule shows us that in paralysis of the subsidiary adductors (superior rectus inferior rectus), we expect to find a crossed diplopia, and in paralysis of the subsidiary abductors (the two obliques) we shall find an homonymous diplopia; also in paralysis of the superior rectus and superior oblique an in-tilting and in paralysis of the inferior rectus

[18] This table serves for determining combined paralyses. Thus, if there is a DL (left hyperphoria) in Eu and r, diminishing to nothing as the middle line is approached and changing to a DR (right hyperphoria) in Eu and l, there is a paralysis of both superior recti; if there is a DR in Eu and l changing to DL in Ed and l, there is paralysis of the left superior and inferior recti.

Behavior of the Double Images in Paralysis of the Ocular Muscles

(The false image has a dotted outline.)

Left-sided Paralysis.

Fig. 154

Right-sided Paralysis.

Fig. 155

External Rectus.

Diplopia appears in looking toward the paralyzed side.

The lateral separation of the images increases as the paralyzed eye is abducted.

Fig. 153

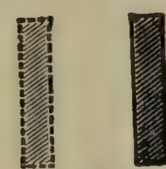

Fig. 157

Internal Rectus.

Diplopia on looking toward the sound side.

The lateral separation of the images increases in adduction of the paralyzed eye.

Fig. 158

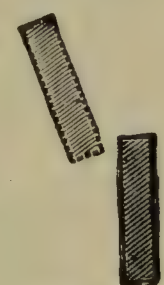

Fig. 159

Superior Rectus.

Diplopia on looking up.

The vertical distance between the images increases as the paralyzed eye is elevated and abducted.

The obliquity increases in adduction.

The lateral separation of the images diminishes as the eye is abducted; increases as it is adducted.

Fig 160

Fig. 161

Inferior Rectus.

Diplopia on looking down.

The vertical distance between the images increases as the paralyzed eye is depressed and abducted.

The obliquity increases in adduction.

The lateral separation of the images diminishes as the eye is abducted; increases as it is adducted.

Fig. 162

Fig. 163

Superior Oblique.

Diplopia on looking down.

The vertical distance between the images increases as the paralyzed eye is depressed and adducted.

The obliquity increases with the abduction.

The lateral distance between the images diminishes [as the eye is adducted, increases as it is abducted.

Fig. 164

Fig. 165

Inferior Oblique.

Diplopia on looking up.

The vertical distance between the images increases as the paralyzed eye is elevated and adducted.

The obliquity increases with the abduction.

The lateral distance between the images increases as the eye is elevated and abducted.

and inferior oblique an out-tilting of the false image. As before remarked, however, the lateral diplopia may be absent or reversed[19] and the tilting is often but little marked, so that neither is a safe guide in diagnosis.

The facts with regard to the *tilting of the double images* in paralysis are shown in Figs. 158-165 and may also be stated in the following tabular form:

Muscle paralyzed	Image tilted to	Tilting most marked in
R. superior rectus	Left	Eu & l.
L. inferior oblique	Left	Eu & l.
R. inferior oblique	Right	Eu & r.
L. superior rectus	Right	Eu & r.
R. inferior rectus	Right	Ed & l.
L. superior oblique	Right	Ed & l.
R. superior oblique	Left	Ed & r.
L. inferior rectus	Left	Ed & r.

Tilting of the double images may be determined by taking the patient's judgment as to the relative position of the two images of a candle; or, better, may be determined and also measured with the Maddox rod or the clinometer (§ 305), which will also indicate surely in which eye the tilting takes place if the test is made with each eye separately.

Frequently a patient affected with a paralysis of a depressor (inferior rectus or superior oblique), instead of seeing double images on a different level says that one *image is back of the other,* (see § 288).

323. Complex Cases.—The diagnosis as to which muscle is paralyzed is often difficult if the case is complicated. This occurs—

1. When several paralyses are combined, particularly in both eyes, and the paralyses are partly complete, partly incomplete. (See footnote, p. 314.)
2. When there is an esophoria or exophoria along with the paralysis. This latent deviation is converted into a manifest one when the paralysis sets in, as the latter renders binocular vision impossible, and the diplopia, homonymous or crossed, due to this manifest deviations then complicates the diplopia due to the paralysis (cf. § 320).
3. When fixation is performed with the paralyzed eye and particularly, when it is performed alternately by the paralyzed and sound eye (§ 312).
4. In old paralyses with contractures.

Even these complex cases can usually be unravelled, if care is taken to use all the methods of diagnosis (excursion comitance, screen and diplopia tests) in all the diagnostic directions of the gaze. In occasional doubtful cases of slight paralysis the use of the Maddox rod or the clinometer to determine which eye is pathologically tilted and which, therefore, is affected by the paralysis, may be of service.

324. Etiology; Site of Causal Lesion.—Paralyses of the ocular muscles are the result of a lesion which may be situated anywhere in the course of the nerve tract, from its very beginning in the cerebral cortex to its termination in the muscle itself. According to the site of the lesion, paralyses are distinguished into intracranial and orbital.

[19] It is to be noted that an homonymous or a crossed diplopia which increases to the right or left does not necessarily mean paralysis of a lateral rotator, unless the diplopia is great and increases rapidly. An homonymous diplopia which is of slight amount and increases moderately in looking to the right may mean (*a*) paresis of *any* abductor (externus, superior oblique, inferior oblique) of the right eye; or it may mean (*b*) paresis of *any* adductor (internus, superior rectus, inferior rectus) of the right eye if combined with an esophoria sufficient to neutralize the crossed diplopia that such paresis would naturally produce. So a crossed diplopia which increases to the right may mean paresis of *any* muscle of the left eye.

In *intracranial* paralyses the focus of disease lies within the cranial cavity. It may affect the centres of highest rank which lie in the cortex of the brain (cortical paralysis), or the association centres, or, lastly, the centres of lowest rank—i. e., the nerve nuclei upon the floor of the fourth ventricle (nuclear paralysis). The bands of fibres, likewise, that connect these centres may be affected, as may also be those fibres that run from the nuclei to the surface of the brain and unite there to form the nerve trunks (fascicular paralysis); and the nerve trunks themselves may be affected in their course along the base of the skull (basal paralysis).

Orbital paralyses are those in which the lesion is seated in the nerve trunk and its branches, after the entrance of the nerve into the orbit, or those in which the muscle itself is affected.

1. Lesions of the *centres of higher rank*, situated above the nerve nuclei (that is, the cerebral cortex, the association centres, and the fibres connecting these parts with one another and with the nuclei—regions which are all comprised under the name of intra-cerebral tracts), never cause paralyses of individual ocular muscles. If, therefore, isolated paralyses are present, lesions of as high a situation as this can be excluded. The only exception is *ptosis*, as this sometimes is met with as an isolated phenomenon in cortical affections. Otherwise lesions of the higher centres always cause *conjugate paralyses.*

2. Lesions of the *nuclei* on the floor of the ventricle (nuclear paralyses) produce for the most part paralyses of several ocular muscles. In this way there is developed what is known as *central ophthalmoplegia.* As a rule this is bilateral, for since fibres go from each oculo-motor nucleus to the muscles of both eyes, a lesion of the nucleus even though unilateral must also make its appearance in the muscles of both eyes. The ptosis, in comparison with the complete paralysis of the other muscles, is often conspicuously slight. In most cases the interior muscles of the eye (those of the pupil and accommodation) remain exempt from the paralysis—i. e., it is an *ophthalmoplegia exterior.* In the latter, the lesion is generally seated in the nerve nuclei. Ophthalmoplegia exterior, however, may also be due to fascicular, basal, or orbital lesions (cf. remarks under hereditary ophthalmoplegia exterior, § 325).

If there is an *ophthalmoplegia totalis*—that is, one in which all the muscles are paralyzed without exception—the site of the lesion may vary. In bilateral ophthalmoplegia we may be dealing with a nuclear paralysis, while a unilateral ophthalmoplegia totalis commonly originates in a lesion of the trunk of the nerve at the base of the brain, or even within the superior orbital fissure.

Ophthalmoplegia occurs either in an acute or a chronic form. *Acute ophthalmoplegia* develops within a few days and is combined not infrequently with drowsiness. It is observed after poisoning (by alcohol or lead, botulismus, carbon monoxide gas) and after acute infectious diseases (diphtheria, influenza, measles, etc.), in which case also it is probably a toxic affect that is in question. It occurs, moreover, under the form of a disease analogous to the poliomyelitis of children. In all these cases there is an acute inflammation in the region of the nuclei (Wernicke's polioencephalitis superior). Such cases may go on to recovery, but may also end in death by transfer of the process to the deeper motor nuclei of the medulla oblongata with consecutive respiratory paralysis. A similar affection associated with usually transient nuclear paralyses of the eye muscles is encephalitis lethargica. In *chronic ophthalmoplegia* the paralysis slowly attacks one muscle after another. The anatomical change that lies at the bottom of it is not an inflammation, but a gradual degeneration and atrophy of the gray substance. The most frequent cause is syphilis, and chronic ophthalmoplegia also occurs in tabes, progressive paralysis, disseminated sclerosis, myasthenic paralysis, and exophthalmic goitre. Chronic ophthalmoplegia is as a rule incurable. There are also cases of congenital ophthalmoplegia.

Ophthalmoplegia interior (paralysis of the sphincter iridis and the ciliary muscle) is a frequent sign of cerebral lues, and also occurs in the early stage of tabes and paresis.

Cases that develop acutely are generally referable to poisoning (by substances resembling atropine in action and in botulism). (Cf. § 380.)

Paralyses of *individual exterior muscles* also may arise as a result of lesion of the nerve nuclei. In this category belong, above all, the paralyses which appear in the beginning of tabes, and, though somewhat less frequently, in disseminated sclerosis, and which in most cases are of nuclear origin. Tabetic paralyses often disappear in a surprisingly short time, in spite of the progress of the causal disease. But they are apt to recur, and in many cases they remain permanently. By a nuclear lesion the abducens can be paralyzed at the same time as the facial, since the nuclei of these two nerves lie close together.

3. *Fascicular paralysis* due to lesion of the fibres between their point of departure from the nerve nuclei and their emergence at the base of the brain may be diagnosticated, if there is paralysis of the oculo-motor nerve of one side with simultaneous paralysis of the extremities of the opposite side (crossed paralysis) or such a paralysis combined with tremor of the opposite arm (Bénédict's syndrome). In this case, then a focus of disease must be assumed to exist in the lower part of the pedunculus cerebri (*a*, Fig. 139). Such a focus of disease causes injury both to the fibres of the oculo-motor nerve as they pass through the peduncle, so that the oculo-motor nerve of the same side is paralyzed, and to the pyramidal tract; but as the latter decussates below this point, the extremities are paralyzed on the side opposite to the lesion. Such a paralysis, however, might also be produced by a focus of disease at the base of the brain, if the disease were situated so near the peduncle as to cause injury to it. In analogous fashion a crossed paralysis of the extremities and of the abducens (and also of the facial) argues the existence of a focus of disease in the posterior part of the pons, or in the portions of the base of the brain adjoining it (*b*, Fig. 139).

4. Lesions at the *base of the brain* may likewise affect one nerve or several, and not infrequently affect both sides at once. The facts which with more or less probability lead us to infer the existence of a basal paralysis are: (*a*) When a whole series of cerebral nerves upon one side, such as those supplying the ocular muscles, the facial, auditory, the trigeminal, the optic, and the olfactory nerves, are paralyzed one after another. (*b*) When the affection of the trigeminus begins under the guise of a neuralgia, the latter not being observed in central paralyses. (*c*) When disturbances of sight of a certain sort are present. Among these are blindness of one eye without evidence of disease in the eye itself and without implication of the other, a condition which proves a lesion of the intracranial or intra-orbital segment of one optic nerve; also temporal hemianopsia which indicates a lesion of the chiasm. Homonymous hemianopsia occurs both in affections at the base of the skull through implication of the optic tract and also in lesions situated higher up. Hemianopsia from involvement of the tract is rare and should be assumed to exist only when there are other factors that argue the presence of a basal affection.

Basal paralyses of the eye muscles often result from fracture of the base of the skull. The abducens is particularly often paralyzed, as it runs close by the apex of the pyramid of the petrous bone and is readily injured by it (Panas). Because also of its closeness to the petrous bone the abducens is often implicated in otitic processes. Furthermore, transient abducens paralysis often occurs after lumbar anæsthesia.

To the paralyses of basal origin belong, too, most of the cases of *periodic paralysis* of the eye muscles. Such a paralysis which is regularly unilatural but may be bilateral, most frequently affects the oculo-motorius, occasionally the abducens. The attacks are ushered in by headache, which is often associated with vomiting (ophthalmoplegic migraine). After these symptoms have lasted for some days, the paralysis sets in. After some days or weeks this either disappears entirely or leaves a paresis of the muscles that lasts till the next attack. The disease often begins in childhood, and ends either in recovery or in permanent paralysis. Some of these cases are of a purely functional nature (hysterical), in others there is a basal lesion (a circumscribed exudate or small new growths that press on the nerve).

5. The diagnosis of an *orbital* paralysis must be made from the accompanying symptoms, when these are indicative of an affection within the orbit. Among such

symptoms are pain in the orbit, either spontaneous or excited by pressure upon the eyeball or upon the margin of the orbit, a tumor deep in, discoverable by palpation, protrusion of the eyeball, unilateral optic neuritis due to pressure of the optic nerve, and finally the evidences of antecedent trauma affecting the orbit.

325. Nature of Causal Lesion.—As regards its nature, the lesion may develop as a *primary* affection in the nerves or in their areas of origin, these being attacked by inflammation or by simple degeneration. Much more frequently, however, these structures suffer *indirectly* as a result of disease in their vicinity, such as exudates (especially in the meninges), thickenings of the periosteum, neoplasms, hæmorrhages, injuries, etc., by which the nerves or their nuclei are inflamed, compressed, or in some other way subjected to injury. Vascular changes producing lesions of the nerves are arteriosclerosis, aneurysm, occlusion and rupture of the vessels.

The *cause* of the lesion in the intracranial paralyses is most frequently syphilis in its later stages. This affects the eye muscles either directly or by the roundabout way of tabes and progressive paralysis. Diseases of the central nervous system causing paralysis are disseminated sclerosis, vascular changes with their consequences (embolism, hæmorrhages), injuries, inflammations, and tumors. Among acute infectious diseases, diphtheria is the most frequent cause of paralyses of the eye muscles, and among disorders of metabolism, diabetes.

The causes of orbital paralyses may be tumors, injuries, or inflammations of the orbit or its vicinity, and as regards the latter the transfer of inflammation from the accessory sinuses of the nose is particularly to be thought of (see §§ 815 and 886). In the majority of these paralyses, which from the accompanying symptoms we must regard as peripheral, none of these causes are discoverable. Since these paralyses which as a rule take a favorable course, are frequently referred by the patients to a cold, they bear the name of rheumatic paralyses.

Some cases of orbital paralysis are myogenous, i. e., due to inflammation, degeneration, or non-development of the muscles themselves. Probably here belongs the rare *hereditary ophthalmoplegia exterior* (Treacher Collins). This is an extremely chronic affection in which one after another the exterior muscles of the eyes including the levator become paralyzed. The interior muscles are never affected. It develops sometimes in adult life (Beaumont), sometimes in infancy (Wilbrand and Saenger), and, though usually referred to nuclear changes (cf. § 324) is most probably due to slowly progressive degeneration (abiotrophy) of the muscles themselves.

Myasthenia often begins in the eye muscles, usually under the form of ptosis. In this case the paralysis may pass over to the other eye muscles besides the levator but the interior muscles always remain exempt. The paralyses are characterized by the fact that they are made to increase rapidly by exhaustion. The diagnosis is made certain if at the same time there is feebleness of the muscles of the face, neck, and masticatory apparatus.

Congenital paralyses.—Congenital paralyses are quite frequent. Many forms have been described. The most common is paralysis, often incomplete, of the *superior rectus*. In some cases this is accompanied by congenital ptosis, but generally is uncomplicated. In these cases the patient often fixes with the paretic eye, the other showing a marked secondary deviation upward (upshoot) when adducted (Fig.53). 1 Paresis of

the *inferior rectus* is also common, although it is often very slight. Another less frequent congenital deviation is the *retraction syndrome* in which the external rectus is nearly or quite paralyzed, the internal rectus is paretic, and the eye when turned inward retracts more or less strongly into the orbit and at the same time turns up (sometimes down) while the palpebral fissure closes (false ptosis). It occurs usually in the left eye and is more common in women. A rare condition, either congenital or acquired in early childhood is *cyclic oculo-motor paralysis*. In the mydriatic phase this presents the appearance of a nearly or quite complete oculo-motor paralysis with ptosis. At intervals of every minute or less there occurs a miotic phase in which the drooping lid rises, the eye converges slightly, the pupil contracts, and through a spasmodic effort of accommodation the refraction increases several D. Attempted adduction of the affected eye may accentuate the miotic phase. The condition is most common in females (Rampoldi, Axenfeld and Schürenberg). Many cases of congenital paralysis are not really paralytic but are due to absence, abnormal insertion, or other structural anomalies of the muscles themselves. In cases of congenital paralysis diplopia can usually be elicited, and in any event true suppression seems to be very rare. Congenital paralyses are frequently associated with head-tilting, which may be noticeable early in infancy and in marked cases may simulate torticollis (see Fig. 152). They not infrequently form the starting point of regular convergent or divergent squint, which develops during the first two or three years of life (§ 336).

326. Course and Treatment.—The paralyses either set in suddenly or develop in an insidious manner. Sometimes relapses occur. The course is always chronic. Even in the most favorable cases six weeks and more are required for a cure, and many paralyses are absolutely incurable. Whether this is so or not depends mainly upon the cause which, therefore, must first of all be taken into consideration in making the prognosis. Another indication of the latter is afforded by the duration of the paralysis, since old paralyses, on account of the secondary changes that set in (atrophy of the paralyzed muscle and contracture of its antagonist), no longer hold out any prospect of a cure.

Treatment has first of all to take account of the causal indication. In this respect syphilitic and rheumatic paralyses afford the best prognosis. In the former, energetic treatment with iodides, mercury, and salvarsan is indicated. In the latter we give salicyl compounds, and employ diaphoresis. For symptomatic treatment electricity is often used (see § 120).

Besides the treatment of the paralysis itself, it seems also requisite, until the cure has been accomplished, to relieve the patient from the annoyance which the diplopia and the vertigo entail. Prisms are rarely useful, because even in slight paralysis the diplopia changes in amount whenever the eye is moved, so that at one moment is it over-corrected, at another under-corrected by the prism; and in more marked paralyses prisms do not suffice to compensate for the incorrect position of the eyes. Then there is no other way of relieving diplopia than to bandage the paralyzed eye, or, better still, to apply glasses which have an opaque plate for the paralyzed eye.

In old paralyses in which contracture of the antagonists has set in, we can get a result only by operative treatment (§ 992).

VII. Spasm of the Ocular Muscles

327. Symptoms.—Spasm of any muscle of one eye causes that eye to shoot ahead of the other when both are moving in some special direction. The symptoms of such spasm are—

1. *Excessive Movement.*—The affected eye shoots beyond the other when the eyes are carried in a certain direction, and its total range of excursion in that direction is greater than that of the fellow-eye.

2. The *deviation* thus produced, which can be made out by the screen and other tests, is greater if the sound eye is used for fixation than if the affected eye fixes—i. e., the *primary is greater than the secondary* deviation. (Cf. § 312.)

3. *Diplopia*, increasing as the eyes are carried in the direction of the field of action of the overacting muscle. This diplopia is precisely the same in character and in behavior as that which would be produced by a paralysis of the associate muscle in the fellow-eye,—i. e., the conditions will be as shown in the following table.

Spasm of right	Corresponds to paralysis of left	In either case			
		If L. eye fixes R. deviates greatly	If R. eye fixes L. deviates somewhat	Diplopia is	Deviation and diplopia increase as eyes are carried
External rectus...	Internal rectus...	Out....	Out....	Crossed......	To R.
Internal rectus....	External rectus...	In.....	In.....	Homonymous.	To L.
Superior rectus....	Inferior oblique...	Up....	Down..	Right[20].......	Up and to R.
Inferior oblique...	Superior rectus...	Up....	Down..	Right[20]......	Up and to L.
Inferior rectus....	Superior oblique..	Down..	Up....	Left[21]........	Down and to R.
Superior oblique...	Inferior rectus....	Down..	Up....	Left[21]........	Down and to L.

Note.—The conditions obtaining in spasm of the left eye (paralysis of the right) may be deduced by substituting right for left and vice versa.

4. *False Projection.*—If the pointing test is made in the manner indicated in § 306, by having the patient close the good eye and try to put his finger on an object situated within the field of action of the affected muscle, it will generally be found that in a case of spasm the patient undershoots his mark, just as in paralysis he overshoots it. Thus, a patient with a spasm of the right abducens who is trying to touch an object on his right will put his finger to the left of it. The reason for this is that the patient underestimates the distance that the eye travels, since he is aware only of the effort that he is putting forth in order to look at the object, and with an overacting muscle this effort is less than normal.

5. An *apparent movement* of objects looked at may be produced by the false projection, and this movement together with the diplopia may cause *vertigo*.

The appearances presented by a spasm of a muscle in one eye closely simulate those of a paralysis of the associate muscle in the other eye. Indeed the diagnosis between the two may be difficult, but in general can be made from the following:

Points in Common

One eye moves faster and further than the other when both are carried in some one particular direction; and this discrepancy between the position of the eyes and also the diplopia, false projection, and vertigo become more and more pronounced, the further the eyes are carried in that direction.

[20] I. e., vertical diplopia with the image of the right eye below.
[21] I. e., vertical diplopia with the image of the left eye below.

SPASM.	PARALYSIS.
The absolute excursion of the faster moving eye in the given direction is greater than normal: that of the other eye is normal.	The absolute excursion of the faster moving eye is normal in the given direction; that of the other eye is subnormal.
The total excursion of the faster moving eye in the given direction and in the direction opposite is greater than normal—i. e., the field of fixation is excessively large in one of its diameters.	The total excursion of the faster moving eye is normal, that of the other eye is subnormal—i. e., the field of fixation of the latter is contracted in one of its diameters.
Fixation is usually performed by the slower moving eye.	Fixation usually performed by the faster moving eye.
The amount of deviation may show great and sudden changes from time to time.	The amount of deviation remains constant or changes slowly and progressively.
If false projection is present, it occurs when the faster moving eye is used for fixation (the other being closed), and the patient undershoots the mark he is trying to touch.	False projection occurs when the slower moving eye is used for fixation, and the patient overshoots the mark he is trying to touch.

Furthermore, if an elevator or depressor is affected the eye which by the Maddox rod or clinometer shows an abnormal torsion is the affected eye.

328. Etiology.—Over-action of an ocular muscle may occur either because the muscle itself is excessively strong and over-developed (*structural over-action*), or because the tendon is inserted so close to the cornea that it exerts an undue leverage on the eye (*insertional over-action*). A marked example of insertional over-action is that produced by the operation of advancement.

True spasm is that form of over-action produced by excessive innervation. It may be either primary or secondary.

Primary spasm of the ocular muscles is rare. It may be produced by meningitis or other irritative affections of the brain, by reflex irritation from diseased teeth, by heightened pressure in the labyrinth, and by tetanus. Sometimes it occurs without known cause. The spasm produced by hysteria or occurring in connection with convulsive attacks does not usually affect the individual muscles but some conjugate movement of the eyes especially convergence (convergence-spasm, see § 330) or a parallel movement (conjugate spasm, see § 349, 351).

Secondary spasm is common. It comprises—

1. *Secondary spasmodic deviation of the sound eye when the paretic eye fixes* (see § 312). This deviation (see Table in § 327) always appears under the guise of a spasm of the associate to the paralyzed muscle.

2. *Spasm of a synergic muscle in the same eye.*—Thus in paralysis of the right superior rectus there may be a sort of compensatory spasm of the right inferior oblique. The resulting condition is quite like that produced by simultaneous paralysis of both superior recti, and differs only in that the diplopia in the left upper field fluctuates considerably and irregularly from one time to another. So, too, in abducens paralysis we sometimes find an associated spasm of the other two abductors (the two obliques) in the same eye. This causes the eye to move by zig-zag jumps a little further out than the externus alone can carry it. If the eye is above or below the horizontal plane, the amount of additional movement thus obtained may be rather large.

3. Irregular and varying *spasm in the antagonists of the paralyzed muscle.*

4. Permanent *spastic contraction* (*contracture*) *of the antagonist* of the paralyzed muscle (see § 317). In this case the paralysis may disappear and the contracture remain. When this happens the picture will be that of a spasm, pure and simple.

VIII. Divergence and Convergence Anomalies; Heterophoria and Squint

329. Nature of Convergence and Divergence Anomalies.—The diagnosis of a *divergence* or *convergence anomaly* is based on the following considerations:

When we look to the right we converge or diverge our eyes with the same facility as when we look to the left, and with nearly the same facility as when we look straight ahead. The deviation produced by a convergence or divergence anomaly, therefore, will remain about the same, no matter how the eyes are turned, provided that the object of fixation is kept at the same distance from the eyes. But since generally we use our convergence strongly when looking at near objects and do not use it when looking at distance, the effect of a convergence anomaly in producing deviations will be much greater in near than in distant vision. The opposite will be the case with a divergence anomaly. Convergence and divergence anomalies then are comitant in that the deviation changes but little as the eyes are carried to the right or left, and are periodic in that the deviation due to a convergence anomaly increases and that due to a divergence anomaly decreases, as the object of fixation approaches the eyes.

Nearly all cases of periodic comitant heterophoria and squint are produced by an anomaly of convergence or divergence. Nearly all cases of continuous comitant squint are produced by an anomaly of convergence combined with one of divergence.

Convergence-excess will produce an active, and divergence-insufficiency a passive, convergence of the visual lines (esophoria or esotropia). Convergence-insufficiency and divergence-excess will produce exophoria and exotropia. The ordinary form of esotropia is due to a convergence-excess combined with a divergence-insufficiency, and exotropia to divergence-excess combined with convergence-insufficiency.

330. Esophoria and Esotropia (Convergent Squint).—The varieties of esophoria and convergent squint are:

A. Non-comitant (*Paretic*) *Esotropia.*—The characteristic mark is that the deviation increases progressively as the eyes are carried either to the right or to the left. It is due to insufficiency of an abductor (externus or an oblique) or over-action of an adductor (internus, or superior or inferior rectus).

May also be due to insufficiency of an adductor, if combined with a comitant esophoria (see below) great enough to overcome the divergence due to the insufficiency. Thus an esotropia increasing to the right may be caused by insufficiency of the right internus (which would produce an exotropia diminishing to the right) combined with an esophoria of constant amount due to a convergence-excess.

In any case an esotropia which increases to the right must be due to insufficiency of some muscle of the right eye (or over-action of some muscle of the left). If it increases *fast* to the right, it must be due to insufficiency of the right externus. (See footnote, p. 316)

B. Esophoria and Comitant Esotropia.—The varieties of comitant inward deviations and the characters by which they are distinguished are as follows:

	Periodic Deviations.		Continuous Deviations.
	Convergence-excess.	Divergence-insufficiency.	Convergence-excess with divergence insufficiency.
For distance all tests (screen, parallax, Maddox rod, phorometer) show	Orthophoria or slight esophoria.	Esophoria marked.	Very marked esophoria or a marked convergent squint at all ranges (somewhat greater for near than for distance if the convergence-excess was the primary condition, and greater for distance than for near, if the divergence-insufficiency was the primary condition).
For near all tests show	Esophoria marked (much more than for distance).	Esophoria slight or none (decidedly less than for distance).	
Convergence near-point.	Normal (or abnormally close).	Normal.	Normal or excessively close.
Prism-convergence.	Normal.	Normal.	If it can be elicited (i. e., if there is no suppression), normal or supernormal.
Prism-divergence.	Normal.	Low (less than 1–1.5 *d*) or negative (i. e., insuperable homonymous diplopia for distance).	Low. Usually (in case of squint, always) negative (i. e., there is homonymous diplopia at all ranges, so that no prism, base in, is overcome, or there is monocular vision with suppression).
Primary and secondary screen deviation.	In all types deviation of one eye behind the screen, measured with prisms, equals precisely that of the other eye (distinction from non-comitant esotropia).		
Field of monocular and binocular fixation.	In all types total side to side excursion of each eye normal and inward rotations are not limited. Each keeps pace with the other in making lateral, vertical, or oblique movements (distintion from non-comitant esotropia). Screen deviation comitant.		
Field of binocular single vision.	Homonymous diplopia if present, not increasing (if anything, diminishing) as eyes are carried to right and left (distinction from non-comitant esotropia). Increases somewhat in looking down, diminishes in looking up.		

Convergence-excess is usually accommodative, i. e., the patient in trying to see well forces his accommodation and hence also forces his convergence. This happens (*a*) when he is trying to overcome a hyperopia or astigmatism; (*b*) when he is trying to see with the aid of an accommodation which has been reduced below normal by debilitating disease or by the instillation of homatropine or atropine; (*c*) when vision is rendered difficult by opacities in the media, reduced illumination, etc. Convergence-excess, sometimes permanent, may also be set up by the use of prisms, base out. As a secondary affection, convergence-excess occurs in consequence of a divergence-insufficiency which has lasted a long time.

Donders was the first to point out the relation between hyperopia and the convergence-excess that leads to the ordinary type of esotropia. He explained this relation in the following way: Hypermetropes have to make an unusually strong effort of accommodation to see distinctly; but as this effort, on account of the connection between accommodation and convergence, is possible only when combined with a strong impulse toward convergence, the latter function acquires a preponderance and a convergence-excess develops.

The rare *non-accommodative convergence-excess* may be due to hysteria or to some of the irritative conditions producing spasm in general (see § 328). But often the cause

remains unknown. Both accommodative and non-accommodative convergence excess may appear under the guise of a tonic, perhaps suddenly developing and suddenly varying *convergence-spasm.* Extreme cases of this sort are produced mainly by hysteria. In these at every attempt to perform fixation of an object, either far or near, both eyes at once assume a position of extreme convergence, the pupils at the same time being greatly contracted, and the accommodation thrown into a state of spasm. In other cases, the spasm occurs only at times and principally in fixing on a near object, especially one on which the patient is directed to converge as it is brought closer and closer to his eyes. In some cases the accommodation is not affected. The eyes may be so fixed in a condition of convergence-spasm that they can move out little or not at all. The condition is then sometimes mistaken for an abducens paralysis. It can be differentiated from the latter by the fact that when one eye is shut the other can then be rotated out freely (Lafon).

Divergence-insufficiency is usually secondary to a progressive convergence-excess (see § 331) although it may be primary. The most marked instance of the latter is the rather rare *divergence-paralysis.* This is characterized by homonymous diplopia and a convergent squint which, great in amount for distance, diminish steadily as the object of fixation is brought toward the eyes, until at some comparatively near point there is single vision with orthophoria. (For the way the deviation varies at different distances in divergence-paralysis see foot note, p. 328). Contrary to what takes place in a paralysis of the abducens, which this condition superficially resembles, the squint and the diplopia diminish or remain the same as the eyes are carried to the right and left, and the excursions of both eyes are of normal extent. The condition usually develops suddenly and is often due to organic brain disease.

331. An esophoria or esotropia may disappear if the conditions causing it are abolished or off-set. Thus an accommodative convergence-excess usually disappears if the causal hyperopia and astigmatism are corrected early enough; and it disappears in presbyopes who no longer accommodate excessively because they can not. Again, in some cases an esotropia disappears in youth, because in the growth of the face the orbits get more divergent so that the interni work less easily than before (Weiss).

This "out-growing" of the squint, which occurs about the time of puberty, is a cosmetic cure only. The amblyopia of the formerly squinting eye remains, and real binocular vision is rarely restored.

In other cases an esophoria increases. This is seen in the *development of esotropia,* which usually begins as an accommodative convergence-excess. The steps in this development are as follows:

(1) A child with a good deal of hyperopia or astigmatism, as soon as he begins to use the eyes much for near work, develops a varying, evidently more or less *spasmodic esophoria,* (convergence-spasm see supra) which is marked for near when his convergence is called into play, but which is little or not at all marked for distance.

(2) Little by little the deviation increases until binocular fixation for near becomes impossible, and the periodic esophoria is transformed into a *periodic esotropia.* This at first is intermittent and occasional, afterwards more and more constant. There is now more or less esophoria for distance

also, although always less than for near, and there is still binocular fixation for distance.

Careful tests at this stage often show that *the squint is associated with diplopia.*

(3) After this condition has lasted for a long time, perhaps for some years, the patient gives up binocular fixation for distance, so that he now has an esotropia for both distance and near (*continuous squint*). The deviation as measured by the screen, however, is still much more marked for near than for distance. Diplopia can still often be elicited by the tests. The condition is still mainly one of convergence-excess.

(4) Later still, the amount of deviation for distance begins to increase until ultimately the squint for distance equals that for near points, and both are absolutely greater than they were. This change is due to the superadding of a divergence-insufficiency on to the primitive convergence-excess; the conditions now, in fact, are those shown in the table as obtaining for *a convergence-excess and divergence-insufficiency combined.* The rotations of the eyes are still normal in amount, indicating that the muscles themselves are as yet unaffected.

At this stage, diplopia, if it can be elicited at all, no longer obtrudes itself upon the patient's notice nor causes confusion. In many cases it cannot be elicited by any tests, suppression having now occurred (§ 291).

(5) Later, *secondary muscular changes* (contracture of the internus, stretching and weakening of the externus) take place in the squinting eye producing an absolute reduction in the rotation outward and excess of rotation inward.

In some cases it seems that an esotropia may *develop from a divergence-insufficiency.* The steps in this case are, first, an increase of the esophoria for distance, then the superadding of a convergence-excess producing an esophoria for near. The result is, first a periodic convergent squint (esotropia for distance, esophoria with binocular fixation for near); later a continuous squint for all ranges. Until late in their course such cases are distinguished from those starting with a primitive convergence-excess by the fact that the deviation for distance always considerably exceeds that for near.

332. Exophoria and Exotropia.—The varieties of exophoria and divergent squint are:

A. Non-comitant (*Paretic*) *Exotropia.*—The characteristic mark is that there is an outward deviation which increases progressively as the eyes are carried either to the right or to the left. It is due to insufficiency of an adductor (internus or superior or inferior rectus) or over-action of an abductor (externus or an oblique).

It may also be due to insufficiency of an abductor if combined with a comitant exophoria (see below) great enough to overcome the esotropia due to the insufficiency. Thus an exotropia increasing to the right may be caused by insufficiency of the left externus (which would produce an esotropia diminishing to the right) combined with an exophoria of constant amount due to a convergence-insufficiency.

In any case, an exophoria which increases to the right must be due to insufficiency

of some muscle of the left eye (or over-action of some muscle of the right). If it increases fast to the right it must be due to insufficiency of the left internus. (See footnote, p.316.)

B. Exophoria and Comitant Exotropia.—The varieties of comitant outward deviation and the characters by which they are distinguished are as follows:

	Periodic Deviations.		Continuous Deviations.
	Convergence-insufficiency.	Divergence-excess.	Convergence-insufficiency with divergence-excess.
For distance all tests (screen, parallax, Maddox rod, phorometer) show	Orthophoria or slight exophoria.	Exophoria marked.	Marked exophoria or marked divergent squint at all ranges (somewhat greater for near than for distance if the convergence-insufficiency was the primary condition, and greater for distance than for near if the divergence-excess was the primary condition).
For near all tests show	Exophoria very marked (much more than for distance).	Exophoria decidedly less than for distance.	
Convergence near-point.	Remote (more than 9 cm. from base line connecting centres of eyes).	Normal.	Remote. In case of actual squint, often none (patient may try to converge but cannot secure binocular fixation at any distance).
Prism-convergence.	Low and accomplished only with effort and difficulty.	Normal.	Low. In case of actual squint negative (i. e., there is either crossed diplopia at all ranges so that no prism is overcome, or there is monocular vision with suppression).
Prism-divergence.	Normal.	Abnormally great (over 4.5*d*).	If it can be elicited (i. e., if there is no suppression) it is much greater than normal.
Primary and secondary screen deviation.	In all types deviation of one eye behind the screen, equals precisely that of the other eye (distinction from non-comitant exotropia).		
Field of binocular fixation.	In all types total side to side excursion of each eye normal and unless secondary muscular changes have occurred outward rotations are not limited. Each eye keeps pace with the other in making lateral, vertical, or oblique movements (distinction from non-comitant esotropia). Screen deviation comitant.		
Field of binocular single vision.	Crossed diplopia not increasing as eyes are carried to right and left (distinction from non-comitant exotropia). Increases somewhat in looking up, diminishes in looking down.		

Convergence-insufficiency is often *accommodative,* that is, it occurs in those who do not require to use their accommodation in the normal way. The accommodation and convergence being intimately associated, the disuse of one means the disuse of the other. A myope, for example to see objects near by, needs to use either little or no accommodation; consequently with him the impulse to converge is too weak. This disuse of the convergence often shows itself at first simply by the presence of a large amount of exophoria for near without any recession of the convergence near-point, but later it develops into a true convergence-insufficiency.

Accommodative convergence-insufficiency occurs in myopes who are not using concave glasses, and may also occur (from sudden relaxation of the accommodation) in hyperopes and presbyopes when putting on convex glasses for the first time. A somewhat similar relaxation of the convergence is found in those who for any reason (presbyopia, marked hyperopia, anisometropia, etc.) can not see well for near; and in this case the convergence-insufficiency may be relieved by convex glasses.

The relation between myopia and the form of accommodative convergence-insufficiency that leads to exotropia was first pointed out by Donders.

A *non-accommodative* convergence-insufficiency—i. e., one which develops without any reference to a refractive error—occurs quite often in hysteria, traumatic neuroses, neurasthenia, anæmia, and debilitated conditions, however caused; also in connection with intra-nasal disease, and not infrequently seems to occur in those who are apparently healthy. It is also often secondary to a divergence-excess (see § 333).

An extreme variety of convergence-insufficiency is *convergence-paralysis*. This is characterized by a crossed diplopia and an exotropia which increase progressively as the object of fixation is carried toward the eyes.[22] The deviation and the diplopia do not increase either to the right or to the left. Movements of each eye outward and inward are normal. The convergence reaction of the pupil is absent. Convergence-paralysis occurs in tabes, multiple sclerosis, and other central nervous diseases, either organic or functional. It may be associated with paralysis of accommodation (see § 380).

Divergence-excess is common as a primary condition. It also occurs as a regular secondary effect of a progressive convergence-insufficiency (see § 333).

333. A convergence-insufficiency, especially if due to temporary causes, may disappear; or it may increase by the superadding of a divergence-excess. A divergence-excess often remains stationary for a number of years, but it may also increase by the superadding of a convergence-insufficiency.

These combined forms (convergence-insufficiency with divergence-excess) constitute the ordinary continuous *divergent squint.* This develops most often from a convergence-insufficiency, the steps of development being as follows:

(1) Marked exophoria for near with at first a normal near-point of convergence; orthophoria for distance and normal diverging power (*periodic exophoria*).

(2) Beginning divergent squint for near with recession of the convergence near-point; beginning exophoria for distance with increase in the prism-divergence (*periodic divergent squint*). This squint, at first intermittent, is afterwards constant.

(3) Marked increase of the exophoria for distance with gradual transformation into squint (consecutive divergence-excess) converting a periodic into a *continuous squint.*

(4) *Muscular changes* (contracture of the externus, stretching and weakening of the internus) producing absolute impairment of rotation inward and excess of rotation outward.

A divergent squint also quite often develops from a divergence-excess; the steps in the development being as follows:

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[22] In a complete convergence-paralysis the amount of deviation (exotropia) at 1 metre is 3°, at 0.5 metre 7°, and at 0.25 metre 15° more than for distance. So in a complete divergence-paralysis the amount of esotropia at 1 metre, 0.5, and 0.25 metre, respectively, is 3°, 7°, and 15°, less than that for distance. In incomplete paralyses the differences are not so great as these.

(1) Marked exophoria for distance with excessive prism-divergence; exophoria for near, little or none, and near-point of convergence normal (*periodic exophoria*).

(2) Beginning exotropia for distance with crossed diplopia and still further increase in the prism-divergence; beginning exophoria for near, but with convergence near-point still normal (*periodic exotropia*). This squint is at first intermittent, afterwards constant.

(3) Marked increase of the exophoria for near and gradual recession of the convergence near-point (*consecutive convergence-insufficiency*), producing a squint for near, which, however, is less than for distance.

(4) Marked divergent squint which is of about the same amount for both distance and near (*conversion of the periodic into a continuous squint* by the superadding of a convergence-insufficiency to the primitive divergence-excess). Diplopia still often present, but now usually but little noticed by the patient. The rotations of the eye inward and outward are still about normal.

(5) *Muscular changes* (weakening of the interni, contracture of the externi) causing impairment of rotation inwards and excess of rotation outwards.

334. How Squint Develops.—As seen from § 331 and § 333 an esotropia usually develops out of a simple convergence-excess to which is added a divergence-insufficiency; and an exotropia develops out of a simple convergence-insufficiency to which is added a divergence-excess or out of a divergence-excess to which is added a convergence-insufficiency.

The processes by which these changes take place are not located in the muscles themselves, for until very late in the development of most cases of squint the rotations of the eyes are normal, indicating that the power of the muscles per se is neither impaired nor excessive. The changes that take place are doubtless *central*, following, as Hoffmann pointed out, the law promulgated by Sherrington, that excessive stimulation of the centre for one movement produces inhibition of the centre for the opposing movement, and vice versa. In other words, in cases of convergence-excess the centre for convergent movement, being subjected to continuous over-stimulation, becomes more and more active, producing a continually greater excess of convergence action and at the same time causing greater and greater inhibition of the centre for the divergent movements. The consequence is that, superadded to the convergence-excess, we have ultimately a progressive divergence-insufficiency. It is only late in the course of the condition that the persistent stimulation of the internus, coupled with the maintenance of one eye in the forced inward position, produces hypertrophy or contracture of this muscle and stretching or atrophy of its opponents.

A moderate proportion of cases of squint do, however, *develop out of a true muscular defect.*

Thus, some cases of convergent squint, both in childhood and in adult life, develop out of a primitive paralysis of the abducens which has been converted into a comitant deviation by consecutive overaction and contracture of the internus (see § 317). Still more frequently it happens that a vertical deviation due to congenital insufficiency of the superior rectus (less often of other muscles) is the starting point of an esotropia or exotropia (§§ 325, 336). Furthermore, a convergent squint due to a convergence-excess may be aggravated by the presence of a primitive weakness of the externi or overdevelopment of the interni. Again, a divergent squint may be produced by develop-

mental changes in the orbit causing a relative preponderance of the externi (see § 331), or by weakness of the internus due to operation. Such factors, however, play a subordinate part in the production of squint.

Age at which Squint Develops.—Some cases of squint are *congenital* or at least develop in the first months of life. These however, are in nearly all instances due to congenital weakness or absence of one or more of the muscles and therefore come under the head of paralytic squint, to which therefore reference should be made (see § 325). Ordinary esotropia develops, as a rule, at the age—i. e., between one and four—when the attempt at accurate and long-maintained fixation begins and thus puts a greater strain on the accommodation. This is true not only of the ordinary type of accommodative squint, but also of the lateral squint that develops as the result of a congenital vertical deviation (§ § 325, 336).

Unlike convergent squint, exotropia does not usually develop in very early life but later on in childhood, or in youth. This is naturally the case with the accommodative exotropia due to myopia, since myopia itself does not begin in early, but in late childhood; but it is likewise true of the many cases of exotropia due to non-accommodative conditions (non-accommodative convergence-insufficiency, divergence-excess).

In myopes as the myopia increases, the work imposed on the accommodation and consequently the impulse for convergence steadily decrease, while the demands made upon the convergence increase in proportion, owing to the approximation of the near point. This state of things must ultimately lead to a point where the convergence is no longer able to answer the demands made upon it. The convergence first becomes incompetent in the fixation of near objects, for which a stronger effort is required, and thus one of the eyes deviates out. Many myopes remain all their life in this condition of periodic strabismus; in others a constant strabismus develops from this condition, one eye getting to deviate out in looking at distant points as well. Quite the same thing obtains for the progress of non-accommodative exotropia (see § 333).

A spontaneous cure, such as is sometimes observed in esotropia never occurs in exotropia; on the contrary, it tends to increase with age.

Contrary to the rule that short-sighted persons squint outward, esotropia is sometimes found conjoined with high myopia. This is distinguished from ordinary esotropia by the fact that it does not develop in childhood, but in the later years of life, and is often associated with troublesome diplopia. This esotropia for distance may be associated with an exotropia for near, due to the defective convergence, that such patients often show

335. Why Squint Develops.—It seems quite certain that *in their inception the different periodic deviations* (*convergence-excess, convergence-insufficiency, divergence-excess, divergence-insufficiency*) *are accompanied by diplopia.*

Now the more nearly continuous (non-periodic) a deviation is, the easier it is for the patient to ignore the diplopia to which it gives rise, and the less pronounced, consequently, are the subjective disturbances which are dependent on this diplopia.

Again, a large deviation generally causes much less subjective trouble than does a small one: First, because the image projected by the deviating eye is so faint and so far from the other that the patient can readily ignore it; second, because the deviation being so large that the patient can not possibly overcome it, he does not even make the attempt to do so, and thus

escapes the reflex disturbance that such an attempt often produces. Accordingly, it is for the patient's interest that *if he has a periodic deviation it shall be transformed into one that is as nearly continuous as possible;* and, second, *that if it is small it shall increase in amount until the diplopia is readily negligible.*

As we have seen (§§ 331, and 333) both kinds of change occur regularly in the conversion of a heterophoria into a convergent or a divergent squint, and the squint itself, therefore, is to be regarded as the final outcome of a series of instinctive endeavors that the patient makes *to avoid diplopia and confusing images.*

336. Conditions Favoring the Development of Squint.—The development of a squint out of a heterophoria is much more apt to occur if there is any condition such as anisometropia, opacities of the media, fundus disease, congenital anomalies, etc., that *renders the sight of one eye much worse than that of the other.* In such a case, the image of the worse eye often confuses that of its fellow instead of reinforcing it, and hence the patient really sees better when he allows the worse eye to deviate. In many cases, it is apparent that he actually forces it to deviate so as to get the disturbing image out of the way. This is particularly the case in exotropia.

There is still another way in which the presence of unilateral visual defects may effect the development of squint. In the ordinary esotropia of childhood developing as the result of hyperopia, the hyperope is placed in the following dilemma: If he wishes to see distinctly, he has to make too strong an effort of accommodation; but he can do this only by putting forth an excessive convergence, so that he sees double. But if he converges only so much as is necessary, he can not bring the proper amount of accommodation into play, and hence sees indistinctly. He is, therefore, given the choice of seeing distinctly and double, or of seeing single and indistinctly. He prefers the former alternative whenever, from the fact that the image in one of the eyes has grown dim, diplopia is made less disagreeable to him. A similar explanation may hold for the development of exotropia.

Both explanations are quite certainly true, the second holding particularly when the amblyopia in one eye is extreme or when as in blind eyes it is absolute. Blind eyes, in fact very often squint (usually outward).

Another condition that favors the development of a lateral squint is the presence of a congenital vertical deviation (especially a congenital paralysis of the superior rectus). The patient in this case, being unable to fuse the double images on account of their difference in level, tries to obviate the confusion they produce either by tilting the head (§ 316) or by separating the images laterally as far as possible. This he does by diverging or converging the eyes.

That this is the cause of the lateral squint in these cases is proved by the fact that n some of them, particularly before the lateral deviation has become inveterate, the latter disappears when the vertical deviation is relieved by an operation on one of the elevators or depressors.

A factor which by many is regarded as the predominating element in the production of squint is the *lack of development of the fusion-faculty*

(Worth). The effect of this would be that the patient would have no tendency to blend the two retinal images, and hence would be unable to maintain binocular fixation. Consequently, if there was a deviation of the eye, he would not try to overcome it, but would allow one of them to squint.

337. Vision in Squint.—As a result of one of the eyes' being in an incorrect position, a man with a squint should see double. But this is really the case only at the commencement of strabismus. The diplopia soon disappears, and afterward can be evoked only by the use of all sorts of artifices, or can not be evoked at all. The disappearance of the diplopia is due to suppression (§ 291), which develops at a very early age. As a result of it, a man with squint has mere monocular vision; he does not, therefore, have stereoscopic sight in the proper sense of the word.

There is no doubt but that in the beginning of strabismus there is *diplopia* quite as much as there is in paralysis of an ocular muscle. But since the development of squint usually takes place in childhood, we hear nothing about diplopia, and by the time that the children are old enough to give a trustworthy account, they have already learned to perform suppression. But when the squint does not develop until later in life, as is the exception in convergent, but the rule in divergent strabismus, diplopia is then one of the regular symptoms. Sometimes it is so disagreeable as to furnish the main reason for the patient's visit to the physician.

While ordinarily no diplopia exists in old cases of strabismus, it is just in these cases that it is very apt to develop after an operation for squint. This postoperative diplopia as a general thing soon disappears, but in very rare cases it obstinately persists and becomes extremely troublesome to the patient. In some cases of this sort and also in some cases of squint not operated on, the diplopia is *paradoxical.* Thus, the eye may be straight or slightly converged, and yet the patient may have crossed diplopia (see § 291.)

If a man with squint does see with both eyes, he still resembles a one-eyed man in this, that like him he has deficient perception of depth and deficient or wanting *stereoscopic vision.* Those who squint do not notice this defect themselves, because they have learned by practice to form from attendant circumstances conclusions as to dimensions of depth, even though they do not appreciate them directly, and are able, though not perfectly, to form in many cases the same sort of judgments as those having binocular vision.

The *visual acuity* of the squinting eye is usually diminished as compared with the other or sound eye (except in alternating squint). In many cases, probably a certain degree of enfeeblement of sight exists even before the inception of the strabismus, and, in fact, constitutes one of the reasons for the development of the latter. The enfeeblement of sight, however, becomes greater and greater the longer the strabismus lasts, since an amblyopia ex anopsia develops on account of the exclusion of the eye from the act of vision (§ 241). This amblyopia finally reaches such a pitch that reading becomes impossible, and the sight may even be reduced to the ability to count fingers at a short distance. Such an eye has unlearned its ability to perform fixation; when the sound eye is covered, the squinting eye remains rigidly fixed in its false position or wanders uncertainly or may actually squint more strongly than before.

338. Squint a Bilateral Affection.—In squint the patient appears simply to fix with one eye and to turn the other inwards or outwards. In

reality, however, both eyes are turned. Thus, as shown in Fig. 166, when a patient with convergent squint is told to look at the object A, both eyes tend to converge equally, so that they really look at some nearer point, like B; then, in order to get the image of A on the fovea of at least one eye, the patient turns both eyes to one side—say to the right—through the equal angles BRA and BLD. The right eye, R, is now directed towards A or fixes it, the left eye squints in by the angle ALD = 2 × BLA. Thus, here really the squint is bilateral, each eye actually turning in by an equal amount, yet apparently the left eye alone squints, because the patient happens to use the right for fixation.

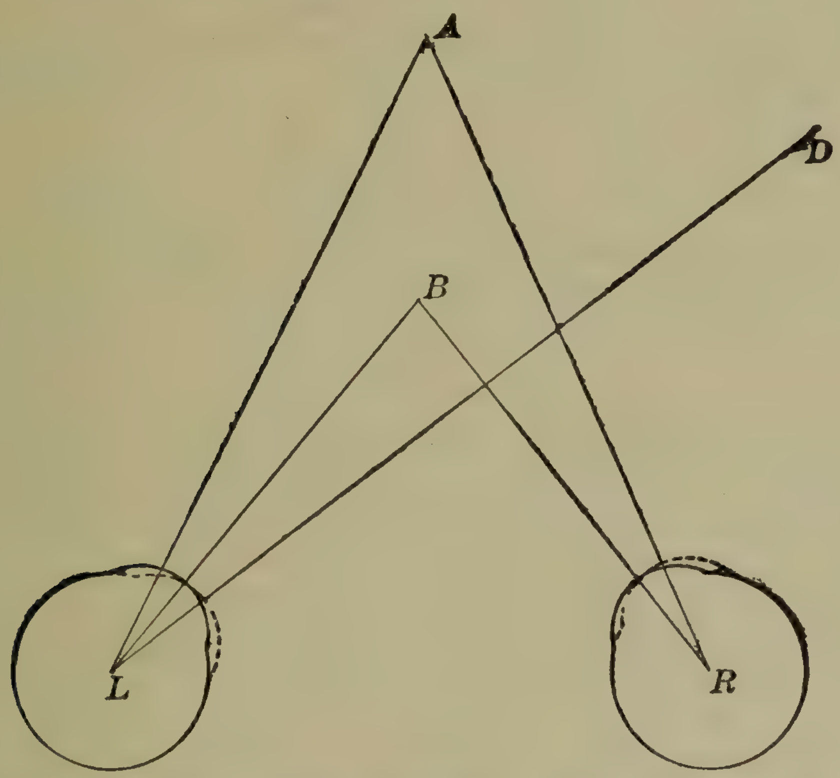

Fig. 166.—Bilateral Nature of Squint. (After Posey and Spiller.)

Which eye the patient actually uses to fix with depends usually upon the vision. If he sees much better with one eye than with the other, he will fix with the better and squint with the worse eye; the squint will then be unilateral. If the two eyes are about alike in vision and refracting power, we often find an alternating squint, the patient using either eye indifferently for fixation and squinting with the other. In some cases, particularly in anisometropia, the patient may fix with one eye for distance and with the other for near (see § 282).

339. Hyperphoria and Vertical Squint.—Hypherphoria in a great many instances is caused by paresis, or at least insufficiency of one of the elevator or depressor mucles of the eye. In this case the deviation and its evidences (vertical diplopia, etc.) increase and decrease characteristically in looking in some particular direction of the gaze (§§ 321, 322). Hyperphoria of high degree and real vertical squint (hypertropia and hypotropia) are almost always of this origin, being, therefore, *non-comitant* deviations.

In some cases, even when thus originating, a hyperphoria or vertical squint may become nearly comitant by virtue of consecutive contracture of the opponents of the paretic muscles (see § 317).

In a few instances with very high degree of hyperphoria a true vertical squint may be truly *comitant* from the start—i. e., we can find no evidence of insufficiency of any elevator of depressor muscle. Hyperphorias of low degree are usually comitant.

Some cases of hyperphoria seem to be *spasmodic* in origin, changing rapidly and lasting only a short time. Sometimes a spasmodic hyperphoria of this sort seems to be due in some way to accommodative action, since it disappears apparently as the result of the correction of a refractive error. In most cases hyperphoria once developed lasts indefinitely. It does not, as a rule, tend to increase, except in the form due to marked paralysis of an elevator or depressor where the deviation is often exaggerated as time goes on, either by secondary contracture of the opposing muscles or because the paretic eye fixes and the sound eye hence deviates excessively.

340. Symptoms of Heterophoria and Squint.—These are—

1. *Manifest deviation* of the eyes.

2. *Diplopia.*—This produces confusion, and, when the images are so close as to overlap, produces blurring of sight.

3. *Head-tilting*, due to the attempt to avoid diplopia (§ 316). This may even result in false torticollis.

4. *Vertigo.*

5. *Amblyopia* from disuse of the deviating eye (§ 241).

6. *Reflex Symptoms Induced by the Effort made to Correct the Deviation.*—These are asthenopia (asthenopia muscularis), pain in the eyes, conjunctival irritation, headache, which may be supra-orbital, orbital, temporal, or occipital, and other pains situated in various parts of the body. Occasionally, also, we find spasm of the facial muscles, and sometimes other forms of spasm; not infrequently also digestive disturbances, nausea, and interference with the general nutrition.

These symptoms, and especially the asthenopia, are the result of the strain imposed on the exterior muscles in the attempt to correct the deviation. If the patient does not correct the deviation, he sees double, and objects look confused; if he does correct it, the muscles after a time get tired from the continuous effort. Not infrequently there is an alternation between distinct vision with a sense of strain, and diplopia or confused vision when the strain is relaxed.

Reflex symptoms are especially pronounced when the deviation is so *slight* that the patient can overcome it, and consequently does so, although with effort. They are particularly marked when the deviation is inconstant and varies under different conditions, because in this case the patient cannot as readily allow for the changing position of the eyes as he can when the deviation is the same all the time. Consequently, reflex disturbances are *greater in heterophoria* than they are in squint and *greater in a periodic squint* than they are in one which is continuous. Indeed, as we have seen, this very fact seems to constitute the reason why a periodic is regularly converted into a continuous squint.

The most marked and disagreeable symptoms are found in hyperphoria and in divergence-insufficiency—conditions in which the movements required to overcome the deviation are specially hard to make (§ 299). Here the symptoms are usually most troublesome in *distant vision.*

In convergence-insufficiency the chief symptoms are asthenopia, headache, and conjunctival irritation. These are developed generally by *near work* and are much more likely to occur in the non-accommodative form.

A characteristic feature of the symptoms due to a muscular trouble is that they disappear if one eye is closed.

341. Treatment of Heterophoria and Squint.—1. *Correction of the refraction* is a very important element in the treatment of all of these anomalies and may alone suffice to effect a cure, particularly when the deviation is still of the periodic type. It is of special importance and efficacy in convergence-excess, particularly convergence-excess which is developing into squint. Here it is necessary to correct the whole of the hyperopia[23] and astigmatism present as found under atropine, and make the patient wear the glass all the time. Similarly, in convergence-insufficiency accompanying myopia, the patient must wear a glass fully correcting his error and wear it for near as well as for distance.

In cases in which the deviation is no longer periodic—i. e., in which, in addition to a convergence anomaly, there is superadded an anomaly of divergence—correction of the refraction is less apt to afford a cure, and in the case of a continuous squint rarely does more than diminish the amount of the deviation. In any case, the effect of correction of refraction is rarely an immediate one; it usually takes some months for the full effect of the glasses to be secured. Moreover, if the correction with glasses is to do any good, it must be kept up continuously.

The reason why glasses often effect only a partial cure in continuous squint is that they eliminate simply the accommodative element. Thus, in a hyperope with a combined convergence-excess and divergence-insufficiency they eliminate the former only, so as to convert the deviation from a continuous to a periodic one. Later, to be sure, the residual divergence-insufficiency may diminish and even disappear, since the very relief of the strain on the convergence may diminish also the tendency to divergence inhibition.

The earlier in the course of a muscular trouble, the treatment with glasses is initiated, the more likely it is to succeed, especially in cases of squint. Hence it is important to start it, if possible, at the very beginning of squint. As Worth shows, glasses may be worn even at the age of 1 or 2 years, and there should be no hesitation in prescribing then the approximate correction determined by objective tests (skiascopy). But, though we should endeavor to give glasses as early as possible in these cases, it must not be forgotten that their application sometimes succeeds when it is deferred till youth or even till adult life.

A fully developed exotropia is rarely relieved by glasses, because here the converging muscles are generally weakened. But in cases of beginning exotropia, especially, in youth and in cases of accommodative exophoria the deviation can often be lessened and its progress arrested by this means.

Cases in which the accommodative element is lacking do not yield to correction of the refraction. Such are especially cases in which the deviation is due either directly to a congenital weakness of the muscles or arises indirectly from such weakness, as is the case, for example, when a large vertical deflection of congenital origin causes an acquired lateral deviation to develop later (see § 325). Even in the latter case, there may be an accommodative element that acts to increase the deviation; and so far as the latter is due to this accommodative element it can be relieved by glasses.

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[23] A deduction of 0.25 to 0.50 D. may be made from the full correction in some cases, particularly in older subjects. But, as a rule, the nearer to the full correction we give, the better results we get.

The treatment of *heterophoria with glasses* is subject to the same rules as squint. The more fully the element of a convergence-excess enters into the case, the fuller should be the correction of the hyperopia. On the other hand, to presbyopes who show a marked convergence-insufficiency we should be careful to give as weak a reading addition as they can conveniently take, since the stronger the glass and the greater, therefore, the approximation of the near work, the more strain there is put on the weak converging power. Such patients often complain that their glasses "draw" and are too strong, and are much relieved when these are reduced in strength.

Cases of convergence-excess, especially with low accommodative power, in which the amount of esophoria for near is greater than for distance, may be helped by bifocal glasses in which the lower segments represent an addition of +2D or +3D to the correction for distance. The results from this treatment are said to be good, although the translator has not had much success with it.

342. (2) *Exercise of Fixation and Vision in the Squinting Eye.*—In squint, we must try to prevent the squinting eye from habituating itself to exclusion and thus declining more and more into a state of amblyopia. With this object we force the squinting eye to fixate by bandaging the non-squinting eye several hours every day. Since this measure is often baffled by the children's resistance, we may, instead of it, instil a drop of atropine into the sound eye every second or fourth day, so that for near it does not see clearly and the child is compelled to use the squinting eye.

This measure does not succeed if the squinting eye is quite amblyopic already (vision less than $\frac{6}{60}$), in which case bandaging of the sound eye must be resorted to. Atropinization of the sound eye if it straightens the squinting eye makes the sound eye squint, and if kept up too long can make the sound eye amblyopic.

When excluding the good eye in this way, the patient should systematically exercise the squinting eye in seeing and fixing—by kindergarten games with minute objects, if he is very young, and by reading, if he is older. The exercises, which should be made with the correcting glass and combined with the orthoptic exercises, should be done several times a day and should last from 10 to 30 minutes—their duration being increased as the child gets more expert. If they are kept up steadily for months, sometimes very substantial gains in vision are secured, and thereby the likelihood of a cure by orthoptic exercises or by operation is increased (see § 241).

343. (3) *Orthoptic Exercises.*—With a squint that develops in early life much can be done by educating the fusion faculty. This can be done with the *stereoscope*, or, better still, with Worth's *amblyoscope*. Both of these instruments, by compelling the eyes to act together, train the fusion faculty. Both are at first adjusted for the patient's angle of squint, then by successive adjustments the patient is gradually taught to look into the instruments with the eyes more and more nearly straight and yet maintain fusion. In this way the angle of squint is steadily diminished, and there is finally orthophoria.

This is not simply an exercise of fusion, but also and probably mainly an exercise of the divergence or convergence, as the case may be.

In applying the amblyoscope and, indeed, in all orthoptic exercises, the vision of the two eyes should as far as possible be placed on an equality. This is done by reducing the vision of the good eye with a dark glass or in the case of the amblyoscope by dimming the picture seen by this eye with an extra thickness of celluloid.

Another orthoptic exercise is *bar-reading*. This, as already stated, (§ 305) consists in reading with a bar or pencil held between the eyes and the print. Unless the patient is using both eyes at once for seeing, the bar will hide some of the letters. The systematic use of the bar first held rather from the page, then gradually closer and closer is an exercise in binocular vision.

If the patient can recognize *double images* at all, he can be practiced in recognizing and, if possible, in overcoming diplopia. This is best done by placing a red glass before one eye and a green glass before the other—darkening the sound eye with an extra thickness of glass—and making the patient look at a light. As soon as he can be got to recognize the diplopia readily, it is well to replace this exercise with the amblyoscope and bar-reading.

344. (4) *Exercises in Moving the Eyes.*—Exercises of the eyes in converging on a pencil carried towards the nose or in overcoming prisms, base out, are useful in convergence-insufficiency and occasionally in divergence-excess. Sometimes they relieve the symptoms when they do not essentially diminish the deviation.

In practicing with prisms, base out, the test-object should be placed at the reading distance, if we are dealing with a pure convergence-insufficiency, and at six metres if we are dealing with a divergence-excess. In mixed conditions, exercise at both distances should be used. Convergence-insufficiency may also be treated by exercising the con-convergence with the amblyoscope (see supra).

Convergence-excess may be treated by exercising the divergence with the amblyoscope or by practice in overcoming prisms, base in, the object in this case being held at the reading distance.

Other exercises have been tried, e. g., rotating the eyes from side to side, or up and down, or turning the head from side to side, while keeping the eyes fixed. Such exercises may be indicated when the muscles per se are weak, but would hardly have any influence in divergence or convergence anomalies.

345. (5) *Prisms for Wear.*—Deviations of low degree may be corrected by prisms worn constantly, either alone or combined with the glass correcting the refraction, or the same object may be effected by decentering the glass (§ 152). The apex of the prism is always placed in the direction in which the eye deviates. Prisms are most serviceable in hyperphoria. In esophoria and exophoria, prisms constantly worn often tend to increase the deviation, and their use is not generally advisable. Moreover, it is not usually possible for a patient to wear prisms of more than 1.5, or at most 2 *d*, before one eye, so that the total amount of deviation that we can correct by this means would not be more than 4 *d* at most. Prisms are rarely serviceable in paretic deviations (§ 326).

346. (6) *General treatment* must be employed in neurasthenia and hysteria and in conditions of debility from whatever cause. This will often relieve the symptoms without producing any material change in the mus-

cular condition of the eyes. The treatment of (7) other affections, particularly intra-nasal disease, which may possibly be *sources of reflex trouble*, should never be neglected.

347. (8) *Operation.*—When everything else has been tried and has failed, an operation is indicated. For methods see §§ 979-988, and for choice of operation see § 991.

In heterophoria an operation, if performed with judgment and with careful regard to the underlying conditions of the heterophoria, often relieves, sometimes to a surprising degree, the asthenopia, confusion, headache, and other symptoms. In squint the result obtained by the operation is, as a rule, simply cosmetic. The sight of the squinting eye is not influenced by the operation, and in only a few instances is even binocular vision restored. Nevertheless, the results that we secure by operation are not to be undervalued. A man with squint scarcely ever complains of the bad sight in his squinting eye, or of the absence of binocular vision; he is ordinarily unaware of the existence of either. He only desires relief from his disfigurement, and is very grateful if this is accorded him.

IX. Derangements of the Associated Parallel Movements. Conjugate Paralysis and Spasm

348. The derangements of the associated parallel movements of the eyes comprise—A. Paralysis and spasm of the parallel movements (conjugate paralysis, conjugate spasm). B. Tremor and ataxia of parallel movements (searching movements, nystagmus, pseudo-nystagmus). C. Dissociated movements.

349. Conjugate Deviation.—Conjugate deviation i. e., the condition in which both eyes are deviated in the same direction, is usually due to paralysis of one of the parallel movements (*conjugate paralysis*). Thus, if there is paralysis of right rotation, so that neither eye can rotate to the right beyond the middle line, the two eyes and often the head also are usually deviated to the left by over-action of the left rotators. Occasionally the head is deviated in the opposite direction to the eyes.

In some cases conjugate deviation is due to *conjugate spasm.* For example, we may find both eyes deviated to the left, not because the right-rotators are paralyzed, but because the left-rotators are in a state of primary spastic contraction. Such a condition is distinguished from paralysis by the fact that, although strongly deviated in one direction, the eyes can usually be moved more or less freely in the direction opposite.

350. Symptoms and Course.—When the deviation is alike in the two eyes, conjugate paralysis and spasm do not cause *diplopia.*

In conjugate deviation of pontine origin, a complete paralysis of the abducens may be combined with a paresis of the internus so that with the conjugate paralysis there is a convergent squint with homonymous diplopia.

In some cases of conjugate deviation there are *vertigo* and *false projection* (A. Graefe; M. Sachs). In a conjugate deviation to the left, due to paralysis, the patient would overshoot the mark when trying to touch an object situated on his right, while if the deviation were due to spasm he would undershoot the mark when trying to touch an object situated on his left.

Conjugate paralysis occurs in all degrees of *intensity*, being sometimes very slight, sometimes complete. It usually develops suddenly, and is

often transient. The conjugate deviation which the paralysis produces often disappears before the paralysis itself. This is especially true in cerebral disease.

351. Varieties of Conjugate Paralysis and Spasm.—1. *Paralysis of lateral movement* occurs under the following forms:

(A) Neither eye, when attempting to follow an object that is moving to one side, say the right, can move beyond the middle line. But the left eye can move to the right when converging on an object which is brought in towards the eyes in the median plane (retention of convergence).

(B) Neither eye can follow an object that is carried to the right, provided both eyes are open, but the left eye can follow the object to the right if the right eye is covered. This is really the same case as A, the movement inwards of the left eye when the right eye is covered being simply a movement of convergence (Jeffries, Moebius).

(C) The right eye can follow an object that is moving to the right, but the left can not. The left eye, however, can move to the right in performing convergence. This condition, which may be called conjugate hemiparalysis, is thus distinguished from a paralysis of the left internus in which the left eye can move inwards neither when converging nor when attempting to make a conjugate movement to the left.

(D) Both interni may be paralyzed for conjugate movement (Wilbrand and Saenger).

(E) Neither eye can follow an object that is carried to the right, and the left eye, moreover, cannot move to the right when attempting to converge (combined paralysis of lateral movement and convergence).

Paralysis of lateral movement is sometimes bilateral—i. e., both eyes fail to follow an object that is carried either to the right or to the left, although it may be that both can still converge.

2. *Spasm of lateral movement* occurs under the following forms:

(A) Both eyes are deviated spastically to one side, but can be moved freely in all directions (pure spasm of lateral movement).

(B) Both eyes are deviated spastically to one side, and can not be moved to the side opposite (excessive spasm of one side-to-side movement, or spasm of one side-to-side movement combined with paralysis of the movement in the opposite direction).

3. *Paralysis* and *spasm of up and down movements* are rare.

Most common are paralyses of movement upward, then of movement both up and down. Paralysis of downward movement alone is exceedingly rare (Spiller, Wilbrand and Saenger).

352. Site of Lesion in Conjugate Paralysis and Spasm.—*Paralysis* of lateral movement is caused by destructive lesion in—

1. The *cerebrum* and either in the cortex, in the subcortical association paths, or in the tracts connecting the cortex with the oculo-motor and abducens nuclei. Such lesions are usually of sudden onset and associated with apoplectoid symptoms (coma, hemiplegia). A lesion of the right hemisphere produces paralysis of movements to the left, and consequently a conjugate deviation to the right (the patient turns his eyes towards the lesion). Convergence is always retained.

2. The *pons*. Lesions on the right side of the pons will produce paralysis of right-hand movements, and vice versa—i. e., the eyes look away from the lesion.

Small lesions in the pons involving the abducens nucleus or its immediate connections cause paralysis of lateral movement without paralysis of convergence (paralysis of types A and B, § 351). Lesions high in the pons involving only the fibres connecting the

abducens and the oculo-motor nucleus produce a conjugate hemiparalysis of the opposite internus for parallel movements but not for convergence (type C, § 351). Larger lesions in the pons above and below the abducens nucleus cause combined paralysis of lateral movement and convergence (paralysis of type E, § 351). (Jeffries, Schoeler.)

3. The *cerebellum.* The deviation here is sometimes toward the lesion, usually away from it.

Spasm of conjugate movement is caused by an irritative lesion in—

1. The *cerebrum* (in the cortex only). The eyes are turned away from the lesion.
2. The *pons* (very rare). In this case the eyes look toward the lesion.

Paralysis of up and down movement is due usually to a lesion involving the anterior parts of both oculo-motor nuclei or the corpora quadrigemina or to one occupying the middle line close behind the corpora quadrigemina. It also occurs as a remote effect in lesions of the pons and the pineal gland.

Some cases *simulating conjugate paralysis* are due to symmetrical defects in the muscles or to symmetrical lesions of the two oculo-motor nerves at the base.

A conjugate paralysis due to a lesion remote from the nucleus, e. g., a lesion of the cortex or the paths descending from the latter or a paralysis due to inhibition (as in hysteria) is distinguished by the fact that only the voluntary movements are absent, while the movements set up by a reflex impulse are present. Such reflex movements include those set up by vestibular stimulation (sound), fixation movements (maintenance of fixation on an object when the head is passively turned), and movements involuntarily made to compensate for the deviation when a strong prism is thrust between the eyes and the object (Graefe's test; § 321). In a paralysis due to basal lesions these reflex movements are absent.

X. Tremor of the Associated Movements. Nystagmus and Pseudo-Nystagmus

353. Searching Movements.—Both eyes make an occasional wide, comparatively slow, sweeping movement from the primary position in some direction, and then, either immediately or after a time, return to the primary position. The movements are apparently purposeful, as if the patient were trying to look at an object situated to one side of him. They may take place in any direction, vertical, oblique, or horizontal. Searching movements are found in blind eyes and in eyes which from disease at the macula and other causes have lost the power of central fixation.

354. Pseudo-Nystagmus.—One or both eyes when carried to a point near the limit of their excursions in some direction, make a series of jerky movements on from this point and back to it again. These jerks may be either horizontal or rotary and apparently represent an extreme effort to keep up the original excursive movement of the eyes, the smooth steady pull of the muscle that is carrying the eye along being converted into a series of discontinuous, spasmodic tugs.

Pseudo-nystagmus is sometimes unilateral, more often bilateral.

Pseudo-nystagmus occurs almost constantly in hereditary ataxia and very often in multiple sclerosis, but also occurs in a great many other nervous affections and often in persons who are quite healthy. It seems to denote usually weakness or fatigue of the muscles which move the eye in the direction in which the pseudo-nystagmus occurs.

355. Nystagmus.—True nystagmus is the condition in which the eye while still in the primary position or near it, swings back and forth in a

series of very rapid, usually quite regular and rather small vibrations. It comprises:

1. Vibratory nystagmus (nystagmus vibrans, pendulum-like nystagmus), in which the swing in one direction is just as swift as that in the opposite.

2. Resilient nystagmus (nystagmus resiliens, rhythmic or jerky nystagmus), in which the eye makes a slow swing in one direction followed by a quick return.

Pendulum-like nystagmus is the distinguishing mark of cases characterized by amblyopia (cases of visual nystagmus), but also occurs in other forms, e. g., miners' nystagmus. Resilient nystagmus characterizes cases due to disturbance of innervation in the labyrinth (aural nystagmus).

In *direction* the oscillations may be horizontal, vertical, rotary (wheel-like), or mixed. The last includes combined horizontal and rotary and combined horizontal and vertical (i. e., oblique) oscillations. In *circumduction* nystagmus the centre of the cornea by making successive horizontal and vertical movements describes a circle or ellipse. Horizontal and vertical nystagmus are called collectively nystagmus oscillatorius.

True nystagmus is nearly always bilateral and then the movements are almost invariably equal, simultaneous, and parallel in the two eyes (*conjugate* nystagmus). Bilateral nystagmus is usually horizontal, less often rotary. Mixed or vertical bilateral nystagmus is rare. In miners' nystagmus (§ 356) the movements are often irregular.

Very rare forms are (a) *disjunctive* nystagmus in which the two eyes swing rapidly toward and away from each other, i. e., make rapidly alternating converging and diverging movements, or revolve toward and away from each other or diverge and converge in a vertical plane, so that one eye rises while the other falls (see-saw nystagmus—Maddox); (b) *dissociated* nystagmus, in which the movements in the two eyes are quite dissimilar in direction or in extent; (c) *unilateral* nystagmus. Unilateral nystagmus is usually vertical.

In many cases nystagmus changes greatly in intensity or in character when the eyes are turned in some special direction, or when they are converged, or when either or both are covered. If it develops only when one eye is covered it is called *latent* nystagmus. It may be specially marked when the attempt is made to fixate an object (*fixation* nystagmus). It is increased by observation and also by illumination—abating in the dark and when the eyes are closed.

Uhthoff gives the name of *quivering* nystagmus to very small, exceedingly rapid usually symmetrical vibrations occuring periodically or only when the eyes are examined by oblique illumination.

The rapidity, extent, and character of the oscillations may be recorded by an instrument—the cinematograph or nystagmograph (Buys Abrahams, Coppez, Ohm). The ophthalmoscope also is useful for detecting faint vibrations, otherwise undiscoverable.

356. Etiology of Nystagmus.—Nystagmus is caused by—

(A) Conditions producing bilateral amblyopia in early infancy (*visual* or so-called congenital nystagmus). Such conditions are opacities of the cornea, especially from ophthalmia neonatorum; congenital cataract; hæmorrhage, disease, or abnormalities of the retina and chorioid; total congenital color blindness, and albinism. Refractive errors, even when great, do not usually cause nystagmus, although they may do so.

Visual nystagmus is probably not often really congenital, but develops during the first few months of life. Its genesis may be conceived of, as follows. Fixation is not a faculty inborn in man, but has to be learned by practice. When an eccentric spot of the retina has impressed on it an image that excites the attention, there is set up by reflex action an eye movement which is calculated to place the fovea opposite the object. As

the eye moves and the image thus approaches the fovea, the image becomes more and more distinct, and most distinct of all precisely when the fovea is reached. The moment this occurs the movement ceases, for should the eye be carried by the position required for fixation, the object would at once appear more indistinct again (cf. § 301). But such an inhibition of movement at the right moment presupposes that as regards vision the fovea greatly surpasses the adjoining portions of the retina. If—on account of the affections above mentioned—this is not the case, an arrest of movement at the right moment fails to occur, and the eye vibrates in small excursions about the position of fixation. If fixation has been acquired before amblyopia sets in, nystagmus fails to develop.

Other explanations (congenital neuropathic tendency), disturbed cerebellar actions, have been given to account for visual nystagmus.

A certain degree of visual power must be present for nystagmus to exist; persons who are born blind, or who become totally blind very early do not acquire nystagmus. In such persons the eyes move slowly and aimlessly about, making large excursions.

Unilateral amblyopia very rarely produces nystagmus but tends rather to produce squint.

Sometimes a nystagmus dating from infancy, occurs without there being any obvious lesion of the eyes to cause it. Such a nystagmus, according to A. Graefe, is probably attributable to a congenital retinal hæmorrhage which subsequently cleared up.

In small children a combination of head-nodding and nystagmus occurs as a transient affection which is known as *spasmus nutans*. According to some it is produced by the child's being kept in a dark room, according to others by rickets (craniotabes). The nystagmus often assumes aberrant forms and may be unilateral.

(B) Conditions developing *in later life* causing constant *strain of the eyesight*. Such conditions are usually those incident to the patient's occupation (hence *occupational* nystagmus). The commonest form of this is miners' nystagmus, which seems to be due mainly to insufficient illumination.

In miners' nystagmus the light sense is greatly reduced. The movements are often unlike in character or degree in the two eyes. Rotary nystagmus is most common, horizontal the least. Vertical, circumduction, and oblique movements are quite frequent (Ohm).

Nystagmus due to insufficient illumination occurs occasionally in other occupations (Frost), and a twitching (probably not a true nystagmus), which Snell ascribed to constrained position of the eyes, occurs in compositors, etc.

In the same category belong those cases in which visual nystagmus develops after the age of infancy as a result of high astigmatism or of traumatic cataract.

(C) Irritation arising from the labyrinth (*vestibular* nystagmus). — This may be produced by actual disease of the labyrinth or artificially by changes in labyrinthine circulation and pressure due to rapid rotation of the body, syringing the ear with hot or cold water, or galvanization of the ear—rotation, caloric, (or thermic,) and galvanic nystagmus.

In vestibular nystagmus the movements are of the resilient character and their direction can be precisely regulated by varying the stimulus provoking it. The nystagmus is increased if the patient looks in the direction of the quick movement, diminished if he looks in the opposite direction. It is associated with tendency to fall to one side, vertigo, and past-pointing, i. e., when the patient tries quickly to touch an object in front of him his finger deviates to the side opposite to the quick movement (cf. § 306). An allied condition is the badly termed *optical* nystagmus, a resilient nystagmus caused by fixing the gaze on an object in real or apparent motion (e. g., in looking out of a car window).

(D). *Nervous Diseases.*—Nystagmus is very frequent in cerebellar abscess and tumors of the cerebello-pontine angle and is almost constant in tumors of the cerebellum itself. In multiple sclerosis a vibratory nystagmus occurs in 12 per cent of the cases (Uhthoff) and a resilient nystagmus of vestibular character is still more common (Barány). Nystagmus is also very frequent in hereditary ataxia, usually denoting an affection of the fibres going to Deiters' nucleus (Wilbrand and Saenger). It is seen fairly often in cerebral abscess and encephalitis (in each, in about 10 per cent of the cases, according to Uhthoff), being apparently specially frequent in encephalitis lethargica; also in syringomyelia, Little's disease (16 per cent of cases), and idiocy (28 per cent of cases).

In other nervous diseases it is rare.

(E) *Poisons*, especially ether and chloroform; rarely also alcohol, morphine, cocaine, sulphonal, veronal, arsenic, lead, quinine, ergot, sewer gas, chloroform, etc.

Nystagmus, whether visual or due to nervous disease, is sometimes *hereditary*. Some persons can produce nystagmus *voluntarily*.

Theory of Nystagmus.—The remarkable regularity and parallelism of the movements in nystagmus show that we have to deal with a perversion of the centres for parallel and parallel-rotary movements and not with peripheral lesions of the muscles themselves or their nerves. There is, however, no unamity of opinion as to the way in which this perversion is produced.

357. Symptoms of Nystagmus.—They are—

1. *Apparent Movement of Objects.*—In miners' nystagmus and vestibular nystagmus this is a very prominent and distressing symptom. It is also often, but not always, present in the nystagmus due to nervous disease. On the other hand, in visual nystagmus due to infantile amblyopia, it is very rarely perceived. Sometimes in unilateral nystagmus and under exceptional conditions, in bilateral nystagmus it can be evoked (A. Graefe). Even then, however, it seems hardly ever to be troublesome.

2. *Reading in Vertical Lines.*—That the apparent movement of objects even when unrecognized, yet causes indistinctness of things seen, is shown by the fact that some patients with horizontal nystagmus in reading hold the book sideways, so that the lines of print run vertically. This partly obviates the confusion produced by the oscillations of the letters.

3. *Vertigo.*—This is marked in some of the late acquired forms especially in miners' and vestibular nystagmus.

4. *False Projection.*—This is seen in the past-pointing of vestibular nystagmus.

5. *Nodding Movements of Head.*—These often occur in visual nystagmus. They were formerly thought to be compensatory in character, acting to prevent the tremor of objects produced by the oscillation of the eyes. This, however, is not the case.

6. *Head-Tilting.*—When the nystagmus is less in some one direction of the gaze, the patient avoids looking in that direction by turning his head. In miners' nystagmus the head is often tilted back.

7. *Poor Vision.*—In most cases of nystagmus of infantile origin, the vision is quite poor, and can not be brought up to normal by glasses. This is in part due to the blurring that the nystagmus produces, but much more to the pathological condition causing the nystagmus. In those cases in which the nystagmus increases greatly in one direction of the gaze and diminishes in another, the sight is very much better in that position in which the oscillation is slight—a fact which the patient gets to appreciate and utilize.

8. *Photophobia* may be present.

Course of Nystagmus.—Infantile nystagmus often lasts through life, although it may disappear. Nystagmus due to aural or nervous disease persists as long as its cause. Miners' nystagmus disappears if the patient gives up his work and lives under proper conditions as regards illumination and eye-strain. Nystagmus due to spasmus nutans and hysteria is probably always transient. A transient nystagmus may also result from traumatism, tenotomy, or other causes.

358. Treatment.—Infantile nystagmus is rarely affected by any treatment, although isolated cases have been cured by removal of a congenital cataract, by tenotomy or advancement causing relief of a squint, by exercises with a stereoscope and in reading with the aid of perforated diaphragm, by exercises in fixation in various positions of the gaze, and by exercises with rotating prisms. Some cases of nystagmus, especially acquired nystagmus, are relieved by the correction of astigmatism or other refractive errors. In miners' and other forms of occupational nystagmus change of occupation is imperative.

XI. Dissociated Movements

359. In some cases the eyes no longer follow the law of associated movements, but each moves irrespective of the other. This may occur in eyes that have been totally blind from birth, in patients deeply under the influence of chloroform, and occasionally in other cases. Various forms of this anomaly have been described.

In one form each eye on alternate covering goes up or each eye goes down behind the screen. In these cases there may or may not be binocular fixation when both eyes are uncovered (see § 282, Note 6). In these and other cases described the dissociation is often apparent only. Thus when several muscles are paralyzed in one eye and yet this eye is used for fixation, the secondary deviations of the sound eye, rapidly changing their form and varying their amount as the eyes are carried in different directions, make it appear as if the eyes were moving quite contrary to the ordinary laws of association.

XII. Paralysis and Spasm of Lid Muscles

360. Spasm of the Orbicularis (Blepharospasm).—Blepharospasm[24] manifests itself by a squeezing of the lids tightly together It is either a symptom of other diseases of the eye (symptomatic blepharospasm), or it forms a distinct disease by itself (essential blepharospasm).

Symptomatic blepharospasm accompanies all irritative states of the eye, and is hence found in conjunction with the presence of foreign bodies

[24] From βλέφαρον lid, and σπᾶν, to draw tight.

in the conjunctival sac, with trichiasis, with the most various forms of inflammation of the eye, when an intense amount of light falls on the eye, etc. The violence of the blepharospasm is by no means in direct proportion to the severity of the disease of the eye, so that no sort of conclusion can be drawn from it with respect to the violence or the duration of the ocular affection that lies at the root of it. It often renders the examination of the eye extremely difficult. Blepharospasm is usually most violent and most obstinate in phlyctenular conjunctivitis. It reacts unfavorably upon the eye trouble; moreover, it often leads to œdema of the lids, to blepharophimosis, and to spastic ectropion and entropion. Its treatment consists in the removal of the ocular disease which lies at the root of it (cf. § 488).

Essential blepharospasm is distinguished from the symptomatic variety by the fact that in it the eyes themselves are found to be perfectly normal. In young persons, particularly of the female sex, it manifests itself by the eyes' suddenly shutting up and then remaining closed as if in sleep (*blepharospasmus hystericus*). In elderly people blepharospasm (*blepharospasmus senilis*) appears either under the form of clonic spasm—i. e., continual winking (nictitatio[25])—or as a tonic spasm by which the eyes are kept tightly closed for a length of time. Essential blepharospasm is extremely annoying to the patient; indeed, in severe cases it has, so far as he is concerned, almost the same results as a real blindness, since the patient cannot make use of his eyes when they are shut. Hysterical blepharospasm in time disappears of itself, while senile blepharospasm resists treatment for a long time, and sometimes, indeed, is absolutely incurable.

In *hysterical* blepharospasm both eyes shut up suddenly and usually without any known cause. It may take some hours, but it may also take days and even months before the eyes open again, which they do, and just as suddenly. Such attacks may be repeated quite often, and may also vary greatly in their duration. The spasm almost always affects both eyes; once only Fuchs saw unilateral hysterical blepharospasm. Other symptoms of hysteria are often present coincidentally with the blepharospasm. In a young girl who suffered with blepharospasm of this sort, and who had already been treated in vain with different remedies, the blepharospasm was finally relieved by means of a single instillation of cocaine; but a few minutes after the girl had opened her eyes both legs became paralyzed, and remained so for several days (Fuchs).

In the examination of patients with hysterical blepharospasm it is often possible to find so-called pressure points—i. e., portions of the body upon which we simply have to press for the eyes to open as if by magic (Von Graefe). In the majority of cases the pressure points lie in the region supplied by the trigeminus, as at the place of exit of the supra-orbital and infra-orbital nerves at the upper and lower margins of the orbit. But often these points are more difficult to find, being situated, for example, in the cavities of the nose, the mouth (when there are carious teeth), or the throat; sometimes the pressure points are not found in the region supplied by the branches of the trigeminus at all. Thus cases occur in which the blepharospasm can be made to disappear by pressure upon one of the costal cartilages or the vertebræ, or upon some spot of the arm or leg, etc. Often the patient himself calls the physician's attention to the position of the pressure points, the knowledge of which he has already utilized for his own relief. The most frequent cases are those in which the patient allays the blepharospasm by pressure upon the forehead, a pressure which acts upon the branches of the supra-orbital nerve. Young men wear a hat with a stiff brim which they press down hard upon the

[25] From *nictare*, to wink.

face; girls tie a ribbon tight about the head. But as soon as the pressure ceases—e. g., by taking off the hat—the eyes shut again. In many cases diversion of the attention to other things suffices temporarily to relieve a blepharospasm.

The form of hysterical blepharospasm, in which the eyes remain quietly closed without any apparent spasm, presents a great similarity to ptosis. We can, however, readily recognize it to be a spasm if we try to open the eye by lifting the upper lid; for we then feel the resistance that the orbicularis offers to the opening of the eye. As indicative of the contraction of this muscle, we see that the skin of the forehead is thrown into vertical wrinkles, and that the eyebrows are lower than usual. In ptosis the skin of the forehead, owing to the contraction of the frontalis, shows horizontal wrinkles, and the eyebrows are somewhat higher than usual (Fig. 167). In the normal state the situation of the eyebrows corresponds to the upper margin of the orbit.

Senile blepharospasm is often only one of the symptoms of a general spasm of the face (tic convulsif). In this form, too, there are frequently pressure points which influence the spasm.

Just as the normal act of winking is set up in a reflex way by irritation of the terminal extremities of the trigeminus upon the surface of the eyeball, so, too, blepharospasm is in most cases of reflex nature. This is beyond doubt the case in symptomatic blepharospasm, set up by irritation of the trigeminus due to a foreign body, or to inflammation, etc., and reflex action starting from the trigeminus must be assumed to exist in many cases of essential blepharospasm also. A proof of this is the fact that pressure upon branches of the trigeminus so often abrogates the blepharospasm, and that at the same time the pressure points themselves are often sensitive to pressure. *Treatment* has therefore the greatest prospect of success in those cases in which it is possible to find pressure points, as then we can attack directly the starting point of the reflex action. This is done by applying the galvanic current to the pressure points, or by injecting morphine or by using a counter-irritant at these spots. If no pressure points are discoverable, we must think of the surface of the eyeball itself as a source of the reflex action, and may try to render this insensitive by cocaine. In senile blepharospasm it is probable that senile changes in the facial lie at the root of the trouble, and hence the outlook for a cure is small. We may try the galvanic current (anode in front of the lobe of the ear on the trunk of the facial, cathode on the nape of the neck). In addition we employ the remedies used against neuroses in general. Schlosser recommends the injection of alcohol along the trunk of the nerve at its point of exit from the stylo-mastoid foramen. Others have performed stretching or resection of those branches of the trigeminus from which the reflex emanates and stretching of the trunk of the facial.

Children of from eight to fifteen years are frequently brought by their parents to the physician on account of a habit of *continual winking.* This happens not infrequently during the occurrence of a slight conjunctivitis and keeps on independently after the latter has been relieved. For the most part, in this case we have to deal with rather anæmic and nervous children. This affection—frequently attributed to badness by the parents—usually passes off of itself after some time.

Repeated *fibrillary contractions* of single bundles of the orbicularis, which are appreciated by the patients themselves, occur very frequently in perfectly healthy persons with normal eyes. No sort of significance is to be attributed to them.

361. Paralysis of the Orbicularis.—If the case is one of recent paralysis, no changes are noticed while the eye is open; but if the patient has cause to shut his eye, it is apparent that the closure is but incompletely performed, because the lower lid cannot be lifted properly. This is particularly striking in the inner half of the lid. In consequence of the incomplete closure of the lid there is epiphora, which in light cases often constitutes the only complaint the patient makes. After the paralysis has lasted quite a long time further changes set in. The lower lid falls away from the eyeball and keeps drooping lower and lower all the time (ectropion paralyticum). The cornea

during sleep is exposed to desiccation in its lowermost part, so that keratitis e lagophthalmo develops.

Paralysis of the orbicularis is usually caused by an affection of the facial nerve which innervates it. In rare cases it is myopathic, i. e., due to a primary atrophy (abiotrophy) of the muscle itself (Treacher Collins). A facial paralysis due to a lesion situated above the nucleus (supranuclear lesion) chiefly affects the oral branches of the nerve, while the orbicularis is usually unaffected. Hence, if the orbicularis is paralyzed we have ordinarily to do with a lesion of the nerve in the nucleus itself or below it. Most frequently we have to deal with a so-called rheumatic paralysis, the real cause of which is unknown; but the paralysis may also be caused by injury (particularly fractures of the base of the skull and operations in the region of the parotid gland and middle ear), by caries of the petrous bone, by tumors, and by syphilis. The rheumatic paralyses of the orbicularis give a favorable prognosis, but even in them several months are required before the cure takes place.

The *treatment* must first of all endeavor to remove the cause of the paralysis. Symptomatic treatment consists mainly in the application of the electric current, both constant and induced. As long as the closure of the lids is imperfectly performed, the palpebral fissure must be kept closed by a bandage (see § 442), to prevent the development of ectropion and of keratitis. In severe cases the bandage must be worn constantly; in light cases it is sufficient to apply it at night only. If the paralysis proves incurable, tarsorrhaphy is indicated in order to make it possible to shut the eyes.

362. Paralysis of the Levator; Ptosis.—Paralysis of the levator palpebræ superioris manifests itself by a drooping of the upper lid (*ptosis*[26] *or blepharoptosis*). All degrees of ptosis occur from a just noticeable depression of the upper lid to a prolapse of it so complete that it hangs down quite relaxed and devoid of wrinkles, and covers the whole eyeball. The higher degrees of ptosis, in which the lid hangs down in front of the pupil, interfere with vision, unless the patient lifts up the lid with his finger, or unless it is possible for him to draw it up sufficiently by a forced action of the frontalis muscle. By the contraction of the latter the forehead is wrinkled and the skin over it is thus contracted, so that the eyebrows and indirectly the upper lid as well are elevated (Fig. 167). But since this elevation is not sufficient the patient is in addition compelled to throw his head back, because then in looking forward the eyes are directed down, and thus the pupils get to lie in the palpebral fissure even though it is low. The wrinkled forehead, updrawn eyebrows, and backward pose of the head are characteristic of persons with bilateral ptosis.

Ptosis is either acquired or congenital. *Acquired* ptosis may be caused by a lesion either of the muscle itself or of the nerve supplying it. The former condition is most frequently due to injuries about the upper margin of the orbit, by which the tendon or the belly of the levator is divided. But there is also a form (*ptosis myopathica*) which in women of middle or advanced life (very rarely in men) develops very gradually, is always bilateral,

[26]From πίπτειν, to fall.

and depends on a primary atrophy of the muscle and probably also of the surrounding tissue. It is characterized by the fact that a deep furrow forms between the lid and the upper margin of the orbit. Ptosis due to paralysis of the *nerve* supplying the levator is almost always unilateral and generally forms one of the symptoms of an oculo-motor paralysis. Isolated ptosis without any other sign of oculo-motor paralysis is generally due to a central

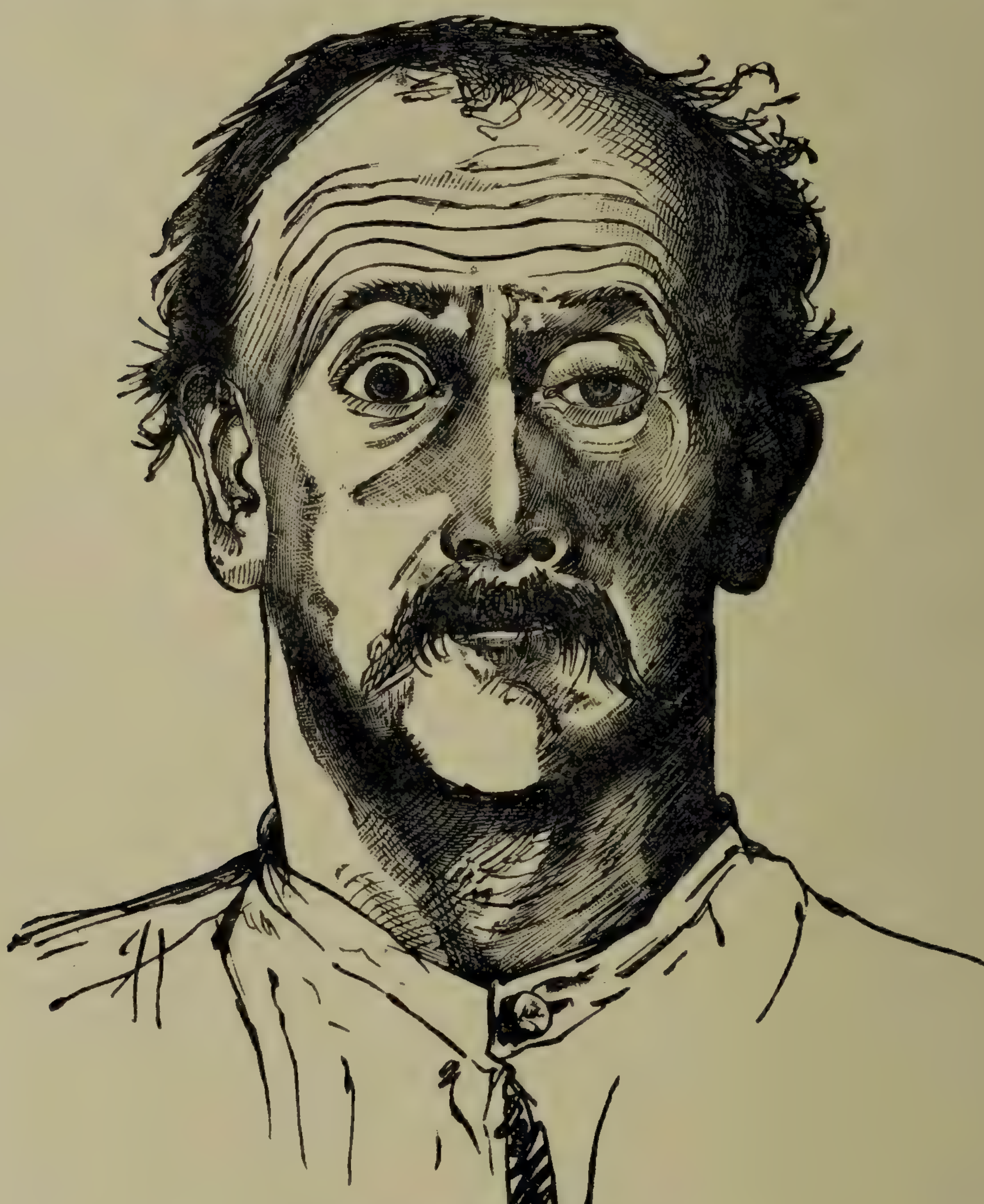


Fig. 167.—Left-sided Ptosis.

The ptosis was not complete, but just great enough for the upper lid to cover the pupil. Now as the left eye was the better of the two, the patient, in order to see with it, lifted his lid by contracting the frontalis muscle. He was not able, however, to do this on the left side alone, but wrinkled his forehead all over, so that the eyebrows are elevated on both sides. Hence, too, the right upper lid is drawn unusually high up, so that a strip of sclera can be seen exposed above the cornea, and the difference between the two palpebral fissures has become even more striking than before. The pupil of the right, amblyopic eye is dilated.

lesion. A ptosis due to nerve lesion may vary with the position of the eye (see § 364).

Congenital ptosis is generally bilateral. It may be due to absence or ill-development of the muscle and is often associated with other congenital anomalies. It is frequently hereditary.

Paralysis of the tarsal muscle produces only a slight degree of ptosis, evidenced by a moderate narrowing of the palpebral fissure. Such a ptosis occurs in paralysis of the sympathetic (§ 374) and in trachoma (§ 467).

Slight ptosis, or at least narrowing of the palebral fissure, is produced by advancement of the superior or inferior rectus or by a paralysis of these muscles not due to a tenotomy (§ 66).

The name of ptosis is incorrectly used for conditions which have nothing to do with an affection of the levator palpebræ superioris—e. g., when the upper lid droops because it has become heavier in consequence of thickening due to trachoma, new growths, etc., or when the upper lid is drawn down by a slight spasm of the orbicularis. So, too, ptosis adiposa (§ 425) does not belong with genuine ptosis.

An apparent ptosis is produced when the eye recedes into the orbit.

In the *treatment* of ptosis we should try first to remove the cause, when this can be ascertained. But if we have to do with a ptosis which has become inveterate, or if it is congenital, improvement can be obtained only by an operation (see §§ 936-939).

363. Spasm of Levators.—In the levator palpebræ spasm does not occur, if we except the anomalous cases described in § 364 and the sort of secondary contracture of the muscle which sets in when the orbicularis is paralyzed and which serves to widen somewhat the palpebral fissure. Tonic spasm of the *tarsal muscle* is produced by the instillation of cocaine and irritative lesions in the cord (see § 373), and is thought also to occasion the elevation of the upper lid in exophthalmic goitre.

A similar blepharodiastasis, or condition in which the palpebral fissure is wider than usual, may be caused by a complete tenotomy with consequent retraction of the superior or inferior rectus (§ 66).

364. Abnormal Associated Movements of the Lids.—Normally there is a certain association between the movements of the eyes and the lids, their association, being effected partly by the palpebral attachments of the ocular muscles (see §§ 54, 66), partly also probably by the connection between the motor nuclei of the eye and the facial nerve (§ 300). Certain abnormal associations have been observed, which have been ascribed to peculiarities in nerve communication. Such a condition is *jaw-winking* in which the upper lid rises when the mouth is opened or lateral movements of the lower jaw are made (Gunn). These movements may occur in a lid which droops somewhat from congenital ptosis or may occur without ptosis.

Sometimes the eye opens wide in one direction of the gaze and shuts when turned in the opposite direction. Thus in some cases of oculo-motor paralysis of central origin there is ptosis if the eye is abducted, while in adduction (or in attempted adduction if the internal rectus is paralyzed) this diminishes or disappears altogether, or even is converted into the opposed condition, so that the upper lid rises abnormally high. This sort of alternation occurs not only in central oculo-motor paralysis, but also in peripheral paralyses of the eye muscles, including especially congenital paralysis of the external rectus due to actual deficiency of the muscle. In this latter set of cases, and rarely in some of the others, the ptosis occurs in adduction, not in abduction. In some of these cases, at any rate, it is not a true ptosis that is in question, but a contraction of the orbicularis, marked by a sinking of the upper and a rising of the lower lid. There are also cases of *cyclic widening and narrowing* of the palpebral fissure, the widening, which is evidently due to spastic contraction of the levator or the musculus tarsalis, being associated with convergence of the eyes and miosis (see § 325).

XIII. The Pupil

365. Function of the Pupil; Alterations in Size.—The iris forms a diaphragm which, as in the case of many optical instruments, is interposed between the refracting portions of the eye. It has a double task to perform: it prevents an excessive amount of light from entering the eye and so

dazzling it and injuring the retina, and it cuts off the marginal rays. These are the rays that pass through the periphery of the cornea and of the lens, and which, being less regularly refracted, would, unless arrested, impair the sharpness of the retinal image. In order to be perfectly impermeable to light, the iris has a pigment layer on its posterior surface. The iris has the advantage over the artificial diaphragms of optical instruments in that its size changes spontaneously to suit the circumstances of the case. For this purpose there exist contracting fibres (sphincter pupillæ) and dilating fibres (dilatator pupillæ).

The *contraction* of the pupil is governed by the oculo-motor nerve, which supplies the sphincter pupillæ through the ciliary ganglion and the short ciliary nerves. By stimulation of the oculo-motor nerve, contraction of the pupil is produced; by its section or paralysis, dilatation of the pupil.

Dilatation of the pupil is dependent upon the sympathetic, which through the long ciliary nerves supplies pupillary fibres derived from the cilio-spinal centre of the cervical spinal cord. Irritation of this centre or of the cervical sympathetic produces dilatation, and paralysis of it contraction of the pupil.

Lastly, the vessels of the iris are of moment in determining the width of the pupil, since by their distention the iris is widened and the pupil consequently contracted, and vice versa.

The width of the pupil may be affected by purely *mechanical* factors. This is the case, for instance, with the contraction of the pupil that regularly occurs when the aqueous escapes. This contraction is of practical importance in the performance of many operations. When, for instance, we perform discission of cataract through the cornea, we take care not to let the aqueous escape, since the consequent contraction of the pupil would expose the iris to greater pressure from the swelling lens. That this contraction owes its origin to purely mechanical causes, is deduced from the fact that it occurs even in the eye of a dead man when the aqueous is let out.

Dilatation of the pupil manifests itself by a sense of dazzling. Sometimes patients also allege that objects appear smaller (micropsia). This phenomenon, however, does not depend on the dilatation of the pupil, but on the paralysis of accommodation, which is generally present at the same time, and is therefore also observed when the latter alone is present. (For its explanation, see § 380.)

In *contraction of the pupil,* sometimes—i. e., if spasm of accommodation is at the same time present—objects appear larger (macropsia). Moreover, obscuration of vision is often complained of, because less light enters the eye through the contracted pupil. and furthermore light adaptation is interfered with (Grafe). In very marked miosis, whether due to miotics or appearing spontaneously (e. g., as the result of tabes), the pupil is frequently found to be irregular and slightly angular.

366. Reflex Reaction; Light Reaction.—The *reaction* of the pupil takes place involuntarily and unconsciously. It is either reflex, in which case the stimulus is transmitted from centripetal nerve channels to the nerves of the iris, or it is associated, in which case the pupillary fibres of the oculomotor nerve are set into action simultaneously with other fibres of the same nerve.

The *reflex action* of the pupil is set in action—

(1) By *light.* This produces *contraction* of the pupil, while conversely, as the illumination diminishes, the pupil dilates. The reflex arc in this case passes through the optic nerve to the superior corpus quadrigeminum, thence to the nucleus of the oculo-motor nerve, and then along this nerve to the eye. The reaction always affects both eyes—i. e., if the light falls into one eye alone, the pupil of the other eye also always contracts (consensual reaction).

The consensual reaction appears simultaneously with the direct and is very nearly as great. For this reason the size of the pupils in the two eyes is under ordinary conditions sensibly the same. Inequality of the pupils is usually pathological (see § 376).

The reaction of the pupil to light is exceedingly sensitive, and is employed with great advantage to determine objectively whether an eye has any sensation of light or not (particularly in children, malingerers, etc.). For the method of determining the light reaction, see § 392.

The diagnostic usefulness of the light reaction is enhanced by the fact that the consensual reaction enables us to tell from the examination of one eye whether perception of light exists in the other. For if light perception or light conduction is absent in one eye, not only will the direct reaction be absent in that eye, but the consensual reaction will be absent in the other, even though the direct reaction is retained in the latter (cf. §§367, 377).

The width of the pupil is not precisely proportional to the quantity of light that enters the eye, but is the same with light of the most varying degrees of intensity, so long as the intensity of the light remains uniform. When this intensity changes the pupil alters its diameter accordingly, but if the light is maintained at this new intensity the pupil gradually returns to its former diameter when the retina has become completely adapted to the new intensity.

Path of the Light Reflex.[27]—The stimulus to pupillary contraction emanates from the retina (probably from the rods and cones). The macula lutea and the region immediately adjoining are the parts most concerned in it and are hence called the pupillo-motor zone; but the other portions of the retina, even to the most peripheral, can also originate this impulse, though to a much less degree, so that the pupil contracts but little when light is directed on them, and contracts very much more when light is thrown directly on the fovea. From the retina the stimulus is conveyed into the optic nerve through fibres (C, D, C_1, D_1, Fig. 168), which are probably distinct from those conveying visual impressions, since in optic-nerve affections sight may be greatly reduced or abolished without a corresponding change in the size of the pupil, and in rare cases the reaction to light is retained even when the eye is completely sightless through inflammation or compression of the nerve (see § 367). Like the visual fibres the pupillary fibres after passing through the optic nerve undergo a semidecussation at the chiasm (see Fig. 168 and § 233), thus passing into both optic tracts, E and E_1. From the optic tract, which they leave in front of the lateral geniculate body, they pass through the brachium conjunctivum to the superior corpus quadrigeminum. Here, according to the most probable view, they terminate; fibres that start from this point putting them in communication through the fasciculus tecto-bulbaris and then through the posterior longitudinal fasciculus with the oculo-motor nucleus of the same side and by decussating fibres (K, K_1, Fig. 168) with the oculo-motor nucleus of the side opposite. The left eye is thus connected with the left oculo-motor nucleus partly by direct fibres ($C\ E\ G\ I$) partly by those ($D\ E_1$, G_1, K_1) which have undergone a double decussation (once at the chiasm and again in front of the nucleus). It is connected with the right oculo-motor nucleus by fibres which have undergone a single decussation either at the chiasm ($D\ E_1\ G_1\ I_1$) or in front of the nucleus ($C\ E\ G\ K$). From the oculo-motor nucleus the efferent fibres pass through the

[27] In this and the following sections much use has been made of the summary by Lutz.

inferior division of the third nerve to the branch which supplies the inferior oblique and which they leave, along with the fibres subserving accommodation (emanating from M) in the twig forming the short root of the ciliary ganglion. From the ganglion they are continued in the short ciliary nerves, which to the number of 15 or 20 pierce the sclerotic round the optic nerve and pass forward in the chorioid and ciliary body to the sphincter iridis. (The fibres subserving accommodation pursue the same course, but terminate in the ciliary muscle). The direct light reflex is effected by the afferent fibres which decus-

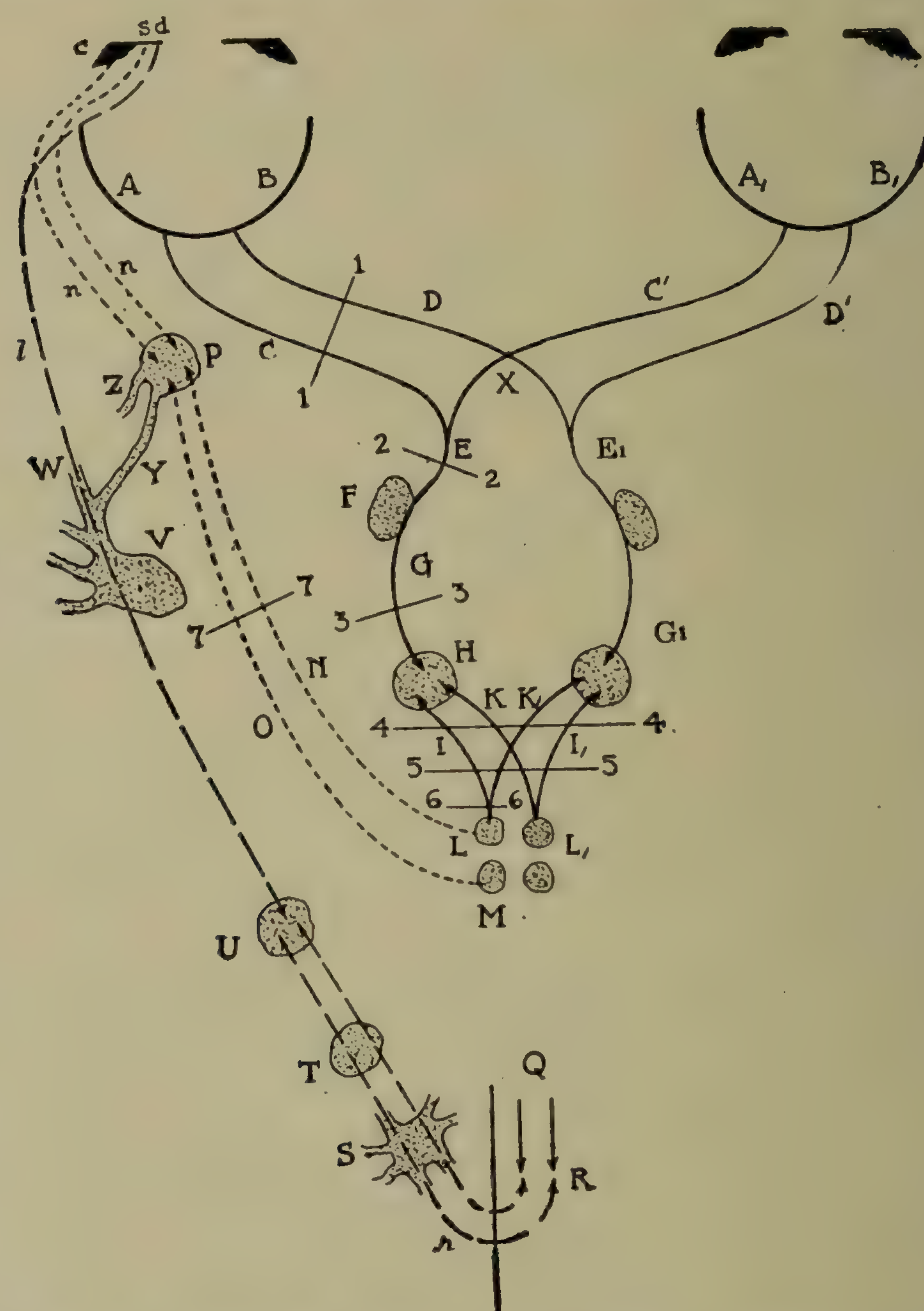


FIG. 168.—PUPILLARY PATHS (modified from Parsons and Lutz).

A, A_1, left halves; B, B_1 right halves of retinæ. C, direct, C_1, decussating pupillary fibres from A, A_1. D, D_1, the same from B, B_1. C and C_1 join at chiasm, X, to form pupillary fibres, E, of left optic tract. In front of F, the lateral geniculate body, they leave the tract and pass in the brachium conjunctivum, G, to the superior corpus quadrigeminum, H. From here they are continued as the fibres I, K, the former running to the pupillary centre, L, in the left oculo-motor nucleus, the latter to the similar centre, L_1, of the right nucleus. The fibres I_1, K_1, of the right side, are similarly distributed to the both nuclei. Efferent fibres, N from L, and O from the centre for accommodation, M, pass in the left oculo-motor nerve and finally through its branch to the inferior oblique (see Fig. 169), to the ciliary ganglion, P. They are thence continued in the short ciliary nerves, n, to the sphincter iridis, s, and the ciliary muscle, c. The short ciliary cerves also contain sympathetic fibres distributed to the blood-vessels of the iris and sensory nerves which are continued back through the sensory root Y, of the ciliary ganglion to the naso-ciliary nerve. The pupillo-dilator paths begin in the afferent sensory fibres, Q, running to the cilio-spinal centre, R, in the cervical cord. Thence rami communicantes, r, pass through the ganglion stellatum, S, and the inferior cervical ganglion, T, to the superior cervical ganglion, U. From here they are continued through the cervico-gasserian branch of the sympathetic to the gasserian ganglion, V, which they leave in the ophthalmic division of the 5th nerve and finally in its naso-ciliary branch, W. From this as the two long ciliary nerves, (l) they pass to the dilatator pupillæ, d, Z, sympathetic root of ciliary ganglion derived from carotid plexus. Sections at *1* causes complete unilateral blindness and abolition of light reflex when left eye is illuminated; at *2* hemianopsia, with theoretically abolition of light reflex when A or A_1, but not when B or B_1 is stimulated (hemikinesis); at *3*, hemikinesis without hemianopsia; at *4* or *5* bilateral Argyll-Robertson pupil; at *6* Argyll-Robertson pupil in left eye; at *7*, paralytic mydriasis with paralysis of accommodation. Break in the dilatator path anywhere from R to d will cause paralytic miosis with absence of sensory reaction.

sate at both chiasm and nucleus or at neither; the consensual light reflex by the fibres which decussate at the chiasm, but not at the nucleus, or vice versa.

Cortical Reflex—If in a darkened room we place a source of light to one side of the eye and make the person upon whom we are experimenting look straight ahead into the darkness, his pupil is dilated. If then we direct the person to concentrate his attention on the light without looking at it, that is while maintaining a straightforward gaze, the pupil contracts (*cortical reflex* of Haab). The existence of this reflex argues a connection between the cerebral cortex and the pupillary centres of the oculo-motor nucleus. This connection is further shown by the fact that stimulation of the calcarine cortex provokes marked miosis.

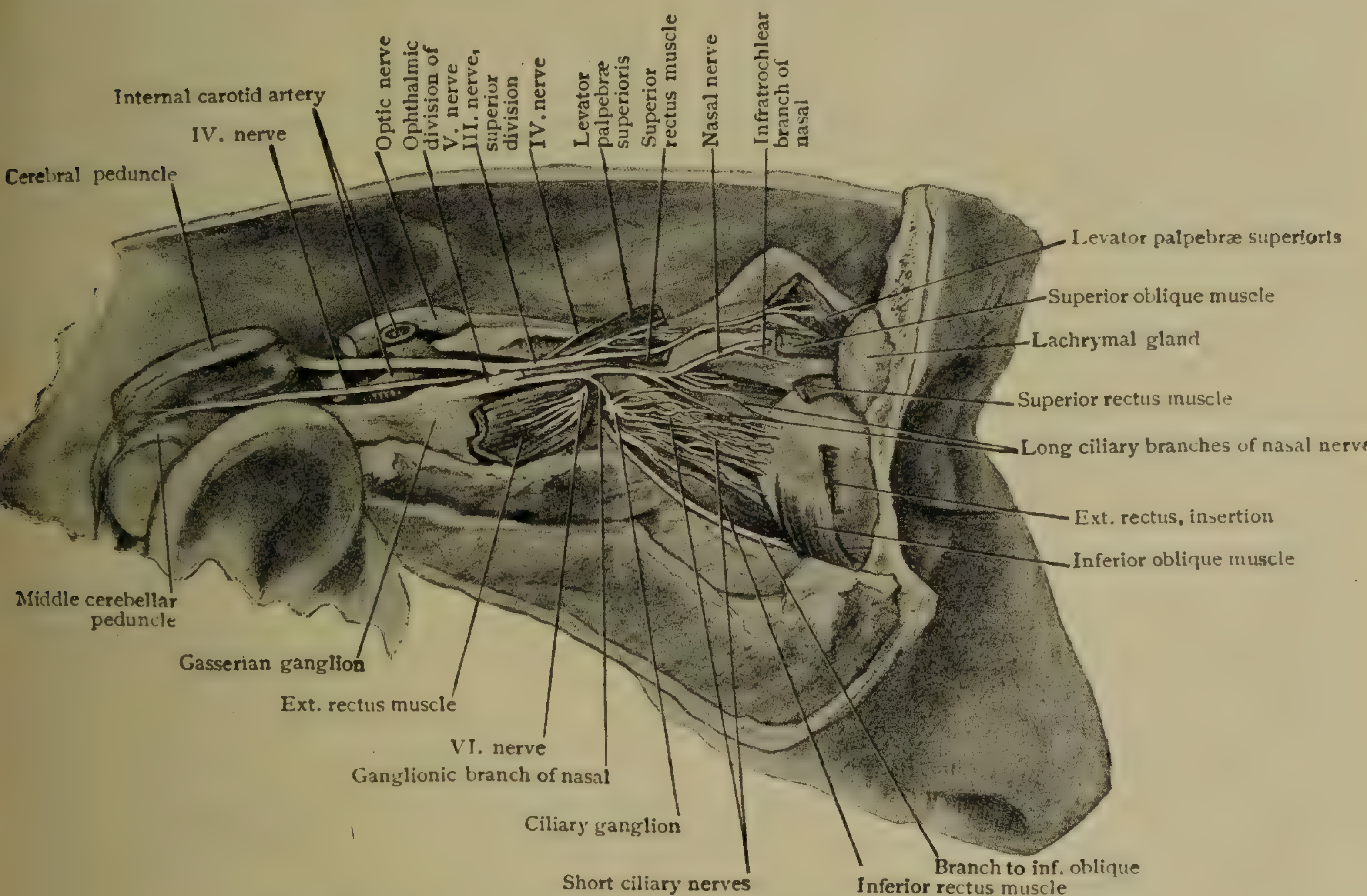


Fig. 169.—Ciliary Ganglion and Connections (after Piersol).
Dissection of right orbit after removal of lateral wall; external and upper eye muscles have been cut to expose ganglion and nerves.

367. Relation of Reaction to Light Perception.—In testing the perception of light by the reaction of the pupils, we must not lose sight of the fact that there are cases in which, although the perception of light is present, the reaction is absent; and, conversely, cases in which, with good reaction, there is yet no perception of light.

(*a*) The cases in which the *pupil does not react to light, although the perception of light is present,* are frequent. They are distinguished by the fact—of considerable diagnostic importance—that, if the affection is unilateral, both direct and consensual reactions are abolished in the affected eye, but both are retained in the other.[28] They may have the following causes: (1) Changes in the iris itself, such as inflammatory swelling, adhesion, increase of tension, laceration in the sphincter, atrophy of the tissue, etc. (2) Disturbances in the motor portion of the reflex path, i. e., in the oculo-motor nerve. Under this head belong paralysis of the oculo-motor terminals in the iris itself produced by mydriatics, and paralysis of the trunk of the oculo-motor nerve or of its nucleus of origin. In all the cases above mentioned the synergic reaction with convergence and accommodation

[28] In some exceedingly rare cases of unilateral Argyll-Robertson pupil the consensual reaction is retained in both eyes (Lutz). See § 377.

is impaired or quite abolished along with the light reaction (*absolute* or *total iridoplegia*). (3) Interruption of the afferent path for the light reflex close to the oculo-motor nucleus. We then find that the reaction for accommodation and convergence is retained, while the reaction for light has disappeared (*Argyll-Robertson pupil* or *reflex iridoplegia* see § 377).

(*b*) It also happens that the *reaction of the pupil to light is present, without there being any perception of light.* This occurs when the lesion is situated high up in the optic pathway. The fibres of the optic nerve ascend to the cerebral hemispheres and terminate in the cortex of the occipital lobe (*B*, Fig. 110). But, some time before this takes place, those fibres (*m*) of the reflex arc which pass to the centre for the pupillary movements branch off from the optic tract. If, then, the optic pathway is interrupted above the place where they are given off (e. g., at *ee*), stimulation of the optic nerve fibres no longer reaches the cerebral cortex and hence excites no perception, and yet the pupillary reflex is still regularly produced. The same thing would happen if the cerebral cortex itself were through some lesion incapacitated for performing its functions. In both of these cases, however, the lesion of the optic pathway would have to be bilateral, as otherwise hemianopsia and not blindness would be present (see § 277). For this reason it is clear why such cases, in which blindness exists, even though the examination of the eye gives negative results and the reaction of the pupil to light is preserved, are very rare (occurring, for example, in uræmic amaurosis, see § 242), so that under these circumstances our first thought would be of simulation or of hysteria, and we would examine for these conditions first. Cases, however, do occur, although very rarely indeed, in which the reaction of the pupil to light is retained although the eye is completely blind, and this may occur even when the blindness is evidently due to disease of the optic nerve itself (Wilbrand and Saenger). This can be explained only on the hypothesis that the fibres for the conduction of the light reflex (§ 366), are more resistant than those conveying visual impressions. The translator has seen one such case. So, too, as Leber points out, in certain forms of complete blindness due to retinal degeneration a very slow reaction to light occurs, the pupil dilating very gradually in the dark and contracting again very gradually on return of light. Such a reaction, again, is no proof of the presence of light perception.

368 Sensory Reflex.—(2) Toward *sensory stimuli*, no matter what part of the body they affect, the pupil reacts by *dilating*. Hence, in deep sleep, and also in profound narcosis, in which sensory stimuli no longer produce reflexes, the pupil is very much contracted, dilating, however, the moment that waking from the sleep or from narcosis occurs. Strong psychic stimuli—e. g., fright—in like fashion produce dilatation of the pupil.

Path of Sensory Reflex.—The stimulus to dilatation of the pupil is communicated through various sensory nerves, especially those of the face, neck, and shoulders (5th cranial, last cervical, 1st, 2nd, and 3rd thoracic). The further course of these afferent fibres (*Q*, Fig. 168) is unknown but they are apparently connected with the posterior longitudinal fasciculus, through which the stimulus is conveyed to the midbrain and the medulla, and ultimately to the cilio-spinal centres. This centre, discovered by Budge, is situated in the spinal cord—according to Jacobsen in the ganglion cells (*R*, Fig. 168) at the apex of the lateral horns of the last cervical and first thoracic nerves. From here the efferent path of the dilator impulse is through the ventral roots of the spinal nerves, and the rami communicantes (*r*) to the superior cervical ganglion (*U*) of the sympathetic, with which they are connected. (On their way to this they pass through the ganglion stellatum and the inferior cervical ganglion, but are not connected with them). From the superior cervical ganglion they pass through one branch (the cervico-gasserian), of the cervical sympathetic to the Gasserian ganglion (V). (The other branch of the cervical sympathetic goes to the carotid plexus and consists of vasomotor nerves simply). From the Gasserian ganglion the path is through the ophthalmic nerve (first division of the fifth), the naso-ciliary nerve, and finally the long ciliary nerves which, two or three in number, enter the sclerotic alongside the optic nerve and pursuing a course like that of the short ciliary nerves terminate in the dilatator iridis.

Dilatation of the pupil is effected by impulses emanating from the cerebral cortex or from the periphery, which act partly by inhibiting the oculo-motor (miotic) centre, partly by stimulating the cilio-spinal (mydriatic) centre.

Ophthalmic Sensory Reflex.—When sensory stimuli act intensely upon the eye itself the pupil contracts instead of dilating. The contraction here is due to the hyperæmia of the iris produced by the stimulus.

369. Associated Reaction.—The associated reaction of the pupil always consists of a *contraction*. It occurs in conjunction with *convergence*, i. e., in synergy with the contraction of the internal recti. Since under physiological conditions every act of *accommodation* is accompanied by a corresponding convergence, and the contraction of the pupil keeps pace with it, too, we have here as a regular thing a uniform consentaneous action of the sphincter pupillæ, the ciliary muscle, and the internal rectus. These muscles are all supplied by the oculo-motor nerve, so that their associated action depends upon a simultaneous excitation of the portion of this nerve that supplies them. The convergence reaction like the reaction to light is of equal intensity in the two eyes. For the method of determining it see § 392.

If a man is told to close the eye tight, but at the same time by holding the lid apart prevents it from closing, we see the pupil contract. This phenomenon comes out most plainly with pupils which otherwise are rigid—*lid reaction* of the pupil (Von Graefe).

370. Size of Pupils.—Since the pupil reacts to stimuli of so many kinds and varying so greatly in degree, it is in a state of constant motion. But, however they vary, the pupils of the two eyes are as a rule, equally large. Inequality of the pupils is usually pathological (see § 376). The mean width of the pupil differs with the individual and also alters with the age. Very greatly contracted in new-born infants, the pupil soon becomes more dilated, and then becomes smaller again in manhood and still more in old age. It is regularly larger in myopes than in emmetropes or hyperopes.

Except in infants the size of the pupil under conditions of ordinary illumination ranges from 2.5 to 5.0 mm.—the usual width being from 3 to 4 mm. in the young and middle-aged and 2.5 to 3.0 in the old.

In old people, the reaction of the pupil becomes sluggish, in consequence of the unyielding character of the tissue of the iris, and especially of the sphincter (rigidity of the sphincter). For the method of determining the width of the pupil and its reactions see §§ 391, 392.

371. Reaction of the Pupil and Ciliary Body to Poisons.—There are poisons which act to stimulate the terminals of the autonomous nervous system in general, i. e., in the eye stimulate the terminals of the oculomotor nerve in the iris and ciliary body, and there are other poisons which act in an antagonistic way upon the same nerves, i. e., paralyze them. The poisons of the muscarine group (including, besides muscarine, eserine and pilocarpine) stimulate, and atropine and its congeners paralyze the interior muscles of the eye. The first named, therefore, cause contraction of the pupil or miosis[29] and are hence called *miotics*, the second cause dilatation of

[29] From *μείωσις*, contraction; hence miosis, and not myosis, as it is generally written (Hirschberg). The derivation of *μυδρίασις* is uncertain. This word was already used by the ancients to signify dilatation of the pupil, and also the blindness that is so frequently associated with it.

the pupil or mydriasis and are called *mydriatics*. In the iris, however, there is also a dilatator which is supplied by the sympathetic and by whose contraction mydriasis is likewise produced. Hence, to the mydriatics in the wider sense of the term belong also those poisons which stimulate the terminals of the sympathetic nerve, namely, cocaine and adrenaline. For the action and therapeutic application of these drugs see §§ 126-130.

Picrotoxine stimulates the nerve centres, not the nerve terminals; producing thus miosis by stimulation of the oculo-motor nucleus. Nicotine is a poison which acts only at the spot when a nerve enters into connection with its last neuron. Thus applied to the ciliary ganglion it produces mydriasis; applied to the superior cervical ganglion it produces miosis.

372. Disorders of Motility of the Iris.—Disorders of motility of the iris manifest themselves in diminished reaction of the pupil to light or convergence or both and particularly in an alteration of the diameter of the pupil. The conditions, or rather the symptoms that may thus be presented comprise excessive dilatation of the pupil (mydriasis), excessive contraction of the pupil (miosis), inequality of the pupils (anisocoria), absence of light reaction, absence of convergence reaction.

373. Mydriasis.—We denote by mydriasis the condition in which either both pupils are abnormally large (over 5 mm. in diameter) or one pupil is decidedly larger than the other, which is demonstrably normal (§376). Normal pupils vary greatly in size (see § 370), and sometimes, especially in young myopes, may be 5 mm. or more. Such pupils are distinguished from those which are abnormally wide by the fact that they are very mobile, reacting sharply to light, while in pathological mydriasis the pupil reacts either slightly or not at all. Pathological mydriasis is either spastic or paralytic.

Spastic mydriasis is due to excessive contraction of the dilatator pupillæ. It accompanies conditions of cerebral irritation of the most widely differing kinds and irritation of the cervical sympathetic. It is produced artificially by cocaine. In spastic mydriasis the pupil usually still reacts (distinction from paralytic mydriases).

Irritation of the cervical sympathetic is characterized by pallor of the face and increased secretion of sweat, dilatation of the pupil, moderate dilatation of the palpebral fissure (from spasm of Müller's muscle), and very slight exophthalmus. It is caused by irritative lesions in the neck and cervical cord (in syringomyelia and as a prodrome of paralysis of the sympathetic). (Wilbrand and Saenger.)

Paralytic mydriasis is far more frequent. It is caused by paralysis of the fibres or the central connections of the oculo-motor nerve, whose branches innervate the intrinsic muscles of the eye—i. e., the sphincter pupillæ and the ciliary muscle. These two muscles are, hence, generally found to be paralyzed simultaneously (ophthalmoplegia interna, or interior). The oculo-motor paralysis may be a diffuse one—i. e., affect several or all of the branches of the nerve—or it may be confined to the pupil (either alone or in conjunction with the muscle of accommodation). Such isolated paralyses occur: 1. In syphilis. 2. In diseases of the central nervous

system, and most frequently in tabes and progressive paralysis, which are also connected with syphilis. 3. Through the action of poisons. Among these belong, above all, the alkaloids known as mydriatics (see § 371), which paralyze the nerve terminals. Paralysis of the pupil and of accommodation also occurs in poisoning by the toxic principles of putrefaction (rotten meat, fish, sausages, etc). In this case the oculo-motor nucleus is the part affected. 4. Sometimes after diphtheria (cf. § 380).

Paralysis of the pupil and of accommodation, occurring after contusions and in case of increased tension, are accounted for by an entirely local lesion of the sphincter and the muscle of accommodation. In the case of contusions, besides the concussion, small lacerations and extravasations of blood into the muscles are also met with. In the case of increased tension the paralysis is produced by the pressure upon the nerves, with which afterward is associated an actual atrophy of the muscular fibres.

The dilatation of the pupil in complete blindness (amaurosis) is not to be regarded as a disorder of motility of the iris, but simply as a physiologic cessation of the pupillary reflex when the perception of light is absent.

374. Miosis.—We denote by miosis, the condition in which either both pupils are excessively small (less than 2 mm.) or one pupil is decidedly smaller than the other which is demonstrably normal (see § 376). Pathological miosis is either spastic or paralytic.

Spastic miosis is due to spasm of the sphincter iridis. It occurs in beginning meningitis, and in association with spasm of accommodation (see § 382) it is produced by the miotics (eserine and pilocarpine) which act by stimulating the short ciliary nerves. A like effect combination of miosis and spasm of accomodation combined with diminution of the intra-ocular tension is produced by ocular injuries (Magitot). For cyclic miosis in oculo-motor paralysis, see § 325.

Paralytic miosis is caused by paralysis of the dilatator pupillæ. It is one of the most important symptoms of paralysis of the cervical sympathetic. Moreover, it very frequently accompanies spinal lesions, especially tabes dorsalis, and also occurs as a spinal symptom in progressive paresis. This *spinal miosis* is often associated with reflex iridoplegia (see § 377).

Spinal miosis may also be spastic. Paralytic is distinguished from spastic miosis by the fact that in the former the pupil does not and in the latter it usually does dilate under cocaine.

The miosis produced by nicotine is paralytic; and that caused by opium, morphine, or general anæsthesia results from the abolition of the inhibitory sensory influences that normally flow from the periphery to the oculo-motor nucleus. The like is true of the miosis of sleep (cf. § 368).

Paralysis of the sympathetic is characterized by a series of symptoms which Horner was the first to describe fully. The pupil is contracted through paralysis of the fibres which dilate it—a fact which is particularly manifested in the non-dilatation of the pupil when the eye is shaded. The difference between the diameter of the pupil in the two eyes is hence more striking in a feeble than in a bright light (Fig. 170). The palpebral fissure is smaller in consequence of drooping of the upper lid. This moderate ptosis is caused by the paralysis of the musculus tarsalis superior, which is supplied by the sympathetic. The eyeball itself often seems to have sunk back into the orbit and to be less tense. An important symptom is the difference in the fullness of the vessels on the two sides of the face. In a recent paralysis the face is redder and warmer on the paralyzed side; afterward, the opposite is the case, the paralyzed side being paler, cooler, and no

longer sweating (a thing easily made out in men by the hat lining, which is stained with sweat on one side and not on the other). The causes of sympathetic paralysis are often the coarser lesions, and most frequently pressure on the nerve from tumors in the neck, such as goitre or enlarged lymphatic glands. More rarely it is caused by traumatisms (among which are fractures of the clavicle) and by operations, e. g., the extirpation of tumors and sympathectomy. Furthermore, tuberculous affections of the apex of a lung, lesions of the spinal cord, such as tabes, syringomyelia, or injury of the uppermost part of the cervical cord, and neuritis of the cervical plexus, have been observed as the cause of sympathetic paralysis. In most cases it is impossible to find a cause. The paralysis, in addition to the moderate disfigurement due to the slight ptosis, causes no annoyance, and not infrequently is first discovered accidentally by the physician. It is commonly incurable.

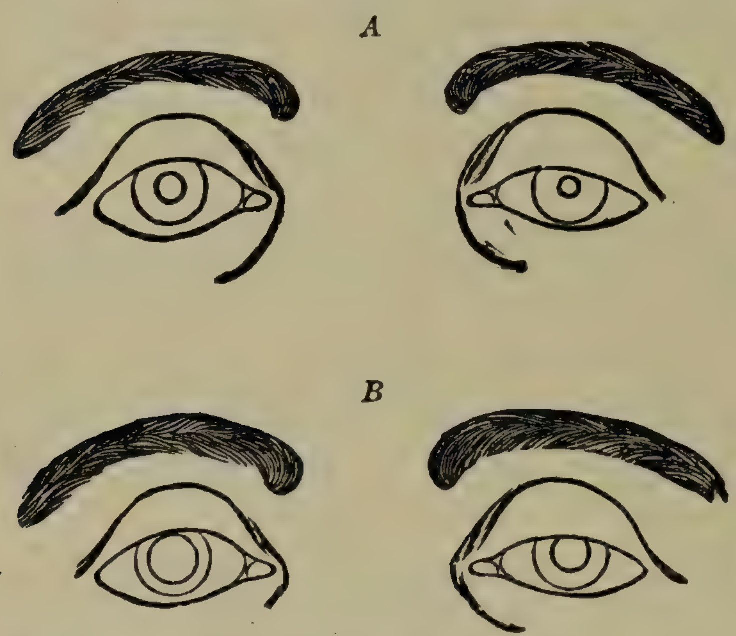


Fig. 170.—Left-sided Sympathetic Paralysis.

A, eyes when turned toward the light, *B*, eyes when turned away from the light. In both cases the left pupil is narrower than the right, but the difference is more pronounced in the second case when the right pupil is widely dilated because of the darkness. The upper lid on the left side hangs somewhat lower (ptosis), than on the right side, and moreover the lower lid rises somewhat higher, as can be made out from the fact that in this eye the distance between the border of the lid and the lower margin of the cornea is smaller. The greater elevation of the lower lid is caused partly by the paralysis of the musculus tarsalis inferior, partly by the enophthalmus, which, however, in this case amounted to no more than a millimetre.

375. Enlargement and diminution of the pupil in themselves cause no considerable interference with vision if they are not combined with paralysis of accommodation. Hence they are but seldom, in themselves, the subject of treatment; their chief significance lies in their being an important symptom of a deeply seated and widespread disorder. This latter, therefore, as a rule, is alone the object of treatment. Paralytic mydriasis may be treated symptomatically by miotics and electricity.

Under the name of *hippus* is designated a pathological condition which consists in a constant and rapid change in the diameter of the pupil. Since even under physiological conditions the pupil never remains quite at rest, it is hard to draw the line between physiological and pathological inquietude of the pupil, and many believe that a genuine hippus does not exist. Quite different in character is periodically alternating (*cyclic*) *contraction and dilatation* of the pupil noted (sometimes in conjunction with a simultaneous alternating dilatation and contraction of the palpebral fissure) in certain rare cases of oculo-motor paralysis (see § 325).

376. Anisocoria.—Owing to the fact that the consensual reaction equals the direct and that the sensory as well as the convergence reaction affects each eye to the same degree, the pupils of each eye are regularly equal. If they are not, we call the condition anisocoria.[30]

When only one eye is illuminated, the pupil of this eye is somewhat narrower than that of the eye which is not illuminated, perhaps because in addition to the reflex contraction on the illuminated side there is also a direct contraction due to the illumination of the iris tissue itself. In lower animals the pupil even in an excised iris contracts distinctly to light, and the same thing is true, though to a very slight extent, of the human

[30] From ἀ, privative: ἴσος, equal, and κόρη, pupil.

iris also. However, the difference between the two pupils when unequally illuminated is so slight in man that it can be demonstrated only by precise measurement, so that, practically speaking, we may say that even in this case the pupils are equal. Cases, however, are quite frequent in which, even when both eyes are alike exposed to the light, there is an inequality of the pupils (anisocoria). This is found in some 10 per cent of persons examined at random, and, while it is often pathological and is always to be regarded with suspicion, indicating, for example, in some cases the presence of tuberculosis (translator), there seems no question but that it may occur in those who are perfectly healthy or who at most suffer from a functional neurosis. In such cases it may be transient. In this non-pathological anisocoria, the pupils are round, and the pupillary reactions are perfectly normal—a feature which distinguishes it from most cases of pathological anisocoria (Uhthoff). For the reasons given anisocoria can never take its origin from the centripetal fibres (optic-nerve fibres), but is always caused by a unilateral disturbance in the centrifugal channels (oculo-motor nerve and its centre or its connections with the sympathetic) or by purely mechanical causes, such as adhesions or rupture of the iris.

If the inequality of the pupils is slight and neither pupil shows any great variations from the average pupillary width, we may be in doubt as to which of the two pupils is to be regarded as the abnormal one. If for example, the right pupil is somewhat larger than the left, it is a question whether a mydriasis of the right eye or a miosis of the left is present. The distinction is to be made by carefully testing the reaction of each pupil, this being done by alternately shading and illuminating both eyes at once. The pupil which shows the less marked reaction of the two is the diseased one.

377. Absence of Light Reflex; Reflex Iridoplegia.—Absence of the light reflex with retention of the convergence reflex is called reflex iridoplegia or Argyll-Robertson pupil. It is to be distinguished from total iridoplegia, in which the iris is completely immobile. Argyll-Robertson pupil is a very frequent and usually early sign of tabes and general paresis (cf. § 816).

It is so rare in other nervous diseases that it furnishes an important diagnostic evidence that one of these two diseases is present or impending. In 25 to 30 per cent of the cases it is associated with marked miosis (so-called spinal miosis), but is also found with a normally wide pupil. In case the diagnosis is doubtful the state of the pupil is often of importance—absolute iridoplegia with dilated pupil being rather an evidence of cerebral lues, and reflex iridoplegia with contracted pupil an evidence of tabes or general paresis. Reflex iridoplegia has also been seen in lethargic encephalitis and after poisoning by lead and tobacco.

The lesion causing Argyll-Robertson pupil affects the afferent pupillary fibres close to the oculo-motor nuclei. If, as is almost always the case it affects the direct and decussating fibres to both nuclei (i. e., occupies position 4 or 5, Fig. 168), the reflex iridoplegia affects both eyes. In certain rare cases the lesion is limited to the direct and decussating fibres to one nucleus (6, Fig. 168), and then the reflex iridoplegia affects only the eye on that side. In exceedingly rare instances the lesion seems to be confined to the direct fibres to one nucleus (Lutz) or possibly to the nucleus itself, so that the direct, but not the consensual reaction is abolished in one eye only. Reflex iridoplegia is usually associated with loss of the sensory reaction (§ 368).

HEMIKINESIS.—Theoretically in tract lesions the light reaction of the pupil should be abolished only when that half of the retina which has been rendered insensitive by the lesion receives the light stimulation (hemikinesis). As a matter of fact, in these cases we usually find an ordinary Argyll-Robertson pupil with hemianopsia (see § 277).

In cases of convergence paralysis the convergence reaction of the pupil is slight or absent while the light reaction is retained (*inverse Argyll-Robertson pupil*).

XIV. Anomalies of Accommodation

378. Varieties of Accommodative Anomalies.—A necessary preliminary to the consideration of the anomalies of accommodation is a knowledge of the physiology of the process (§§ 176-182) and particularly of the limits of the normal accommodation (see table in § 182). From the latter it can, for example, be deduced that at 40 the average man has a range of 5.8 D, while in others who still can be regarded as normal the range varies between 3.4 and 7.9 D. A range lying persistently outside of the limits given must be regarded as abnormal. The accommodation in this case may be either too low (*insufficiency* and *paralysis* of accommodation) or too high (*excess* and *spasm* of accommodation). It may also be unduly *inert;* and it may be *unequal* in the two eyes.

When measuring the accommodation in order to ascertain whether these anomalies are present, we must first provide the patient with the full correction of his refraction as found under a cycloplegic. Otherwise we might, for example, be unable to tell whether a remote near point meant weakness of accommodation or latent hyperopia (cf. § 197).

379. Insufficiency of Accommodation.—(*a*) *Ill-sustained accommodation.* In many cases the range of accommodation is normal for the age, i. e., above the minimum shown in Fig. 91, but soon gives out so that it drops below the normal when the patient tries to use his eyes. This occurs in conditions of weakness in general (convalescence from disease, etc.) and in the other conditions noted below as the causes of accommodative insufficiency—of which, indeed, this ill-sustained accommodation is often but an initial or a terminal stage.

(*b*) In true *accommodative insufficiency,* the patient's accommodative power remains persistently below the normal limit for his age.[31] It is a fairly frequent condition.

It may be *caused* either by undue rigidity (accelerated sclerosis) of the lens or by weakness of the ciliary muscle. In the former type, which may properly be called premature presbyopia, the accommodation drops in much the normal fashion from year to year, but in any one year is always below the normal standard.

In the type due to anomalies of ciliary action, the insufficiency varies from time to time, often in a quite haphazard manner.

The causes producing simple premature presbyopia are unknown. The causes of ciliary weakness are as follows:

1. Toxic conditions due to infectious disorders (intestinal toxæmia, tuberculosis, influenza, whooping-cough, measles, and tonsillar and probably dental infection).
2. Nasal obstruction. This appears to be a frequent cause.
3. Disorders of the internal secretions.
4. Metabolic disorders (diabetes).
5. Neurasthenia and anæmia, brought on by overwork, impaired nutrition, and similar causes.
6. Vascular hypertension.

[31] Obviously, also, a patient must be held to have at least a *relative* insufficiency of accommodation if his accommodative power, although above the minimum normal limit for his age, is persistently below his own lower normal limit, as shown by later tests.

7. Probably excessive action of light.
8. Glaucoma in the prodromal stage.
9. Medicinal agents (cycloplegics—see § 380) when applied in a diluted state so as not to cause a complete paralysis.

In nearly half the cases, especially in those due to nasal obstruction and neurasthenia, accommodative insufficiency is associated with convergence-insufficiency and frequently with retinal hyperæmia and persistent dense injection of the conjunctiva.

The *symptoms*, which in part are caused by the associated conditions, are asthenopia—either simple tiring of the eyes or tiring associated with eye-ache (sometimes of great severity), with marked irritation and burning of the eyes or with headache; blurred vision, especially for near work; vertigo; aprosexia; photophobia (quite a marked symptom in some cases). An occasional finding, especially in the cases with marked convergence-insufficiency, is a concentric contraction of the visual field (cf. § 274).

The intensity of the symptoms varies greatly. In many cases they are such as to render eye work almost impossible. On the other hand, especially in simple premature presbyopia, the only complaint may be of impaired vision for near, which is remedied at once by suitable reading glasses.

The *course* and *duration* naturally depend on the cause. Recurrences may take place, sometimes at long intervals. In simple premature presbyopia the course goes on much as in an ordinary presbyopia, but the necessity for the use of an additional glass for reading begins much earlier—at forty or before.

The *treatment* is directed to the cause, whenever we can discover the latter. The removal of toxic conditions by remedying an intestinal indigestion or a dental or tonsillar infection; the relief of anæmia and malnutrition by the use of tonics and proper diet; the regulation of vascular anomalies; the removal of nasal obstructions; treatment addressed to abnormal glandular conditions—all these are to be employed when required. Eye-strain should be relieved by proper glasses. An associated conjunctival injection should be treated with zinc or other astringents.

For the insufficiency itself the following means are used:

1. For the blurred vision at near, the distance glass may have to be supplemented by a proper addition for reading (presbyopic correction for an unnatural presbyopia). This should not be used unless necessary nor kept up too long for fear of perpetuating the condition.
2. Even when the vision is not much blurred, this presbyopic addition may be necessary, especially if there is an associated convergence-excess.
3. The accommodation may be stimulated directly by means of reading exercises, or exercise in focusing on the fine test-object (line) used in measuring the accommodation (Fig. 88).
4. Especially in cases associated with convergence-insufficiency it is useful to stimulate both accommodation and convergence by exercise with prisms, bases out, when the eyes are directed at a near object. This is supplemented by practice in converging both eyes on a dot or line which is brought closer and closer to the eyes until it doubles.

380. Paralysis of Accommodation.—Paralysis of accommodation[32] is simply an insufficiency carried to an extreme degree. The term, however, is particularly applied to the cases due to the more serious toxic and organic causes. It produces an extreme reduction of the accommodation below the normal (see Fig. 91), as shown by tests made with reading or with the accommodation line (Fig. 88).

The disturbance which paralysis of the accommodation causes varies greatly according to the refractive condition of the eyes. If an emmetrope is affected with paralysis of the accommodation, reading and writing become perfectly impossible, or at least, in case of incomplete paralysis (paresis) of accommodation, very difficult and possible for only a few moments at a time. Distant vision, for which the emmetrope does not require to use the accommodation, is not affected. In the hypermetrope paralysis of accommodation makes itself still more noticeable, since without accommodation he sees poorly even at the distance. The reverse is true of the myope, to whom the abolition of accommodation causes little or no inconvenience; indeed, in the higher degrees of myopia a paralysis of accommodation is often discovered only accidentally at the time when a careful examination is being made. And in old persons in whom accommodation for near has become impossible owing to sclerosis of the lens, paralysis of accommodation would also necessarily remain undetected. But until the patient has reached the age when the minimum normal accommodation is less than 1.5 D—i. e., until he is 48—paralysis of the accommodation can be made out by the tests, and at 40 and 45 is almost as apparent as in youth (see § 182).

In paralysis of the accommodation, whether produced by disease or artificially by a cycloplegic, the statement is frequently made that objects appear smaller than usual (*micropsia*).[33] This phenomenon is explained in the following way. We estimate the size of an object from the size of its retinal image taken in conjunction with the distance at which we judge the object to be situated. In paralysis of the accommodation, the act of accommodation for any given distance of the object costs us a greater effort than under other circumstances, so that we estimate the accommodation at too high a figure and hence believe the object to be nearer than it really is; but as the retinal image is no larger, we think that the object itself has diminished in size. (Cf. the false localization of objects when viewed with the aid of a paralyzed muscle—§ 313). The converse phenomenon, by virtue of which objects appear larger than normal—*macropsia*[34]—is observed in spasm of the accommodation. This, too, originates in a delusion as to distances, resulting from the disturbance of the accommodation. (For retinal micropsia and macropsia, see § 238).

Paralysis of the accommodation arises from a paralysis of the ciliary muscle or of the oculo-motor nerve, which supplies this muscle. It may be simply one of the symptoms of a complete oculo-motor paralysis, in which cases its etiology agrees with that of oculo-motor paralysis in general (see §§ 324, 325). But in many cases the paralysis of accommodation exists alone, or is at most associated with a coincident paralysis of the sphincter pupillæ. These two intrinsic muscles, which under physiological conditions act in conjunction, are also commonly paralyzed together, so that the paralysis of accommodation is combined with mydriasis paralytica (ophthalmoplegia

[32] Also called *cycloplegia*, from κύκλος circle, and πληγή, stroke. Hence *cycloplegic*, an agent—e. g., atropine—paralyzing the accommodation.

[33] From μικρός, small, and ὤψ, sight.

[34] From μακρός long, and ὤψ sight.

interna or interior). The *causes* of paralysis of accommodation (with or without paralysis of the pupil) with which we are acquainted are as follows:

1. *Poisoning by Toxins.*—Under this head is comprised the paralysis of accommodation which occurs quite frequently in *diphtheria,* developing usually in the stage of convalescence. Another post-diphtherial paralysis most commonly associated with it is paralysis of the soft palate, which manifests itself in the nasal character of the speech and also by the fact that food and liquids readily get from the choanæ into the nose when the patient tries to swallow. Isolated paralysis of the sphincter pupillæ or paralysis of the exterior eye muscles, of the muscles of the extremities, or of the trunk itself, occurs less often. Diphtherial paralysis of the accommodation is usually not associated with paralysis of the sphincter pupillæ. It generally passes away of itself in one or two months as the patient gains in strength, and hence affords a good prognosis. Analogous in their behavior to post-diphtherial paralysis of accommodation are the cases due to *influenza,* which occur pretty frequently during some epidemics. Paralysis of accommodation may also be caused by poisoning by *ptomaines,* and by bacterial infection springing from the *tonsils* or other parts (Veasey, Johnston). Possibly of toxic origin also is the paralysis of accommodation sometimes found in *diabetes* and other metabolic disorders. In all these paralyses due to internal toxins both eyes are almost invariably affected.

2. *Poisoning.*—The most complete paralysis of accommodation, combined with paralytic mydriasis, is produced by atropine and the other cycloplegics (hyoscyamine, duboisine, scopolamine, homatropine). These act not only when administered internally, but also locally when introduced into the conjunctival sac. In the latter case the paralysis may be unilateral, whereas in the former it is bilateral.

3. Brain *syphilis* and tabes and progressive paresis, which are connected with syphilis. In these cases the paralysis of accommodation is frequently unilateral, is usually associated with paralysis of the pupil, and generally resists all treatment. It may also occur with or without paralysis of the pupil in non-syphilitic and in functional brain disease. Paralysis of the accommodation, due to brain disease, may be associated with paralysis of convergence (see § 332).

4. In *contusions of the eyeball,* being then a purely local affection.

It is evident that the accommodation is completely abolished when the lens is luxated, or is removed altogether from the eye, but such cases are not properly denoted as paralysis of the accommodation.

The *treatment* of paralysis of the accommodation must first of all be regulated according to the lesion which lies at the bottom of it, and must seek to effect the cure of this latter by appropriate means.

In post-diphtherial and similar paralyses we institute corroborative measures, giving hearty nourishment, wine, iron, quinine, etc. Strychnine given hypodermically to the point of tolerance may help. For local treatment, pilocarpine is employed. This, besides contracting the pupil, also produces a spasm of the accommodation by contracting the ciliary muscle. This, however, is not of long duration, any more than the miosis is; after some hours the muscle relaxes again and the paralysis returns. Nevertheless,

the contraction of the muscle produced by the miotic appears sometimes to exert a favorable influence upon the paralysis itself, perhaps acting in the same way as faradization does when it produces a good effect in paralyses. Pilocarpine, however, has a really beneficial effect only in those paralyses which in themselves give a good prognosis, i. e., in the post-diphtherial paralyses and those dependent on poisoning. In other paralyses mercury and potassium iodide are more in order but rarely do good. If the paralysis presumably will last a long time the indication is to render work possible by means of proper convex glasses.

381. Accommodative Inertia.—Accommodative inertia is the condition in which the patient changes from one accommodative state to another sluggishly or with difficulty. It is analogous to the slow adaptation that some eyes show in passing from light to darkness, or vice versa. It seems sometimes to be a source of real trouble. In such cases exercise of the accommodation and particularly of the convergence may be tried.

382. Excessive Accommodation.—Excessive accommodation may be due to ciliary overaction (*spasm of accommodation*). Apart from what is called the normal tone of the ciliary muscle, which is abolished by atropine and which at most equals 0.25 D, and apart from the continuous tension of the ciliary muscles in hypermetropes when overcoming their hyperopia so as to see distinctly, a state of permanent excessive tension of the ciliary muscle does occur, which is directly prejudicial to vision since it mainly affects myopic eyes, which by it are made to appear more myopic than they really are. Such spasm of accommodation may be revealed by our finding the myopia higher when we make the subjective test (with glasses) than when we determine it objectively with the shadow test. The confirmatory test is made by instilling atropine which paralyzes the accommodation so that now on making the tests with glasses the true refraction is found. Genuine spasm of accommodation is not very common. In some cases it is produced by prolonged straining of the eyes for near work by poor light (Phelps). Other cases have been attributed to toxic causes, sinuitis, reflex irritation from trigeminal neuralgia, traumatism, etc. (Koenigshofer).

Spasm of accommodation is combated by the instillation of atropine, which must be kept up for quite a long time (four weeks and more). Unfortunately, in most cases when the atropine has been discontinued, the spasm returns after a shorter or longer interval. The refraction also must be carefully corrected.

High degrees of spasm of accommodation, sometimes combined with spasm of convergence (§ 330), are found in hysterical subjects. The artificial myopia may reach 13 D.

An artificial spasm of accommodation of high degree combined with contraction of the pupil develops after the instillation of a miotic (eserine and pilocarpine).

Lenticular accommodative excess is the condition in which the accommodation is above the usual limit, because the lens is less rigid than ordinary. It is the opposite of premature presbyopia, and may be called delayed presbyopia. It causes no trouble.

383. Unequal Accommodation.—We sometimes find the accommodation unequal in the two eyes. Sometimes this is due to the fact that the ciliary muscle is weaker in one eye than in the other. This occurs quite frequently in ophthalmoplegia interior due to syphilis, tabes, traumatism, or poisons. It can be readily understood that these conditions may affect one eye more than the other, or may affect one to the exclusion of the other. Since in these cases the sphincter iridis and the ciliary muscle are usually affected simultaneously, the inequality in accommodation is generally associated with inequality of the pupils—the eye with weaker ciliary muscle having the larger pupil.

When not due to the causes above mentioned, unequal accommodation is usually not associated with inequality of the pupils. In this case, too, it may be caused by unequal action of the ciliary muscle, but most of the cases of this sort seem to be due to an unequal rigidity of the lenses in the two eyes. In other words, it is a form of presbyopia in which the sclerosis of the lens has advanced faster in one eye than in the other. It produces no symptons until the accommodation in one eye or the other has sunk so low as to require the addition of a glass for near work. When this occurs, it is important to take account of the inequality and to determine by experiment whether we shall or shall not make an unequal addition for reading to the distance correction. In some cases such unequal addition is quite helpful (see § 215.)

CHAPTER XVIII

EXAMINATION OF THE EYE

384. General Routine of Examination; History Taking.—In examining an eye patient a systematic routine should be followed, to be elastically varied according to the exigencies of the case. Such a routine should include—

1. Getting the history of the case.
2. Examination of the vision and refraction (Chapter XV).
3. Examination of function (color sense, light sense, field; see Chapter XVI).
4. Examination of the motility (§ 309).
5. Objective examination of the eye.

In taking the *history* inquiry should be made for the various symptoms such as increased secretion, or other evidences of ocular inflammation, pain, asthenopia, headache, vertigo, blurred vision, diplopia, metamorphopsia, head-tilting, etc., likely to occur in eye disease (§§ 104-112) and also for any of the possible causes (§§ 83-103). Furthermore, it is important to get the family history, so as to ascertain the presence of any hereditary tendency or disease.

385. Objective Examination.—Some of the tests for refraction and motility are objective. These have already been considered (§§ 219-225 and 303-308). The remaining objective tests concern the examination of the exterior and interior of the eye.

In making the examination too much stress cannot be laid upon the necessity of proceeding systematically, since otherwise matters of importance may very readily be overlooked. We first, therefore, take a survey of the patient's appearance in general, his attitude, the way he carries his head, and the expression of his eyes and countenance; then we examine the eyes themselves, and in so doing proceed successively from the superficial parts—lids, conjunctiva, and cornea—to the deeper portions.

The *record* of a case, while as condensed as possible, should omit no essential fact. In this regard the statement that conditions are normal is often as important as the presentation of abnormalities. A well-devised system of abbreviations saves space and time and helps greatly.

It is important to form the habit of making *sketches*, however diagrammatic, of the conditions presented. A comparison of such sketches made at different times will show, for example, the advance of a cataract, the enlargement of a conus in progressive myopia, the presence of a hæmorrhage or pigment spot where one did not exist before, etc.

386. Examination of Lids and Tear Sac.—In respect to the lids there are to be considered their position and mobility, the width of the fissure between them, and the way they shut together. The character of the skin lining the lids is examined, and especially at their margins where pathological changes are most often found. Apart from the symptoms of inflammation, which is very apt to be localized at the borders of the lids, the things

that we must look for are whether the palpebral edges have perhaps lost their sharply defined form and outline, whether the cilia are correctly placed, and whether the puncta dip properly into the lacus lacrimalis. At the same time, we must investigate the region of the *tear sac*. Should simple inspection disclose no alteration, pressure with the fingers in this region may make the contents of the diseased sac exude through the puncta. Furthermore, we ascertain whether the small lymph gland in front of the ear is perceptible to the touch and is sensitive to pressure.

387. Examination of Eyeball.—The examination of the eyeball itself is often rendered very difficult by strong spasm of the lids—blepharospasm. This is especially the case in children who, the more the physician attempts to draw the lids apart, squeeze them the more tightly together. In these cases the forcible separation of the lids calls for the greatest caution, since, if this is not observed, and a deeply penetrating ulcer is present, it is easy to cause a sudden perforation of the cornea, nay, even the extrusion of the lens from the eye. By dropping a solution of cocaine between the slightly parted lids we try to diminish their sensitiveness; and for separating the lids we can, with advantage, use Desmarre's elevator (see Fig. 388, *d*), with which we shall less readily inflict an injury than we should do if, by using the fingers, we exerted too great a pressure upon the eyeball. Finally, in many cases it is only by using general narcosis that we can get a sufficiently satisfactory view of the eyes. In spite of all these difficulties we should not be deterred from insisting upon an exact examination of the eyes at the patient's first visit, in order to establish the diagnosis and prognosis and determine the treatment.

The best way to examine a small child is to put it on its back in the lap of the mother or an assistant, who is seated opposite the examiner and who controls the child's arms, legs, and body. The examiner, who is also seated, secures the child's head by gripping it tightly between his knees. The child is thus rendered perfectly immobile, and the examiner has both hands free to evert the lids, concentrate light on the eye, make applications, or do anything else that is necessary.

In regard to the eyeball, we must examine its situation in the orbit (§ 53), its size, its position with respect to the other eye, and its motility (§ 309).

It is often important to determine the distance between the two eyes. This is usually ascertained by taking the distance between the centres of the pupils (interpupillary distance. But what in most cases we really wish to measure is the distance between the visual points. The *visual point*, which usually lies to the inner side of the centre of the pupil, is the point where the visual line (line of sight of the eye) pierces the cornea. To find where it is in any eye, we look at the eye through the aperture of an ophthalmoscopic mirror and, covering the patient's other eye, direct him to look straight at the mirror. The spot where the tiny reflection of the mirror appears on the cornea marks the visual point. Noting its position with reference to the margin of the cornea, we proceed to measure its distance from the visual point of the other eye, similarly determined. This distance naturally varies according as the eyes are converged or not. To determine it when the eyes are not converging at all, we proceed as follows: Observer and patient face each other. The observer's right eye and the patient's left eye are closed or covered. The two uncovered eyes (patient's right, observer's left) look straight at each other. The observer holds a millimetre scale between himself and the patient, with its zero mark in line with the visual point of the patient's right eye. Then without moving the scale he

directs the patient to open his left eye and close his right, while he himself opens his own right eye and closes his left. The patient being now directed to look straight into the observer's right eye, the observer notes what part of the scale is opposite the visual point of the patient's left eye. The reading will give the distance between the visual points in mm. when the visual axes are parallel. It, therefore, also gives the distance between the centres of rotation of the two eyes (*interocular* or *intercentral distance*). This reading will be unaffected by the presence of a squint provided there is no absolute inability on the part of either eye to perform fixation. The interocular distance is usually not far from 61 mm., but may range from 53 to 70 mm. It is usually 2 or 3 mm. less than the *interpupillary distance*, which is similarly measured except that the points between which the measurement is taken are the centres of the two pupils or, better, the temporal edge of one cornea and the nasal edge of the other.

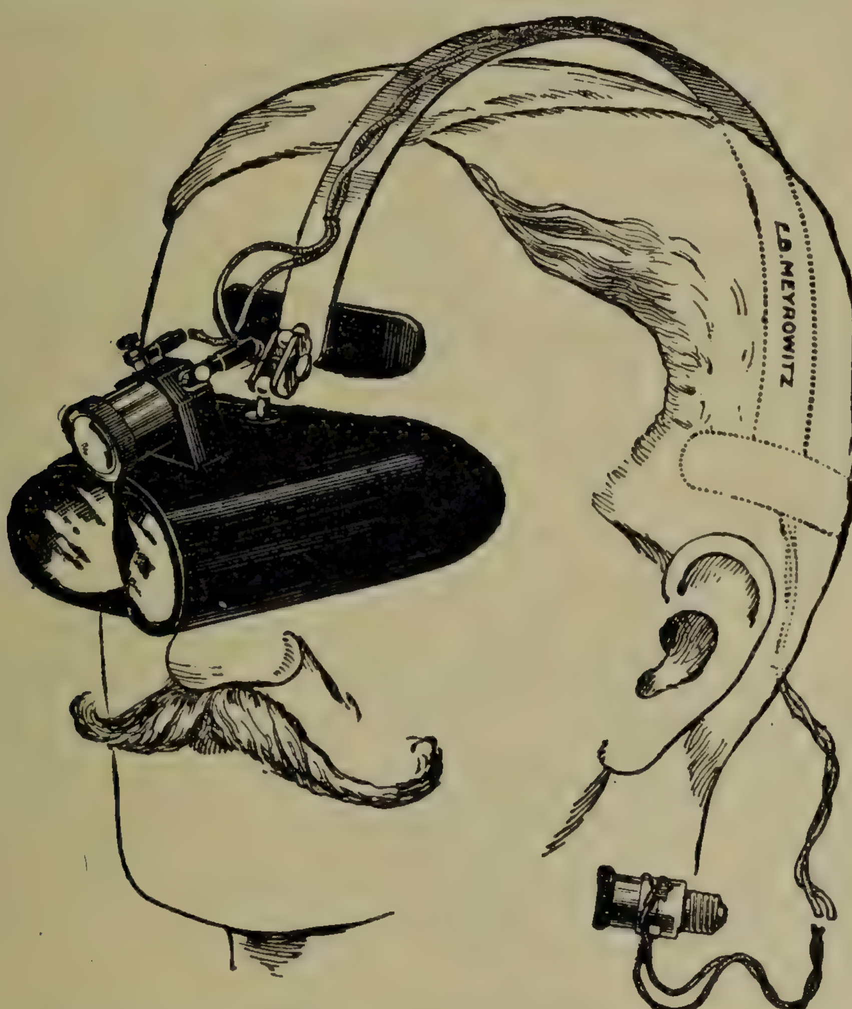


FIG. 171.—BERGER'S BINOCULAR LOUPE WITH ELECTRIC ATTACHMENT.

388. Examination of the Conjunctiva.—The conjunctiva of the lids can be brought into view by everting the latter. With the lower lid, it is sufficient for this purpose simply to draw it down, while the patient is told at the same time to look up. If in pulling the lid down we make slight pressure against it below with a somewhat rolling movement, so as to revolve the lid forward, not only will the lid itself be everted, but the retrotarsal fold will spring up and come into view. With the upper lid, eversion requires a certain degree of skill, which must be obtained by practice. It is the more important to acquire this facility, since it is just the conjunctiva of the upper lid that generally affords the best evidence for the diagnosis of conjunctival diseases, especially trachoma. Further, eversion of the upper lid is very frequently necessary for the removal of foreign bodies. To evert the upper lid we direct the patient to look down, grasp the lashes between the thumb and finger, and draw the lid down and somewhat away from the eyebrow. When the lid is well on the stretch, we place a slender

rod or similar object lengthwise in the groove beneath the brow and just above the tarsus. With the fingers that grasp the lashes we turn the lid sharply outward and upward, keeping it on the stretch all the time and revolving it on the rod as a gate on its hinge. The upper retrotarsal fold can be brought into view if we place a slender rod on the skin behind the everted lid and by a lever-like movement push the latter away from the eyeball.

389. Examination of Cornea; Oblique Illumination and Reflex Images.—An examination of the cornea must have regard to the following points—

1. Its *size* and *form*. Both may be altered either in consequence of congenital defects or because of morbid processes. Overlapping of the cornea by the limbus to an unusual extent, or the presence of marginal opacities in the cornea, not infrequently simulates a diminution in size or an irregularity of form.

2. Its *surface*. This must be examined with regard to curvature, evenness, and polish.

3. Its *transparency*. In this regard we determine the form, extent, and denseness of any opacity that may be present. We make out whether the opacity is in the superficial or in the deep layers of the cornea and further whether it is diffuse or is composed of separate small dots, spots, or striæ. Many an opacity which seems diffuse when seen with the naked eye proves, when viewed with a magnifying glass, to be composed of smaller opacities.

4. Its *sensitiveness*. This is tested by touching the cornea with the tip of a thread or a shred of paper.

In determining alterations in the character of the surface and in the transparency of the cornea, besides a careful inspection with a good magnifier, such as Hartnack's spherical loupe or Zeiss's binocular microscope,[1] there are two main artifices in use—oblique illumination and the examination of the corneal reflex.

Oblique (or *focal*) *illumination* consists in the concentration of light upon a certain portion of the cornea by means of a convex lens. This important method, although already employed by Himly, Mackenzie, and Sanson, was yet very little known formerly, and first obtained general currency through the efforts of Helmholtz. A light (candle or lamp) is placed beside and somewhat in front of the patient. (See Fig. 172.) Then, by the aid of a strong convex lens (of 15–20 D), the rays are concentrated to a cone of light, whose apex is made to fall upon the portion of the cornea to be examined. The point so illuminated stands out with special distinctness because, on the one hand, a great quantity of light is concentrated upon it, and because, on the other hand, the parts immediately surrounding it remain almost completely in darkness. On this latter ground, oblique illumination gives the most advantageous results, if in its application the

[1] There are various corneal microscopes some of which are used in conjunction with the slit lamp (see infra). The binocular loupes of Jackson and Zeiss, which are attached to the observer's forehead by a head band, have the great advantage of leaving both hands of the observer free, and hence are particularly useful for finding and removing foreign bodies. An ordinary head-mirror often serves the same purpose very acceptably.

room is darkened. By lateral illumination we can recognize opacities in the cornea which are perceptible in no other way. The iris, too, and the lens as well, can be examined in this way by varying the depth to which the light is projected. By so doing we have not merely the advantage of getting very sharp images, but also, from the fact that we can at will vary the depth to which the apex of the conical sheaf of rays is projected, we get information as to the depth at which the objects seen are situated. In applying oblique illumination it is well to use two lenses, one of from two to three inches focal distance, held in one hand, to concentrate the light, the other, held in the other hand, to magnify the image. By carefully focusing the two lenses and by making slight shifting movements with the con-

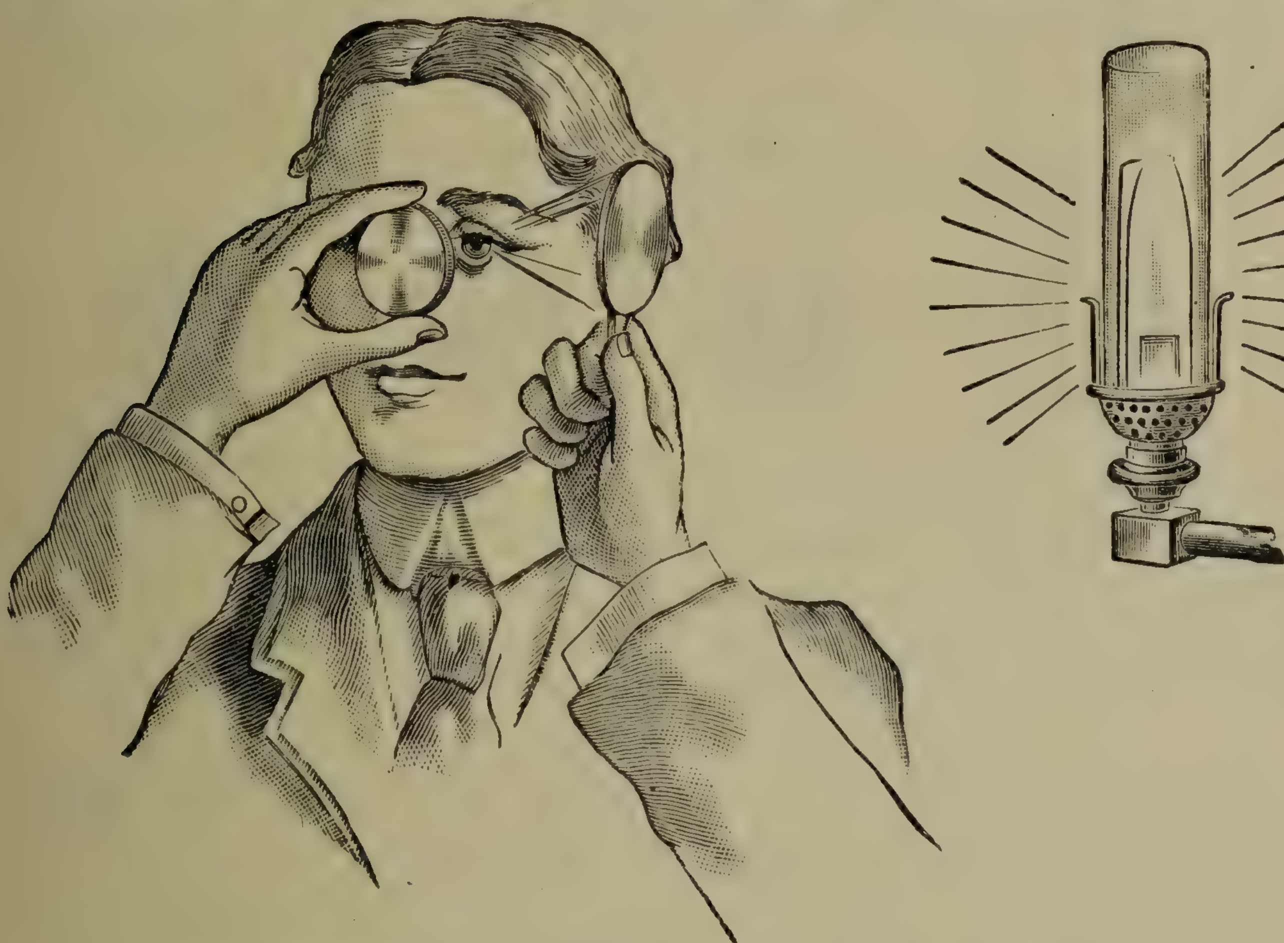


Fig. 172.—Oblique Illumination. (After Posey and Wright.)

densing lens, we get the most accurate idea of the outlines of opacities, the markings of the iris, and other important details. For examining the surface of the cornea it is often advantageous to use oblique illumination by daylight. The patient with his head thrown well back reclines in a chair facing a window, and the observer stands behind him, focusing the daylight or the image of a window on the cornea with a lens and examining the cornea from above with a strong magnifying glass. If the lenses are focused accurately on the surface of the cornea so as to bring to view the flecks of mucus on it, and the patient then moves his eye about, the finest abrasions and also the slightest deformations of the corneal reflex (see Fig. 173) can be made out with ease.

Oblique illumination is best performed with Gullstrand's *diaphragm lamp* (slit or hole lamp). This is so devised as to afford an extremely brilliant light which is condensed by an aplanatic lens into a very sharp and minute image. This image is thrown on the part to be examined, which may be observed with the naked eye or with the corneal microscope. The intense illumination makes it possible to use a high

magnification and owing to its linear or spotlike character the image is confined to an extremely limited area—practically a cross section of the tissue. Furthermore, one is able to study microscopic changes in the conjunctiva, cornea, lens and even the vitreous both from certain features revealed by the intense direct illumination and from the features revealed by the sidelong illumination which the diffracted rays produce in the adjacent parts (Koeppe). Red-free light (§ 410) may be applied by means of the slit lamp and this is useful in examining the conjunctiva and cornea (Koeppe).

Another simpler but helpful modification of oblique illumination is *contact illumination* (Graves). In this the source of illumination (a minute electric light) set in a metal tube is placed in contact with the cornea either at the extreme periphery or at some point nearer the centre. In this way opacities in the cornea and lens are made apparent by light which penetrates deeply because it does not as in the case of ordinary oblique illumination undergo diffusion at the surface of the cornea. This device is specially useful in cases in which view of the deeper parts is obscured by haziness of the cornea.

The second method of examination is by the *reflex images*. These are used to determine (*a*) the *curvature* of the cornea. The cornea acts as a

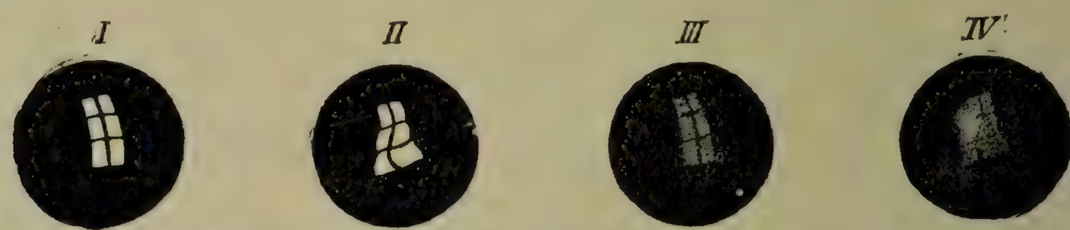


Fig. 173.—Behavior of Reflex Images on the Cornea.

I. In the *normal* cornea. Reflex image brilliant, sharp and regular.

II. In *unevenness* of the surface. Reflex image sharp and brilliant, but irregular. Over the old opacity of the cornea which is associated with flattening, the image of the window is broader because the surface is less convex.

III. In *dulness* of the surface. Reflex regular, but ill-defined and not brilliant. The cornea is stippled and hence the outlines of the reflex image show a very fine zigzag indentation.

IV. In combined *unevenness* and *dulness* of the surface. Reflex image irregular, ill-defined and not brilliant. IV represents II after the cornea has become dull by the development of increase of tension.

convex mirror, the greater the curvature of which the smaller is the reflected image. In order to be able to judge whether the reflex image given by a particular cornea is of abnormal dimensions, we must compare it with the image reflected from another cornea which is healthy, and most conveniently with that from the cornea of the other eye in case it is normal. The diagnosis is easy when the curvature varies at different portions of the same cornea (as, for instance, in keratoconus, in which the central portions have a greater curvature than those at the periphery). In this case we cause the eye to move about in such a way that the reflection of a window opposite falls successively upon different portions of the cornea, and then we see the reflex image becoming larger or smaller according to the varying curvature of the cornea.

We may examine the reflex image in the dark room, using a laterally placed candle as the object reflected (Fig. 176, *a*). In that case, if we use a loupe we can also make out the image reflected from the posterior surface of the cornea (*posterior corneal reflex*). This is erect like the anterior reflex, lies a little to the central side of the latter, and is less bright, less well-defined, and somewhat larger. If the candle is shifted so that the two reflexes approach the centre of the cornea, they get nearer together. Any abrupt change in the distance between the two indicates a corresponding sudden change in curvature of one of the surfaces, particularly the posterior (cf. § 613).

The *evenness* (*b*) of the corneal surface, together with the perfection of its polish, gives to the normal cornea its brilliant lustre. Here, again, the

reflex images afford the best means of testing both of these properties. These images in the normal cornea are *regular* and *clear-cut*. If there is unevenness of the surface, the image at the spot where the cornea is uneven is clear-cut but *irregular;* it looks distorted because its outlines are irregularly bowed in or out (Fig. 173, II). The form and extent of the inequality may be deduced from the kind of distortion of the reflex image. The inequality of the corneal surface becomes particularly evident when we use Placido's keratoscope (see § 225 and Figs. 108 and 109). By the ophthalmoscope, too, we can recognize inequalities of the corneal surface, on account of the irregular astigmatism which they produce (§ 219). The uneven spots upon the corneal surface are either depressions (losses of substance) or elevations. Furthermore, the cornea may be uneven from being wrinkled (rhytidosis[2] corneæ) or from being entirely collapsed (collapsus

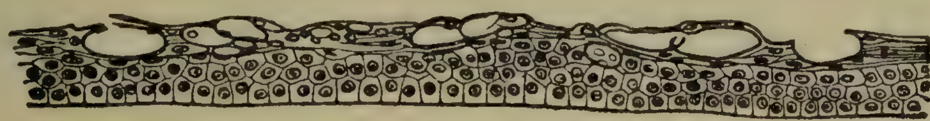


Fig. 174.—Dull Cornea. Magnified 103x1.

The deeper layers of the epithelium are normal. In the superficial layers many of the cells are swollen, and others have ruptured in consequence of being swollen. The former cause small elevations, the latter small depressions in the corneal surface. Cf. also Fig. 375.

corneæ). Both of these conditions occur in cases of great diminution in the tension of the eyeball, hence particularly after the escape of the aqueous or vitreous humors. If (*c*) the *polish* of the cornea is entirely lost, the latter becomes lustreless (dull); it looks like glass that has been breathed upon, or looks as if it had been smeared with grease. The reflex images show their normal size and form, but have *lost their sharp outline.* The cornea, therefore, may be smooth as a whole but at the same time may be dull, just as the surface of a ball of ground glass is. Lack of lustre of the cornea is also caused by the presence of inequalities so minute as to be nearly or quite imperceptible to the naked eye. Such inequalities—in contrast with those of larger size—are situated only in the epithelium and may consist either of elevations or depressions (Fig. 174). The latter are produced by the detachment of isolated epithelial cells in a number of places, thus leaving minute excavations; the surface of the cornea looks as if *stippled* by needle pricks. In other cases we see the roughness of the cornea produced in the opposite way—that is, by its surface being covered with many small elevations, in which case the cornea looks as if it were made of *shagreen.* Here we have to do with multiple elevations, produced by a lifting of the epithelium, and occurring under the form of minute vesicles.

Corneal conditions and particularly opacities can also be examined with the *ophthalmoscope* (page 384).

To demonstrate defects in the corneal epithelium we use *fluorescein.*

If a one-per-cent aqueous solution of potassium fluorescein is instilled into the conjunctival sac, the highly fluorescent liquid penetrates solely into those portions of the cornea that are unprotected by epithelium and such portions consequently are stained a vivid green and, when the excess of potassium fluorescein is removed by washing the eyeball off with water, contrast very distinctly with the uncolored surrounding parts. Fluor-

[2] From ῥυτίς, a wrinkle.

escein is of especial value in demonstrating the very slight defects present in recurring erosions (see § 591.) In very slight defects, the staining also may be very slight, simply a punctate stippling of green showing. In deep ulcers going down to Descemet's membrane only the sides of the ulcer stain with fluorescein, since Descemet's membrane itself does not stain (Butler).

390. Examination of Anterior Chamber.—The anterior chamber must be examined especially as to its depth—that is, whether it is shallower or deeper, as a whole, or whether possibly it is of unequal depth. Further, we look for any abnormal matters which may be present in it, such as an exudate, blood, foreign bodies, etc.

The ophthalmoscope can be used both by the direct method and by axial illumination (§§ 398, 402) to demonstrate the presence of deposits, especially iritic precipitates in the anterior chamber. Furthermore, if the observer, standing to one side, holds the ophthalmoscopic mirror almost at right angles to the patient's line of sight, he can see deposits and adhesions in the angle of the anterior chamber, which ordinarily would not be visible because hidden by the limbus (Salzmann). Such an examination is called *gonioscopy*.

391. Examination of the Iris.—In the iris its color as well as the clearness of its markings must be observed. Special attention must be paid to the margin of the pupil, which should be examined—if need be, with the aid of a loupe—for irregularities (adhesions). In doubtful cases, in order to decide whether adhesions are present, a mydriatic (homatropine, atropine) should be instilled. At the same time we notice whether the iris quivers as the eye makes a sudden movement. Then we determine whether the pupil is round, whether its width is normal and equal to that of the other eye. (If the pupil is of abnormal size we do not neglect to inquire whether possibly some drug has been put into the eye which has caused an artificial alteration of the pupil.) Moreover, we see whether the pupil is centrally placed and of a clear black.

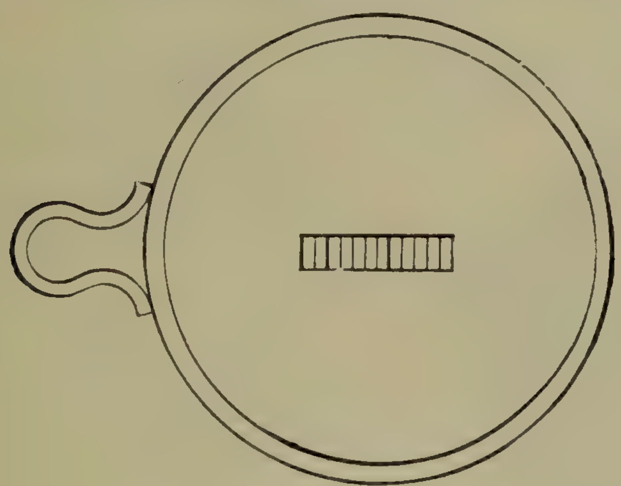
The best way to measure the *width* of the pupil is with the Priestley Smith keratometer (Fig. 175), which, as its name implies, is also used to measure the cornea. For this may be substituted a + 3 D lens, against which is held a millimetre scale. The observer in this case should stand about a foot from the patient's eye and hold lens and scale fairly close to the cornea.

392. Examination of Pupillary Reactions.—We first investigate the *light reaction* of the pupil. To do this we direct the patient to look toward the window and while the left eye is kept screened, we alternately cover and uncover the right, noting whether at the moment of uncovering a contraction of the pupil takes place in both the right eye (direct reaction) and in the left (consensual reaction). The screen is then transferred to the right eye, and the test repeated.

The light reaction of the pupil becomes still more clearly visible if we perform the test in a dark room. In this case the patient looks straight at a light, the eye not under examination being shaded. The observer standing to one side and watching the eye with a magnifying glass focuses the light right on the centre of the cornea by another lens brought suddenly between the eye and the light (Uhthoff). If the faintest light reaction is

present, the pupil will be seen to contract. On the other hand, there will be no reaction for accommodation or convergence, for the patient will have before his eye nothing but a blaze of light which by no possibility can form a clear image on his retina, so that he will make no attempt to focus it.

In testing the light reaction by daylight the patient is directed to fixate a distant object, since, if he shifts his point of vision, the *reaction for convergence* might come into play too. This latter reaction must also be tested. This we do if we make the patient first look at a distance and then, holding an object (finger or pencil) close to his eyes, make him fixate that.



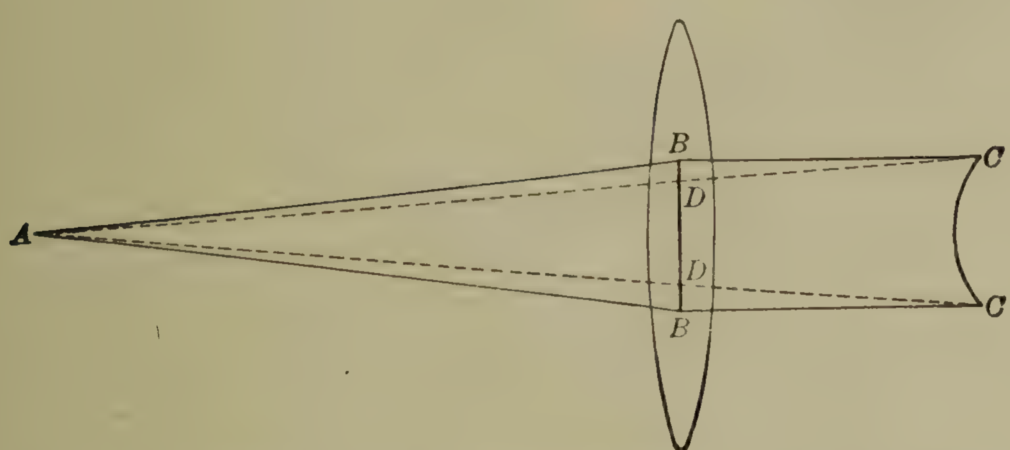


Fig. 175.—Priestley Smith's Keratometer. (After Posey and Spiller.)

This consists of a lens containing a millimetre scale, *B*, *B*. If the observer places his eye at the focus of the lens, *A*, and looks through it at any object, as the cornea or iris, *C*, the size of the latter will be given accurately by the number of divisions that the object appears to cover on the scale.

We must take care that the patient in making this quick change from distance to near does not shift his line of sight up, down, or sideways, nor do anything else that might change the conditions of illumination. We should also test the *sensory reaction* (§ 368). We do this by applying some sharp stimulus, e. g., by stroking the neck with a pin point, when if the reaction is present, the pupil should dilate.

Sometimes, as in testing the hemiopic reaction (hemikinesis; §§ 277, 377), it is necessary to confine the illumination to a portion of the retina of the examined eye. This is done with any apparatus that will throw a small concentrated pencil of rays.

The contraction which takes place in the pupil when suddenly exposed to light is succeeded by dilatation, and this may occur so quickly that the primary contraction escapes us.

393. Examination of the Lens.—Of the lens we see under ordinary circumstances only the small portion of the anterior surface which lies

free in the pupil. If we wish to examine the lens more extensively, we dilate the pupil and use oblique illumination. As long as the lens is still transparent, the ophthalmoscope gives us the best conclusions in regard to its constitution.

Whether the lens is present in the eye at all or not can be determined by investigating the Purkinje-Sanson reflex images. If the candle is placed before the eye and somewhat to one side of it, two brilliant reflections are observed. One of these at once attracts our attention by its size and brilliancy; it is the *anterior corneal reflex*—that is, the erect image of the flame reflected from the anterior surface of the cornea (Fig. 176, *a*). It is this reflex which even from a distance is visible in every eye, and gives to the latter its fire and lustre. The second reflex is quite as bright, but so small that we have to search for it in order to find it. It represents the very small inverted image of the flame which is reflected from the posterior surface of the lens (*posterior lenticular reflex*, Fig. 176, *c*). It is distinguished by moving in the contrary sense to the source of light when the position of the latter is shifted; if the candle is depressed, the shining point rises, and vice versa, in opposition to the corneal reflex, which moves in the same sense as the candle flame. It is this posterior lenticular image that is used in doubtful cases to demonstrate the presence of the lens in the eye. If the image is visible, the lens is present; if the image is not seen, either the lens is absent from its place or else is more or less opaque, so that no reflection can be developed on its posterior surface. Fig. 176 also shows a third reflex image, *b*, placed between the other two and originating from the anterior surface of the lens (*anterior lenticular reflex*). The latter is an erect image larger than the others, but so faint that it can be made out only with difficulty. There is also an erect image, not shown in the figure, formed by the posterior surface of the cornea; (see § 389).

FIG. 176.—PURKINJE-SANSON'S REFLEX IMAGES.

The black circle represents the dilated pupil of the eye that is being tested. The candle flame is supposed to be to the right, the observer's eye to the left of the pupil. *a*, image formed by the anterior surface of the cornea, *b*, image formed by the anterior surface of the lens. *c*, image formed by the posterior surface of the lens.

394. Measurement of the Tension.—Finally, the tension of the eye is to be tested. The eye is closed and palpation is made by means of the two index fingers, which are placed upon the upper lid. The tension that is thus appreciated is not, indeed, identical with the intra-ocular pressure, but is proportional to it, and may hence be employed as an expression of it. In so doing we must, however, take into consideration the fact that the sensation which the finger appreciates when it palpates the eyeball is not dependent simply on the tension of the tunics of the eye. What we appreciate is the depth of the impression which the finger makes on the eyeball and the force which we have applied in making it. But both depend not simply on the tension or the intra-ocular pressure, but also on the elasticity of the coats of the eye, and their flexibility or rigidity. Hence on account of the greater rigidity of the sclera, the eyes of old people feel harder than those of younger persons, even when the intra-ocular pressure is the same.

Furthermore, individual variations occur both in the intra-ocular pressure and in the elasticity of the coats of the eye, so that very slight variations from the usual tension can not be denoted as surely pathological, except

when we can make use of the second, normal eye of the same man for the purpose of comparison. Greater alterations in tension, however, make themselves evident at once. It has been agreed to denote the normal tension by the expression Tn (T = tension or tonus). Of increased tension (hypertony) we distinguish three degrees: T+1, T+2, and T+3, which are arbitrarily selected, and indicate approximately: tension noticeably increased; greatly increased; hard as stone. Similarly, we employ for diminished tension (hypotony) the designations T−1, T−2, and T−3.

The estimation of the tension by palpating the eye requires much practice, and even then is an inexact method. It does not suffice to determine in a positive way very small variations from the normal; and as little can it disclose with certainty small variations of tension which occur in the course of a case of disease, since one cannot designate in any precise way for later comparison the sensation which he receives in making palpation. Hence it is advisable to use a *tonometer*. Just as in the examination with the finger, the tonometer determines the tension by the fact that an impression is made on the eyeball; the force with which this is made is given by the weight used for the purpose; and the depth of the impression which is produced and which on account of its shallowness is better characterized as a flattening, is measured. In Schiötz's tonometer a collar (Fig. 177, *b*) bears at its lower end a concave plate (*c*) which is fitted to the curvature of the cornea, and which, as the patient lies upon his back, is placed upon the cornea, the latter being previously made insensitive. (For this purpose since cocaine diminishes the intra-ocular pressure somewhat, we use holocaine, which does not have this effect.) Through the collar and its footplate, which has a hole bored through its middle, passes the rod (*a*) which glides to and fro in them without friction. At its upper end the rod is loaded with a small weight (*d*), of which there are four different grades. When the instrument is placed perpendicularly upon the cornea the rod, owing to its weight, drops down upon the centre of the cornea. If the rod, cannot flatten the cornea its lower end is arrested at the level of the footplate, and a pointer which is connected with the upper end of the rod fails to show any vibration. The less tense the eye is, the more readily can the rod flatten the cornea and drops correspondingly far out of the collar, as is shown by the pointer.

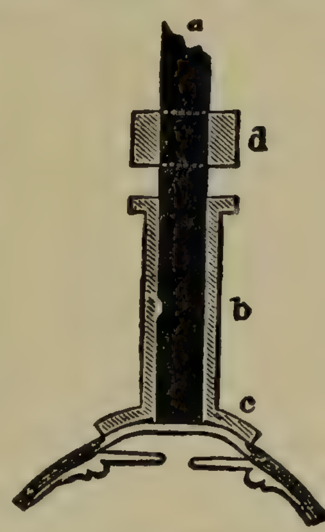


Fig. 177.

In the Schiötz tonometer the excursion of the pointer (which is proportional to the depth of the impression on the cornea) is indicated in mm., the corresponding intraocular tension in mm. of mercury being determined by reference to a graph or table. In the McLean tonometer, in which the weight employed in constant, the instrument is graduated so as to indicate the intra-ocular pressure directly. The McLean instrument is thus more convenient, but the Schiötz tonometer, in which a variable weight is used to produce a minimum impression is in principle more accurate especially for the higher tensions.

The readings of the Schiötz tonometer are best written in the form of a fraction. e. g. 5/6, meaning that with the 5 gram weight the pointer marked 6 mm. on the scale,

Readings in each case should be taken with several weights, but the more reliable are those in which the denominator of the fraction ranges from 2.5 to 6. Tensions of from $5/6$ to $5/3$ (or $7{\cdot}5/8$ to $7{\cdot}5/5{\cdot}5$) may be considered normal. According to the graphs furnished with the Schiötz instrument these fractions represent a pressure of 16 to 25 mm., but, according to the probably more correct indications of the McLean tonometer, 22 to 40 mm. of mercury. Any tension above 28 (Schiötz) or 45 (McLean) should be regarded as pathological; and a tension above 25 (Schiötz) or 42 (McLean) as suspicious. Tensions below 13 (Schiötz) or 20 (McLean) are subnormal.

In applying the tonometer the patient's cornea should be directed straight up, the plunger of the instrument should be vertical, and the centre of its foot-plate should rest on the apex of the cornea (see Fig. 178). The repeated application of the tonometer

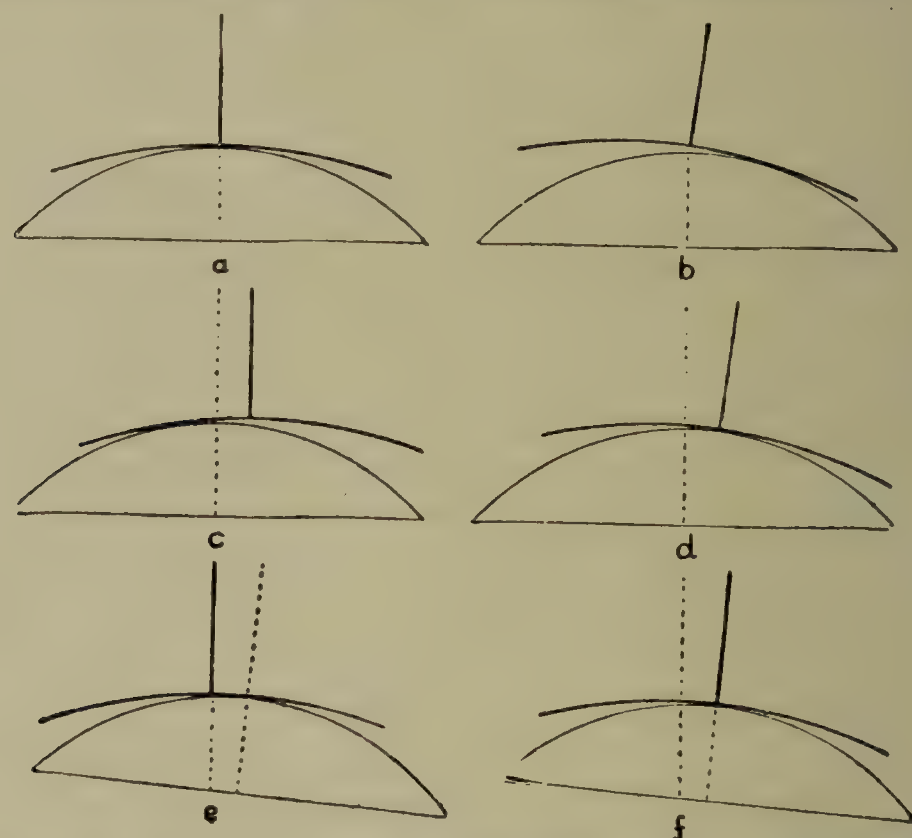


FIG. 178.—APPLICATION OF THE TONOMETER. (After Priestley Smith.)
a, correct position; *b*, *c*, *d*, *e*, *f*, incorrect positions. *c*, causes the most error; *e* and *f* cause the least.

seems to reduce the tension of the eye to below its usual amount. Too many tests, therefore, should not be made close together at one sitting and if the last of several readings is lower than the rest it should be rejected.

As stated above, the tension measured by the finger or tonometer does not precisely represent the intra-ocular pressure, being dependent partly on this, partly on the rigidity of the ocular envelopes. If the cornea is unduly rigid, the tension shown by the tonometer will seem too high, even when the intra-ocular pressure is really normal. If, on the other hand, as in keratoconus, the cornea is unduly yielding, the intra-ocular pressure may be abnormally high, and yet the tension shown by the tonometer may be normal or subnormal. In this case there is said to be a *relative* increase of tension (Strebel and Steiger). And in any case, no matter what the tonometer reading, the intra-ocular pressure must be regarded as relatively too high, if, as in keratoconus and progressive myopia, the ocular envelopes are so yielding as to give way before it.

Since the tonometer plate is curved so as to fit a cornea of approximately normal curvature, it is not strictly applicable when the *curvature of the cornea is abnormal* as in keratoconus and staphyloma. In these conditions, therefore, the readings of the instrument cannot be regarded as absolutely accurate.

395. Ophthalmoscopy.—The invention of the ophthalmoscope by Helmholtz in the year 1851 was one of the most beneficent achievements in modern medicine. It has made the interior of the eye accessible to investigation; blood-vessels and nerves, which in the rest of the body are exposed

only by surgical manipulation, here lie unveiled before us and permit us to study their minutest variations. In ophthalmology, the ophthalmoscope has produced a complete revolution, since it enables us to diagnosticate morbid processes which otherwise would be quite obscure. Furthermore, in general medicine the ophthalmoscope has become an indispensable aid to diagnosis, since many internal disorders produce characteristic changes in the fundus of the eye.

396. Principle of the Ophthalmoscope.—In order to see the fundus of an eye, we must throw light by the aid of suitable apparatus through the pupil and upon the fundus, and receive the light reflected from the latter into our own eye and unite the rays to form a sharp image. In the original ophthalmoscope of Helmholtz this end was obtained in the following way: Before the eye under investigation (*A*, Fig. 179) a glass plate, *P P*, is placed in an oblique position. A source of light, L, placed to one side of the eye, throws upon the glass plate rays, part of which are reflected at the surface of the plate and pass through the pupil into the eye, *A*. The rays reflected from the fundus, *a*, arrive once more at the glass plate and are there in part reflected to the source of light, *L*, while another part goes through the glass plate and enters the observer's eye, *B*, which unites the rays upon its retina into a sharp image, *b*.

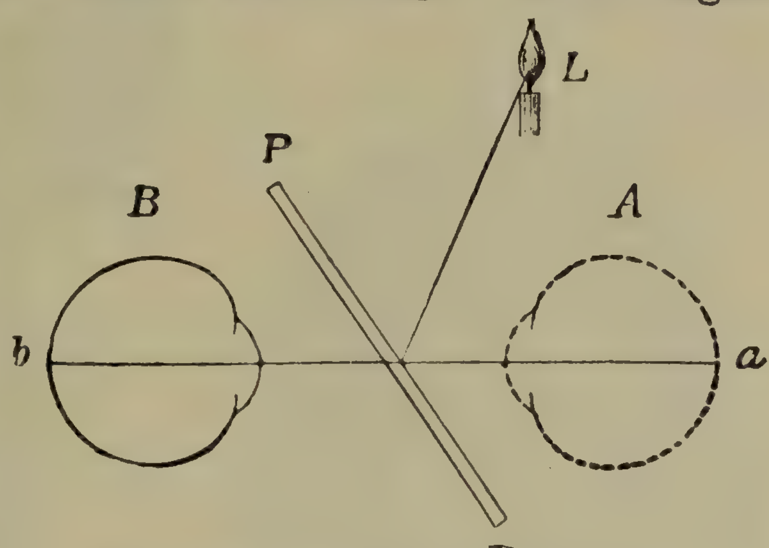


FIG. 179.—PRINCIPLE OF HELMHOLTZ'S OPHTHALMOSCOPE.

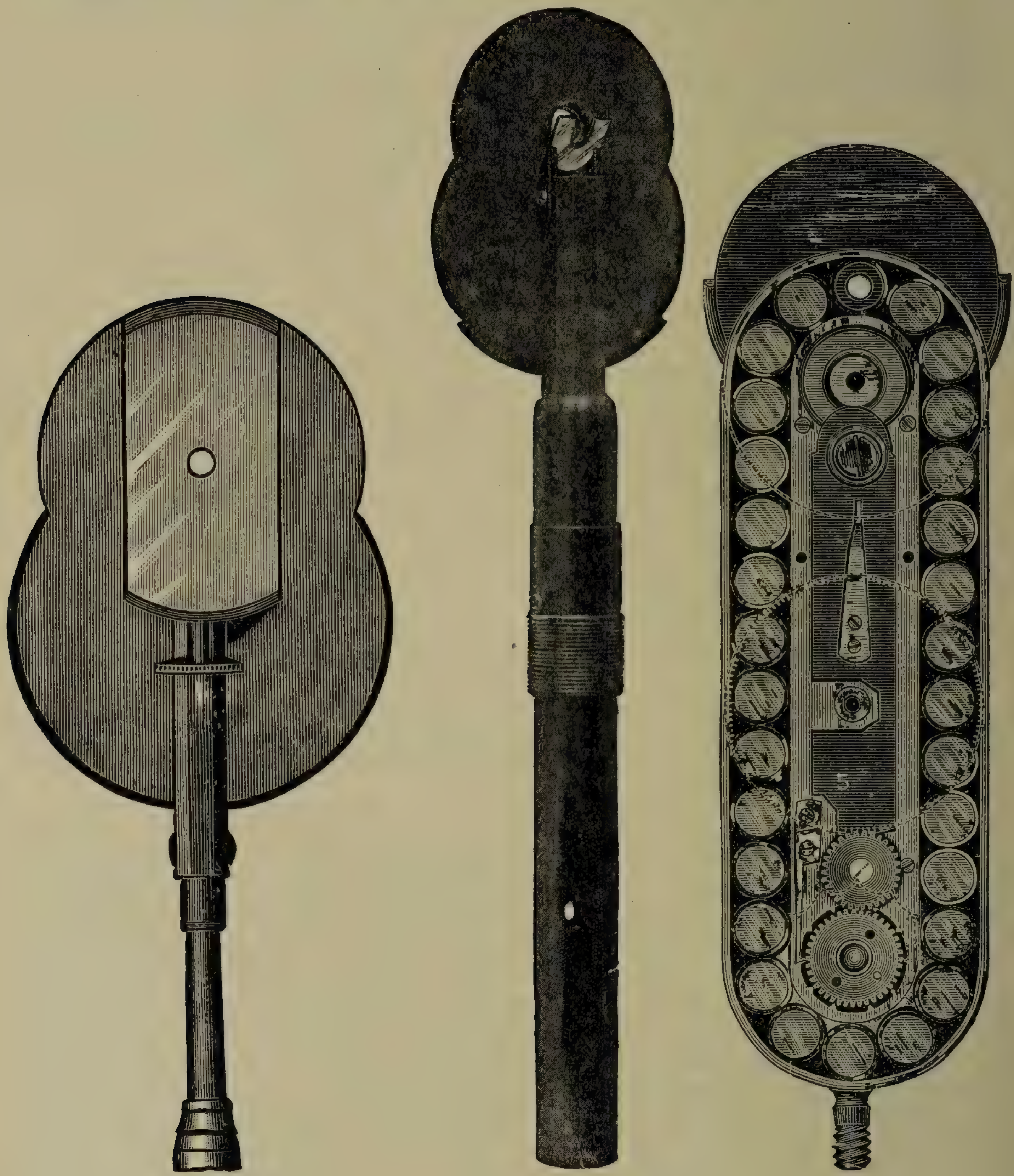
In order to increase reflection and thereby illuminate the background of the eye more intensely, Helmholtz placed three plates one behind the other. A later modification consisted in increasing the reflecting power of the glass plate by lining its posterior surface with a mirror coating, a round hole through the plate or at least through the mirror coating enabling the observer to see through it. The coated mirrors are either plane or concave; the latter, from the fact that they render convergent the rays springing from the source of light, throw a still greater quantity of light into the observed eye. A device is placed behind the mirror, which renders it possible to bring different sorts of lenses before the aperture. In this way it is possible to give the rays of light which fall into the observer's eye any path that may be necessary in order to unite them into a sharp image upon the retina.

In ophthalmoscopy specially good results are obtained by using the Gullstrand *diaphragm lamp* (page 369) as the source of illumination.

Instead of having a separate light as a source of illumination we may attach a minute electric lamp to the ophthalmoscope itself. These *electric ophthalmoscopes* (see Fig. 180, B) give a very clear view of details and frequently may be used where the ordinary ophthalmoscope is difficult to manage, e.g., when the pupil is small, or the patient is restless, or when the examination has to be made with the patient lying flat in bed.

Some ophthalmoscopes are so designed that two or more observers can see the fundus simultaneously; others have been devised for sketching the fundus; and there are binocular ophthalmoscopes which make the details of the fundus stand out in relief.

397. Examination of the Interior of the Eye.—The examination is conducted in a darkened room. The patient sits or stands opposite the physician, and has on the side of the eye to be investigated a lamp as a source of light. Then regularly four methods of examination are applied in succession, viz., oblique illumination, axial (or direct) illumination, the



A. B. C

FIG. 180.—OPHTHALMOSCOPES.

A.—Loring's ophthalmoscope; front view. The perforated mirror in this case is cut into a quadrangular shape, so that it can be rotated slightly from side to side (tilting mirror). This is to make the reflection of light less oblique in using the direct method. The lower disc contains a series of lenses which can be rotated by the pressure of the finger on the serrated edge of the disc, so that any desired lens can be brought in front of the sight-hole. Usually there is added a quadrant on the back, containing additional lenses, which being superimposed over those in the revolving disc make a great variety of combinations.

B.—Marple's modification of Dennett's electric ophthalmoscope.

C.—Morton's ophthalmoscope; rear view, showing the chain of lenses and the mechanism by which each one in succession can be brought in front of the sight-hole in the disc at the top which contains the mirror.

indirect method, and the direct method. In certain cases also we use transillumination.

Oblique illumination shows the condition of the cornea, anterior chamber, iris, and lens (see §§ 389-393).

Axial illumination reveals opacities in the lens and vitreous. If conducted in a special way (skiascopy, see §§ 220-223) it affords a measure of the refraction of the eye.

The direct and indirect methods give a distinct picture of the fundus (optic nerve, and retina). The direct method may also be used to give a distinct picture of other portions of the eye and their relative depth and also determine the refraction of the eye. In making these examinations, if the pupil is narrow, we should dilate it with cocaine, euphthalmine, or ½-per-cent homatropine. Before doing so we must be sure that there is no reason to suspect glaucoma, in which case artificial dilatation of the pupil might have dangerous results, and therefore must not be employed.

To be on the safe side it is well to instil pilocarpine or, in suspicious cases, ¼-per-cent eserine after the examination, in order to contract the pupil again.

398. Axial Illumination; Examination of Transparency of Refracting Media.—In axial illumination light is thrown into the eye by the ophthalmoscope, held at the ordinary reading distance or beyond. If the refracting media are perfectly clear, the pupil shines with a uniform red lustre. If there are small or moderate-sized opacities in the refracting media, these stand out upon the red background of the illuminated pupil as dark points or spots. For the rays which correspond to the opaque spot *t* (Fig. 181) are cut off on their return from the fundus, *a*, so that this spot is not illuminated and hence looks black. This is the case even when the opacities are actually, as seen by light thrown directly upon them, light colored—that is, white or gray. So also even a piece of chalk looks black if it is held in front of a flame. Larger opacities, however, which reflect a good deal of light so as to shine out even upon the vivid red background of the illuminated pupil, appear gray, bluish-white or even pure white.

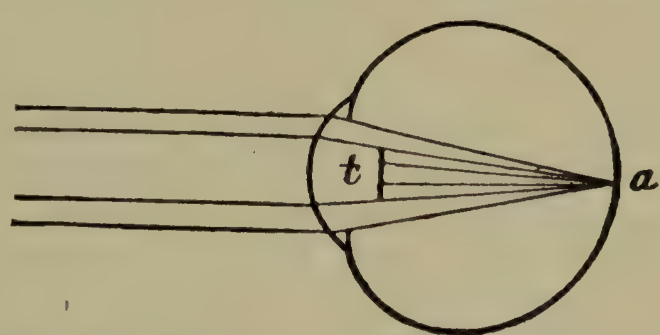


FIG. 181.—VISIBILITY OF OPACITIES IN THE MEDIA BY MEANS OF THE OPHTHALMOSCOPE.

When pronounced opacities are present, we make use of the concave mirror; slight opacities, on the other hand, are discovered only by using the weakly reflective or plane mirror; or by reducing the illumination or by the use of the slit lamp (page 369), which gives especially valuable information. Furthermore in the case of faint opacities it is often specially necessary to dilate the pupil. If the observer is emmetropic, and still more if he is hyperopic, he ought to place a convex glass behind his mirror, so as to be able to get near enough to the eye that he is examining. A myopic observer will not need such a glass.

In examining, we must not neglect to make the eye move in different directions, in order, on the one hand, to obtain a view of laterally placed opacities, and, on the other hand, to stir up in this way opacities which have sunk to the bottom of the vitreous humor. In order to recognize the site of the opacity, we decide, in the first place, whether the latter is movable or fixed. In the former case it can be situated only in the vitreous; in

the latter case—that is, if the opacity moves only with the eye, and not spontaneously—it is probably situated in the cornea or in the lens; but it may still be in the vitreous, since here too fixed opacities are sometimes observed. In many cases this can be decided by employing oblique illumination. If we can obtain no result in this way, we try to determine the site of the opacity, by its *parallactic displacement* with reference to the margin of the pupil. This is accomplished as follows: In the eye, *A* (Fig. 182), suppose four opaque points to be present, which lie at different depths—namely, in the cornea (1), upon the anterior capsule of the lens (2), at the posterior pole of the lens (3), and in the anterior part of the vitreous (4). For simplicity's sake we assume that they are all disposed in the optical axis of the eye. Then, if the observer, *B*, looks into the eye from directly in front, he will see each one of these points precisely in the centre of the pupil, *P*. Suppose, now, that the observer's eye passes from *B* to B_1. The position of the points

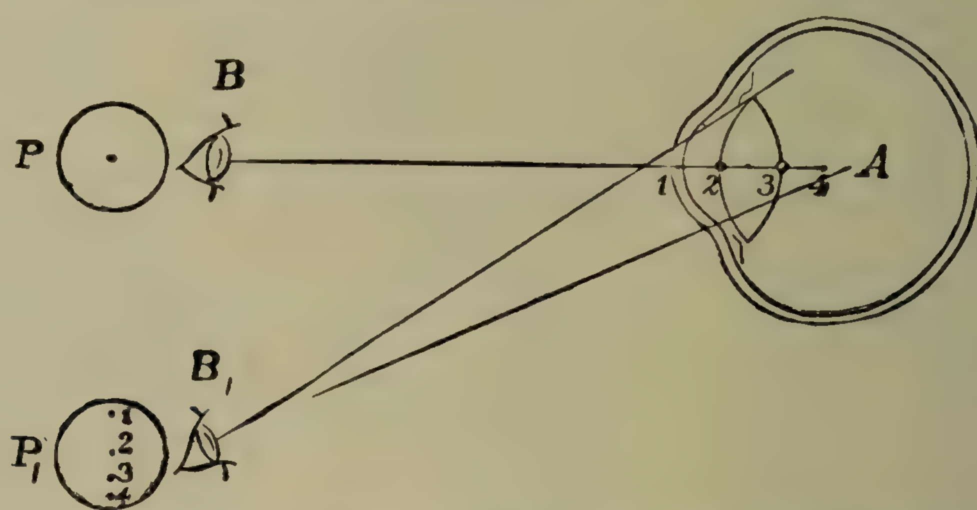


FIG. 182.—DIAGNOSIS OF THE SITE OF AN OPACITY FROM PARALLACTIC DISPLACEMENT.

with relation to the pupil will be changed at once. Point 1 approximates to the upper border of the pupil P_1; point 2, which is situated in the pupil itself, keeps its place unchanged; points 3 and 4 have approached the lower border of the pupil, and 4, on account of its greater depth in the eye, more so than 3. From this example the following rule for the determination of the site of an opacity can be deduced: We look into the eye from directly in front and note the position of the opacity in the pupil. Then, while the patient holds his eye fixed, we move slowly to one side and observe whether the opacity remains in the same spot or not. In the former case, the opacity lies in the pupillary plane (upon or directly beneath the anterior capsule of the lens); in the latter case, in front of or behind this plane—in front of it, if the opacity shifts its place with a movement opposed to that of the investigating eye; behind it, if the opacity moves in the same sense as the eye. The greater the extent of this movement the farther is the opacity from the pupillary plane. (Evidently we can also proceed by keeping our own eye still and telling the patient to move his. But this way is not so good, because if he moves the eye too much, a minute opacity may disappear out of sight and then is often hard to find again.)

The depth of an opacity which occupies the whole pupil can be told by the *shadow cast by the iris* (see § 649).

Another way of determining the site of an opacity is by its relation to the *corneal reflex*. This is the bright reflex (Fig. 176, *a*) which we see about in the centre of his pupil when the patient looks straight at us as we throw the light into his eye. If now he moves the eye in any direction, the reflex leaves the centre of the pupil and approaches the margin of the cornea. At the same time we notice how any opacities that may be situated in the area of the pupil seem to move. An opacity which appears close to the corneal reflex when in the centre of the pupil and keeps close to the reflex in all its movements, must be situated at or near the posterior pole of the lens. Such an opacity, for example is produced by a posterior polar or posterior cortical cataract. On the other hand, an opacity which is any distance in front of the posterior pole of the lens or behind it, will appear to separate from the corneal reflex when the eye is moved, and the more so, the greater the distance is.

Opacities may also be seen and their positions determined by the *direct method* (see § 384 objective).

Dark, ill-defined shadows on the red background of the pupil, which change their position suddenly on moving the mirror, are to be referred to irregularities of the refracting surfaces. (Cf. § 222).

399. Fundus Visible by Axial Illumination.—If when standing 12 or 15 inches from a patient we throw light into his eye with the ophthalmoscope, we sometimes see a clear magnified picture of the fundus. Such an appearance indicates either marked myopia or marked hyperopia. In myopia the image seen is a real inverted image formed by the eye itself at its own far point (see § 172), i. e., in front of the eye. The more marked the refractive error the closer the far point is to the eye. In a myopia of 7 or 8 D or over, the far point and consequently the image will be only a few inches from the patient's eye and

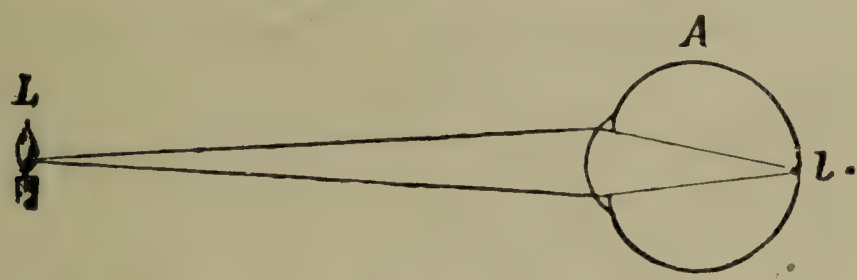


FIG. 183.—PATH OF THE RAYS WHEN THE EYE IS FOCUSED FOR THE SOURCE OF LIGHT.

well in front of the observer. Hence the latter will see it just as he sees the image formed by the indirect method (Fig. 189). In this case, in fact, the extra refractive power of the myopic eye takes the place of the ordinary condensing lens. In hyperopia on the other hand, what we see is an erect virtual image formed by rays apparently emanating from the far point of the eye, i. e., from a point behind the latter. The eye in this case acts just like a magnifying glass through which we are viewing the fundus.

The image in either case is projected on the plane of the pupil, although actually in front of it in myopia and behind it (or, at least formed by rays emanating from behind it) in hyperopia. Hence if the observer moves his head to the right, the image will move in the opposite direction (to the left) in myopia, and to the right in hyperopia. The conditions in fact are the same as when we are on a railway train. The nearby telegraph

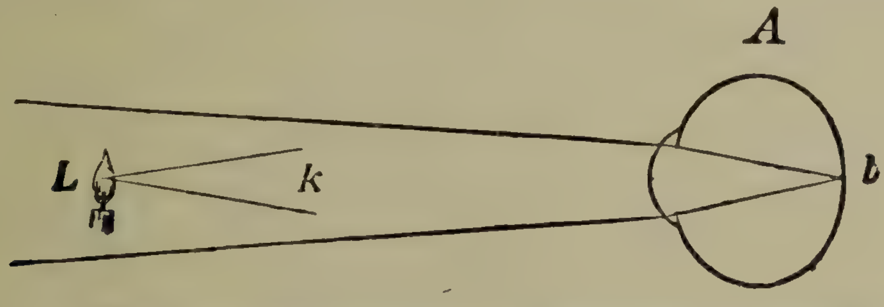


FIG. 184.—EXPLANATION OF LUMINOSITY OF THE EYE.

The source of light, L, throws the conical beam of rays, k, into the eye. The further course of these rays is not shown in the figure, but only that of the rays reflected from the retina at l.

poles being projected on the distant landscape appear to us to fly backward; the distant trees (which we compare with some nearer object) seem to move forward.

400. Luminosity of the Pupil.—Under ordinary circumstances the pupil appears black. The cause of this is as follows: If (Fig. 183) light from a source of light, L, enters the eye, A, and the latter is accurately focused for the source of light, the rays coming from L are united to form a sharp image upon the retina at l. L and l are conjugate foci (§ 154) and can be interchanged; that is, if the rays start from the posterior focus, l, they will come together again at the anterior focus, L. Accordingly, the rays reflected from the illuminated portion of the retina, l, are returned to the source of light, L, and could be seen by an observer only in case his eye was precisely at L. The solution of this problem is another of the discoveries that we owe to the genius of Helmholtz.

The conditions are different when the eye is not focused for the source of light before it. Suppose, for example, that the eye is hyperopic (Fig. 184). Then the rays springing

from the illuminated portion of the retina, *l*, leave the eye as a divergent beam, so that only a part of the rays are returned to the source of light, *L*, while another part passes to the side of the latter and can be seen by an observer stationed near it. Hence comes the striking luminosity of the pupil in so-called amaurotic cat's eye (see § 804), in which the eye is made markedly hyperopic by the pushing forward of the retina. In like manner, luminosity is frequently apparent in an eye which is deprived of its lens and is therefore strongly hyperopic. The luminosity of the eyes of many beasts, especially the carnivora, is also in part to be ascribed to the fact that they are hyperopic, although here the presence of a strongly reflecting layer (the so-called tapetum) in the chorioid contributes to this result.

The luminosity of the pupils of *albinos'* eyes is to be explained in a different way. In such eyes the light passes not only through the pupil, but also through the unpigmented iris, and even through the sclera. Accordingly, in these eyes, not simply a limited district of the retina, but the whole fundus, is flooded with diffused light; and therefore rays from the different portions of the fundus pass out of the pupil in every direction and can very readily be caught up by the observer's eye. That this is the correct explanation is proved by the fact that the pupil of an albino's eye looks black as soon as we hold before the eye an opaque screen having an aperture of the size of the pupil This shuts off from the eye any light which might enter it by other ways than the pupil, and in this respect makes the eye of an albino like that of a normal person.

401. Transillumination.—The presence in the eye of a large-sized solid mass especially a tumor, can be shown by *transillumination* or *diaphanos-*

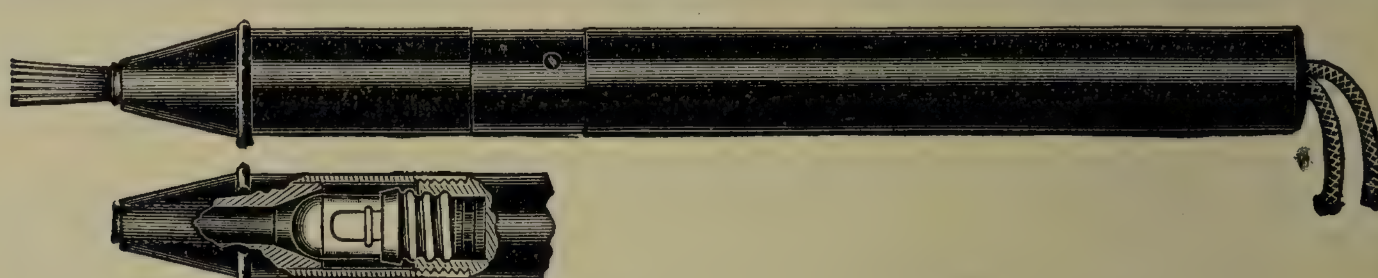


FIG. 185.—WÜRDEMANN'S TRANSILLUMINATOR OR DIAPHANOSCOPE.

copy. This can be used even when the lens is opaque, so that the ophthalmoscope is not available. Special instruments for transillumination of the eye are those of Leber, Sachs, and Würdemann (see Fig. 185), which are applied to the sclera, and the ophthalmo-diaphanoscope of Hertzell. In this last the eye, orbit and nasal sinuses are illuminated by a very powerful light inserted in the mouth. In case of emergency these instruments may be replaced by a small narrow-tipped flash light. With them a red glow is seen in the pupil, which is replaced by a more or less dense shadow, if a tumor or other solid mass happens to lie beneath the point where the instrument is applied.

This test may fail with deeply pigmented eyes (e. g., those of negroes), since these even when otherwise normal may show no red reflex by transillumination (Griscom).

In *pupillary transillumination*, used for determining the presence of tumors in the back part of the eye, a fine brilliant pencil of light is thrown by the diaphragm lamp through the pupil and the observer also looks through the pupil with an ophthalmoscope at the mass, which appears black or red, according as it is opaque or transparent (Gullstrand). In applying this method the light may also be made to emerge through the sclera which appears a brilliant red (Lindahl).

402. Direct and Indirect Methods.—In the examination with the *erect image* (*direct method*), the physician places himself and his mirror directly in front of and very close to the eye that he is observing (see

Fig. 186.—Ophthalmoscopy. The Direct Method with Electric Ophthalmoscope. After Würdemann (from Posey and Spiller).

Fig. 187.—Ophthalmoscopy. The Indirect Method with Reflecting Ophthalmoscope. After Würdemann (from Posey and Spiller).

Fig. 186). For simplicity's sake we will suppose both observer and patient to be emmetropic. If now the observer holds the mirror obliquely in such a manner that he throws the light of the lamp into the pupil of the observed eye, he will immediately get a clear view of the patient's fundus. For (Fig. 188) a certain portion of the fundus of the eye, A, is illuminated by the mirror, S S. The rays reflected from any point, as a, of this illuminated region of the retina, leave the eye parallel with each other (§ 172), pass through the central aperture, o o_1, of the mirror, and fall into the observer's eye, B. Since this eye is emmetropic, these rays, being parallel, are again united at a single point, b, upon its retina so that here there is produced a sharp image of the point a. Since the same process is repeated for all the other points of the illuminated region of the retina of the eye, A, a sharp image of this portion of the retina is formed in the eye of the observer.

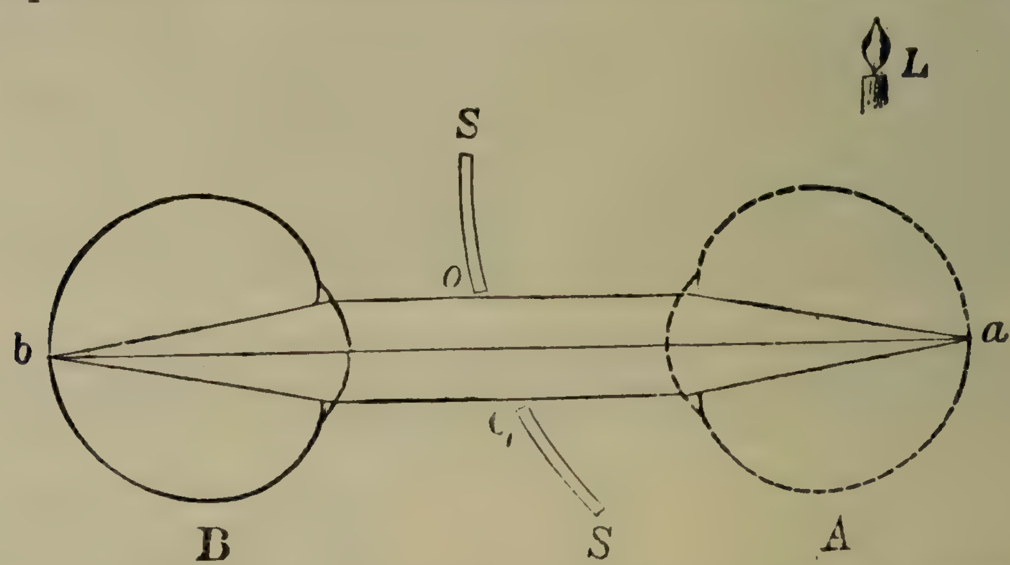


FIG. 188.—OPHTHALMOSCOPIC EXAMINATION BY THE DIRECT METHOD.

The eyes are drawn of the natural size of an emmetropic eye having an axial length of 24 mm The rays that are reflected from the mirror, S S, into the patient's eye are not shown, but only the rays that emerge from the latter.

If not emmetropic, both patient and observer must be rendered so, if the fundus is to be clearly seen by the direct method. This is usually effected by rotating the system of lenses behind the mirror of the ophthalmoscope until the proper correction is secured. The glass so interposed represents the combined refractive errors of patient and observer; or if the observer is emmetropic or rendered so by glasses, represents the patient's refractive error (see § 219). A still better way of securing a clear image by the direct method, particularly when either patient or observer is quite astigmatic or the patient has a high degree (12 to 20 D) of myopia, is for each to wear his correcting glasses during the examination. Then the fundus is seen with great distinctness either with the simple aperture, or, if either patient or observer accommodates, with a weak concave lens.

The direct method may be used not only for examining the fundus at its normal level, but also for examining elevations or depressions in it (§ 411), and also objects (opacities in the vitreous or lens) far in front of it (§ 390). In this case we must interpose behind the mirror a suitable lens, convex or concave, as the case may be, e. g., for opacities in the vitreous a convex glass of 3 to 18 D, and for opacities in the lens and the cornea (and especially deposits on the posterior surface of the cornea) one of 15 to 20 D, the strength of glass depending a good deal on our distance from the patient.

The examination with the *inverted image*, or by means of the *indirect method* (Ruete), is conducted with the aid of a convex lens of about six cm. focus. This lens, l (Fig. 189), is held at about eight cm. from the eye (A) under examination. (See infra Note and Fig. 187). The fundus of this eye is now illuminated by means of the mirror S S. The rays reflected from

the illuminated region, *a*, of the retina pass out in a parallel direction, fall upon the lens and are united at the focus, *f*, of the latter. Thus there is formed at this spot an image of the point *a*. In like manner images from the other points of the illuminated region of the retina are produced in the focal plane of the lens, so that there is formed here an *inverted* image of this portion of the fundus. The observer's eye, *B*, now through the aperture, *o*, of the mirror examines this image at the ordinary reading distance (about thirty cm.), for which purpose the observer, unless he is myopic, must use a certain accommodative effort or else a corresponding convex glass (which he does by revolving the ophthalmoscope disc until a 2 or 3 D convex glass stands before the aperture).

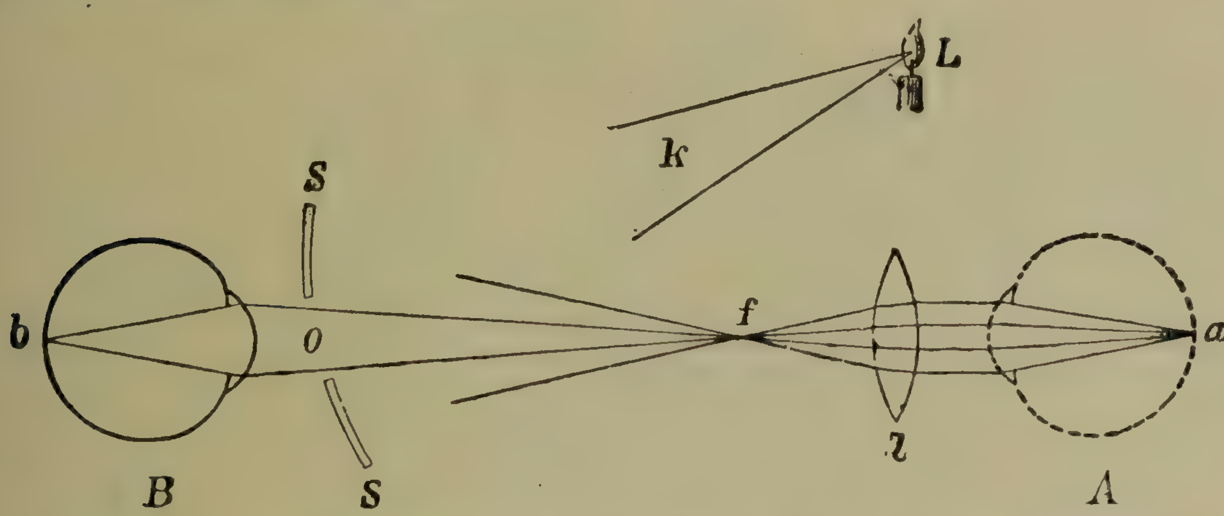


FIG. 189.—OPHTHALMOSCOPIC EXAMINATION BY THE INDIRECT METHOD.
The illumination of the fundus is accomplished by means of the source of light, *L*, from which the cone of rays, *k*, falls upon the mirror, *S S*, and from this is thrown into the eye, *A*. In order not to confuse the representation, these rays are not shown, but only those which pass out of the eye, *A*, again.

Each of these two methods has its advantages. The direct method gives a greater magnification, and is therefore particularly adapted for the recognition of the finer details. The indirect method, on the other hand, affords a larger field of view, and therefore gives a better general prospect. It also gives a more luminous image, and hence, when the refracting media are turbid, will still render the fundus visible when it is no longer to be seen in the direct image. In most cases, both methods are applicable, and then it is advisable to conduct the examination with the aid of both, beginning with the indirect.

The amount of magnification produced with the indirect method is inversely proportional to the strength of the condensing lens that is used. A lens of 12 inches focal length (3.25 D) gives an image about the size of that seen in the direct method, while the 3 inch and 2½ inch lenses, ordinarily employed, give, respectively, an image one fourth and one fifth as large. On the other hand, with a very strong lens we get a little magnified but a very wide general view of the fundus. In all cases the lens ought to be held so that its focus is about an inch in front of the eye; that is, a four inch lens should be held five inches from the eye, etc. If the lens is so held, the distortion of the details produced by astigmatism is reduced to the minimum (see § 405). The corneal reflex, which, the beginner finds, interferes a good deal with his view of the fundus, can be got out of the way by a slight tilting of the lens, if he is using the indirect method, or a slight tilting of the ophthalmoscope itself when he is making a direct examination.

In the "reflexless" ophthalmoscopes of Thorner, Gullstrand, Wolff, and others, the elimination of the reflex is accomplished by making the light enter at one part of the pupil and emerge at the other.

In ophthalmoscopy, especially by the direct method, it is advantageous to keep making slight tilting movements of the mirror, so as to play the light a little this way and that over the fundus, the details and relief of which are thus better shown. Moreover, as Nagel first pointed out, details are often seen best if we so manage the light that the point to be examined lies at the edge instead of the centre of the illuminated area in the fundus ("eccentric illumination" of Burdon Cooper).

403. Examination of the Fundus; Optic Disc.—In making a systematic *examination of the fundus* we begin at the *disc* (or *papilla*). In order to bring the latter into view we make the patient look, not straight in front of him, but a little inward (toward his nose). For the entrance of the optic

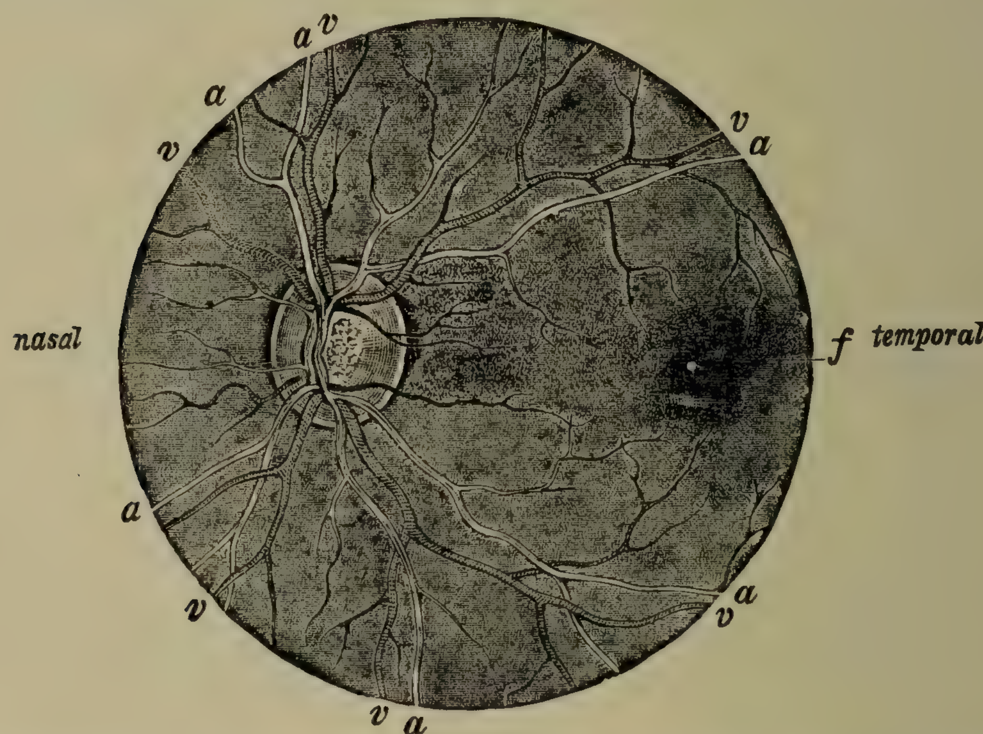


FIG. 190.—NORMAL FUNDUS OF THE LEFT EYE, SEEN IN THE ERECT IMAGE.

The optic disc, which is somewhat oval longitudinally, has the point of entrance of the central vessels somewhat to the inner side of its centre. That portion of the disc lying to the inner side of the point of entrance of the vessels is of darker hue than the outer portion; the latter shows, directly to the outside of the vascular entrance, a spot of lighter color, the physiological excavation, with fine grayish stippling, representing the lacunæ of the lamina cribrosa. The disc is surrounded, first by a light-colored ring, the scleral ring, and externally to this by an irregular black stripe, the chorioidal ring, which is especially well marked on the temporal side. The central artery and vein divide immediately after their entrance into the eye into an ascending and descending branch. These branches, while still on the disc, split into a number of arteries which in the upper half run mainly upwards and outwards (superior temporal artery and vein) and upwards and inwards (superior nasal artery and vein), and in the lower half run downwards and outwards (inferior temporal artery and vein) and downwards and inwards (inferior nasal artery and vein). Moreover, fine twigs (the macular vessels) run from the disc over the temporal border of the latter directly to the macula lutea, while other twigs from the upper and lower temporal vessels push toward the macula lutea, which itself is devoid of vessels and is distinguished by its darker color In its centre a bright punctate reflex, *f*, is visible.

nerve does not lie at the posterior pole of the eye, but on the nasal side of it, and hence is brought directly opposite the observer only after a corresponding rotation of the eye inward. It then comes into view as a bright disc, whose color is a light grayish or yellowish red, contrasting strongly with the red of the rest of the fundus. The shape of the disc is circular or oval; in the latter case generally an erect oval (Fig. 190). Its *size* apparently varies quite a good deal, which, however, is due to the varying degree of enlargement under which the disc is seen. The true size of the disc, measured in enucleated eyes is, as a matter of fact, almost always the same—that is, about 1.5 mm. in diameter[3]. On account of this constancy we use the disc

[3] This corresponds to an arc of 7.5° in the fundus. The blind spot (§ 270) which is the visual correlative of the optic disc naturally subtends the same angle on the perimeter.

for taking measurements in the fundus; we say, for example, that a diseased area is 2 disc-diameters broad.

Circumscribing the disc we can often recognize two rings distinct in color. The inner ring, lying next the border of the disc, is white (in Fig. 190 it runs all the way round the disc, in Fig. 191 it appears only on the temporal side), and is called the scleral (or connective-tissue) ring, because it corresponds to the scleral bundles which jut in between the margin of the chorioid and the head of the optic nerve (Fig. 36). At the margin of the aperture in the chorioid that gives passage to the optic nerve, the chorioid is often marked by a larger accumulation of pigment by which the second, exterior, ring is formed. This is apparent as a black, narrow, sometimes complete, sometimes incomplete, ring, which is called the *chorioidal ring* or pigment ring (Fig. 191; in Fig. 190 it is especially visible at the outer border of the disc). The demarcation of the outline of the disc, produced in this way, is generally much less sharp on the nasal than on the temporal side; for at the nasal side a greater number of nerve fibres pass over the margin of the disc (Fig. 36) and thus obscure it. For the same reason the inner half of the disc looks redder, the outer half paler, because the layer of nerve fibres in the latter situation being thinner, allows the lamina cribrosa to show through more.

FIG. 191.—OPHTHALMOSCOPIC PICTURE OF THE OPTIC NERVE.

Somewhat to the nasal side of the centre of the disc emerges the central vein and, to the nasal side of this again, emerges the central artery. The main trunk of these vessels is itself not visible, since each divides at once into two main branches which run respectively upward and downward. The centre of the disc bordering the temporal side of the central vessels is rather lighter colored and somewhat depressed (the vascular funnel). On the temporal border of the disc can be seen the white scleral ring. The disc is surrounded by the black chorioidal ring.

404. Crescent or Conus.—The gap in the sclera and that in the chorioid, through which the optic nerve passes, together form a short canal, the sclero-chorioidal canal. The shape of this canal is by no means always exactly as Fig. 36 represents it, but it varies pretty considerably even in normal eyes, and to this are due the different pictures which the circumference of the disc is accustomed to exhibit. The canal, as shown in Fig. 193, may keep narrowing all the way in its course toward the interior of the eye. But it may also happen that only one wall of the canal shows an oblique course pointing toward the axis of the optic nerve, while the other wall runs straight forward or is actually turned away from the optic nerve. In the latter case, which is represented in Fig. 192, the head of the optic nerve appears to be displaced bodily to one side within the sclero-chorioidal canal. This state of things occurs most frequently in myopia (see § 745); but, is often also found in emmetropia and even in hyperopia. If now we examine the optic nerve represented in Fig. 192, with the ophthalmoscope, we shall on the temporal side look right into the scleral canal, because the tissue of the optic nerve-head itself is transparent. Then the temporal wall of the scleral canal is visible in perspective fore-shortening from *b* to the edge of the chorioid *c*, i. e., from the point where the canal begins to dilate (about corresponding to the place where the fibres of the lamina cribrosa are given off). With slight degrees of displacement the appearance is that of a rather broad scleral ring, but when the displacement is greater it is like a white crescent adjoining the margin of the

disc. Such a *scleral crescent*, which is also called a *conus* (Jäger), is most frequently met with at the temporal border of the nerve.

A light crescent at the border of the disc may also be produced in another way. In the case represented in Fig. 193, the sclero-chorioidal canal is narrowest anteriorly, so that we could not see into it with the ophthalmoscope. But on the temporal side the pigment epithelium ceases at some distance from the margin of the opening of the optic nerve (at *d*). Over the area *b d* where this occurs, the chorioid, which is also itself somewhat rarefied, is exposed, and hence this spot appears as a light colored crescent adjoining the optic nerve. This *chorioidal crescent* or conus unlike the scleral crescent, described above, is not pure white, but shows remains of chorioidal vessels and pigment.

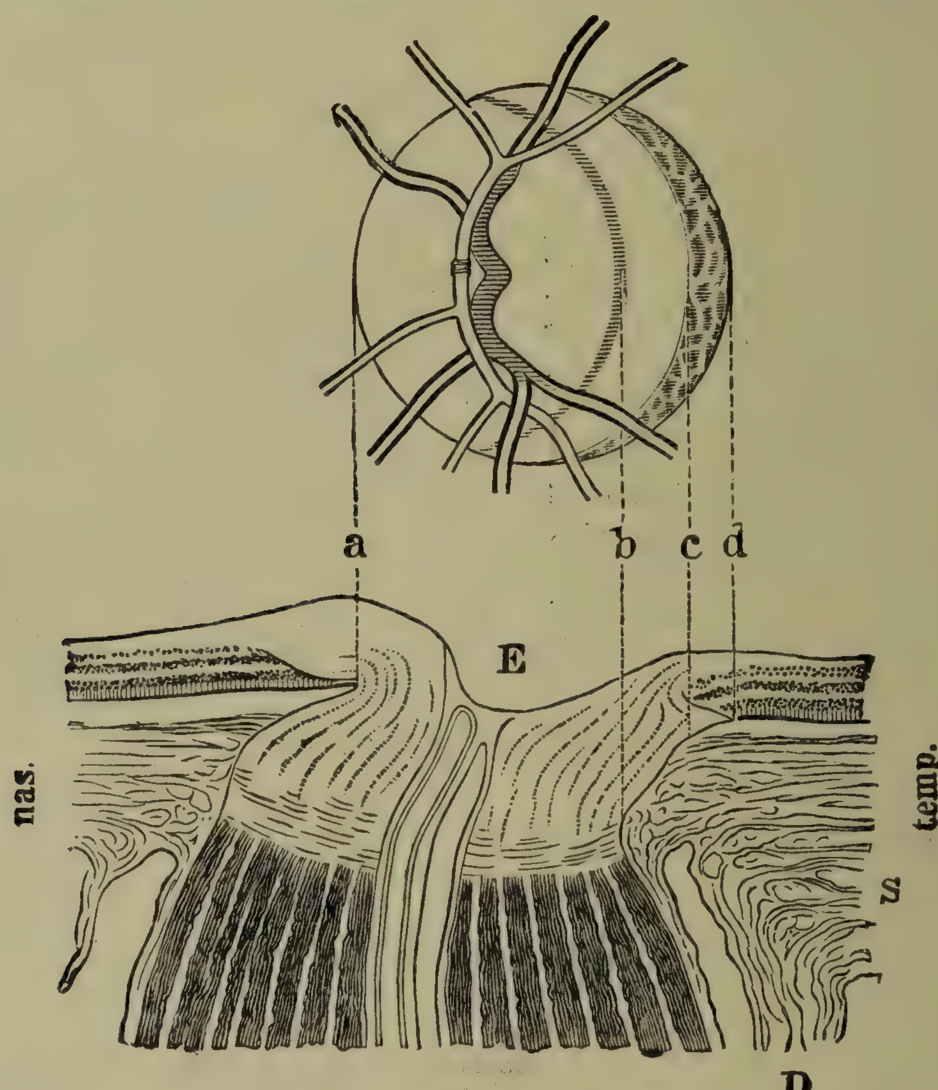


FIG. 192.—LONGITUDINAL SECTION THROUGH THE HEAD OF THE OPTIC NERVE IN A CASE OF SCLERAL CRESCENT AT THE TEMPORAL SIDE TOGETHER WITH THE CORRESPONDING OPHTHALMOSCOPIC PICTURE. Magnified 20x1.

In the section which has been stained by Weigert's method the nerve fibres of the trunk of the optic nerve being medullated have taken on a dark color. They become light colored at the spot where they lose their medulla, that is, at the level of the lamina cribrosa, which crosses the optic nerve in a slightly curved arch having its concavity forward. As far forward as the lamina the trunk of the optic nerve undergoes a uniformly conical narrowing. On the nasal side this narrowing is continued forward and even more markedly since here the wall of the sclero-chorioidal canal juts in more than ever toward the axis of the nerve. But by the same amount that this wall juts in toward the nerve is the temporal wall of the sclero-chorioidal canal separated from the nerve, so that the anterior border of the scleral opening lies at *c*, and the wall of the scleral canal becomes visible with the ophthalmoscope as a white crescent in the space *b c*. The margin of the lamina vitrea and of the pigment epithelium lies still further toward the temporal side, at *d*, and at this point the fibres of the optic nerve are drawn over to the temporal side to form a pointed process. Between *c* and *d* the outer layers only of the chorioid are present, and these appear with the ophthalmoscope under the form of a spotted (chorioidal) crescent On the temporal side the outer layers of the retina come to a stop sooner than do the inner ones, while on the nasal side of the nerve-head the reverse obtains. The nerve-head shows a shallow depression (physiological excavation, *E*). S, sclera; its inner layers extend up to the optic nerve, its outer ones at some distance away from the latter are reflected backwards into the dural sheath, *D*.

These two kinds of crescents are frequently found combined, as in the case shown in Fig. 192 (cf. also Fig. 331). For here the posterior layers of the chorioid extend pretty close to the optic nerve, in fact as far as *c*, while the lamina vitrea together with the pigment epithelium has receded to a greater distance, i. e. to *d*, at which spot also the fibres of the optic nerve are drawn out into a point through a gap in the border of the

lamina vitrea. Consequently, the posterior lamellæ of the chorioid are exposed from *c*, the anterior border of the scleral canal, to *d*, the anterior border of the chorioidal canal. The resulting appearance in the ophthalmoscopic picture is as follows: Close to the disc is a pure white crescent (*b c*), and adjoining this is a narrower crescent (*c d*) which is lighter colored than the rest of the fundus, and presents some chorioidal vessels as well as a little pigment.

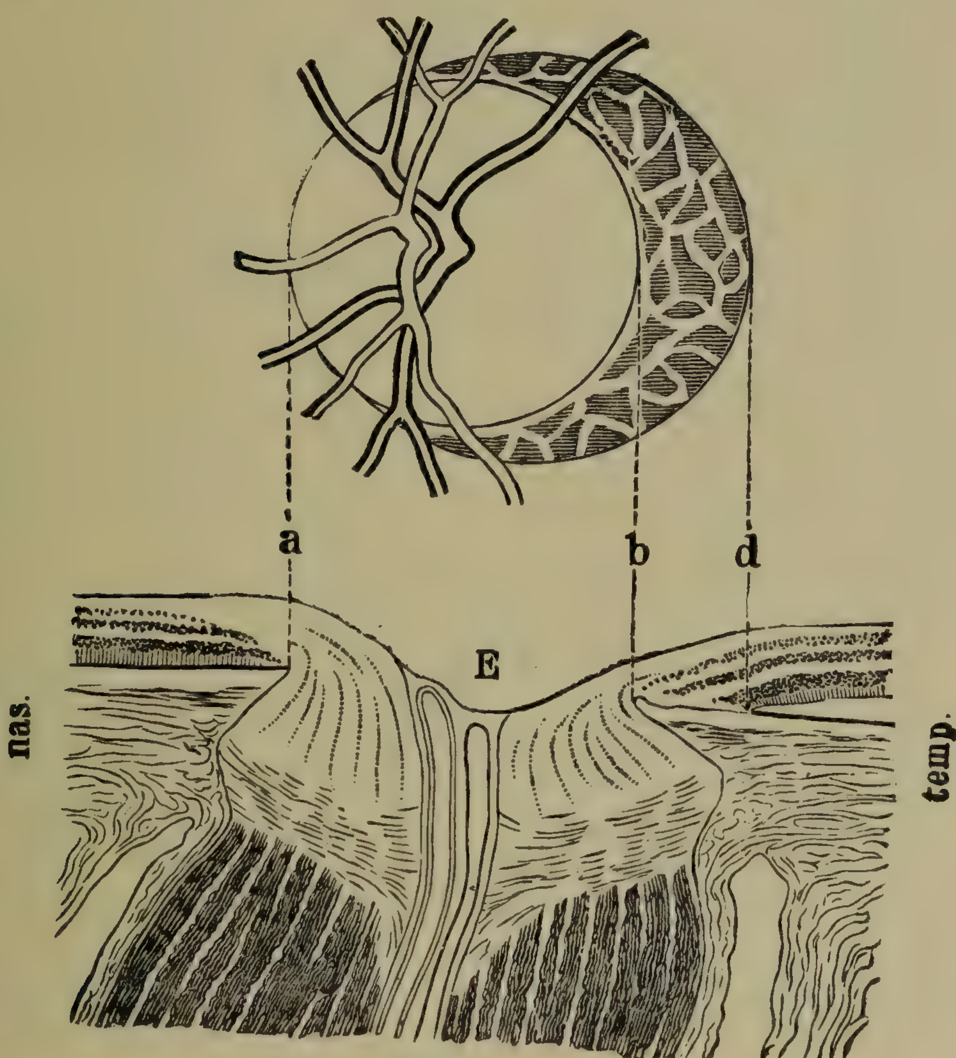


FIG. 193.—LONGITUDINAL SECTION THROUGH THE HEAD OF THE OPTIC NERVE IN A CASE OF CHORIOIDAL CRESCENT ON THE TEMPORAL SIDE TOGETHER WITH THE CORRESPONDING OPHTHALMOSCOPIC PICTURE. Magnified 20x1.

Here the medullary coating of the nerve fibres disappears further back in the central than it does in the peripheral bundles. In its course from behind forward the scleral canal shows first a dilatation then again a contraction which becomes still more marked in the chorioidal canal. At the temporal side the pigment epithelium comes to an end at *d*, some distance from the edge of the chorioidal opening. At *d* the layer of rods and cones of the retina also ends. The space from *d* to *b*, the border of the sclero-chorioidal canal, appears with the ophthalmoscope under the form of a crescent, in which is recognizable the structure of the chorioidal stroma. There is a shallow physiological excavation, *E*.

White crescents are also observed at the *lower* border of the papilla (Fig. 195). These resemble in their appearance the acquired crescents due to myopia, but have an altogether different significance. They are congenital and are associated frequently with astigmatism and almost always with incomplete acuity of vision which, however, in many cases can be raised nearly or quite to the normal by proper glasses. Congenital and acquired crescents may occur in the same eye.

405. Change of Size and Shape of Disc in Ametropia.—If when we are examining a patient by the indirect method we first hold the condensing lens close to the eye and then gradually withdraw it, the disc will appear steadily to enlarge if the eye is myopic and grow smaller if the eye is hyperopic. If the eye is emmetropic the disc will not change in size as the lens is withdrawn. In an astigmatic eye a disc really circular will look oval when the condensing lens is held close to the eye. If, as usually is the case, the vertical meridian of the eye is the most refractive, the disc appears a horizontal oval. Then as the lens is withdrawn, the disc grows less oval, then circular, and finally turns into a vertical oval, which becomes more and more elongated as the lens recedes.

If the condensing lens is placed with its focus at a certain distance (usually just

about an inch) in front of the patient's cornea, there will be no distortion from astigmatism, and the disc will appear of the same size whether the eye is emmetropic, hyperopic, or myopic. This is the position of the lens, therefore, which should be habitually used in the indirect method.

If an astigmatic eye is examined by the direct method, the disc appears oval, being elongated in a direction corresponding to the most refracting meridian of the eye. All other objects in the fundus are similarly drawn out, so that in high astigmatism points become converted into streaks and lines into bands (see § 170). Vessels which run parallel to the line of elongation look distinct, those at right angles, i.e., those which are parallel to the emmetropic meridian, look most obscured. This astigmatic blurring is cleared up if the patient puts on his correcting glasses when the ophthalmoscopic examination is made.

It must not be inferred that an oval disc necessarily means astigmatism. Many discs are naturally oval. In this case the disc will appear oval both by the direct and indirect methods, and with the latter will not change its shape as the condensing lens is withdrawn.

406. Pseudoneuritis.—Not infrequently there are discs which are dark grayish red, and which, especially on the nasal side, have a very indistinct margin. Sometimes there is a cloudy gray areola about the disc or a gray radial striation emanating from it which obscures the borders of the disc; there may even be a slight protrusion of the disc. Such discs are sometimes found in perfectly normal eyes, but more frequently in eyes affected with marked hyperopia, astigmatism, or congenital amblyopia. They are often found in conjunction with irregularity in the shape of the disc, an abnormal division of the vessels, or a crescent at the lower border of the disc, from which it follows that they are to be regarded as congenital abnormalities. Since such cases are usually regarded by beginners as instances of neuritis, they have received the name of *pseudoneuritis*. Still more likely to cause confusion with neuritis are the rare cases in which either from congenital or acquired anomalies there is excessive tortuosity of the retinal arteries and veins (see § 772). Another mistake often made is to diagnosticate neuritis when the disc, owing to the presence of astigmatism, appears hazy by the direct method (see § 170). This mistake will not be made, if the indirect method is also employed, for then the disc will appear distinct, which it would not do if it was a case of actual neuritis.

407. Physiological Excavation.—The optic disc under normal conditions lies in the plane of the retina, and does not, therefore, form a projection as the name papilla would lead one to suppose. On the contrary, it very frequently contains in its centre a depression, which is produced by the fibres of the optic nerve separating from each other comparatively early and thus leaving a funnel-shaped space between them (*vascular funnel*, Figs. 36 and 191). The central vessels ascend on the inner wall of the funnel. The color of the vascular funnel seems white to us because we see the white lamina cribrosa at its bottom. Often, instead of a small funnel-shaped depression, a large excavation (*physiological excavation*, Fig. 192, *E*) is present. This is situated in the outer half of the disc, to whose external border it often reaches. The blood-vessels come out upon the inner border of the excavation (Fig. 190), and at the bottom of the latter are seen grayish dots, the lacunæ of the lamina cribrosa. With the brilliant white of the excavated exterior half of the disc the grayish-red hue of the unexcavated interior half is in vivid contrast.

Sometimes the physiological excavation is so large that it takes in the larger part of the disc, but, in contrast with the pathological excavation, it never takes in the whole. There is always a part (though it may be a small part) of the disc that is unexcavated, this being, as a rule, on the nasal side, while on the temporal side the excavation does often

extend to the border of the disc, though it never has a sheer wall there like the pressure excavation (cf. § 827). It must be remarked, however, that the distinction between the physiological and the pressure excavation is not always easy to make. In some instances at least, in the translator's belief, the latter develops out of the former.

408. Vessels of Fundus.—The central vessels of the optic nerve divide at the head of the nerve into a number of larger and smaller branches, which pass over its edge into the retina, where they keep on branching in an arborescent fashion. They can readily be distinguished into arteries and veins. The former are of a brighter red, narrower, and run a straighter course (Fig. 190, *a a*); the latter are darker, of greater calibre, and more crooked (*v v*). The arrangement of the vessels in the retina is not always the same; most frequently it happens that two main branches run upward and two downward, while only small and short twigs pass to the outer and inner sides of the disc (Fig. 190). The region of the macula lutea is devoid of larger vessels; the larger trunks running to the outside above and below, encircle it and send finer branches in toward it. (See Fig. 34.)

In the larger vessels we notice a shining white streak running along the centre of the vessels. This streak, which is more distinctly visible in the arteries than in the veins, is called the *reflex streak* (Jäger).

A *pulsation* is frequently observed in the vessels at the spot where they first come to view upon the disc. The way in which this displays itself is that that portion of the vessel which lies closest to the point where the vessels come out alternately becomes empty, so as to disappear from view entirely, and then full again. This is not, therefore, an instance of the ordinary pulse, in which there are simply variations in calibre, but a completely intermittent inflow of blood (in the case of the arteries) or an intermittent outflow (in the case of veins).

A venous pulse is a physiological occurrence: in the same eye it is sometimes present, sometimes absent. In the latter case, slight pressure upon the eye with the finger suffices to produce it.

Donders gives the following explanation of the venous pulse: At each systole of the heart an additional quantity of blood is driven into the arteries of the interior of the eye, and the blood pressure in these arteries is consequently heightened. This increase in the arterial tension reacts at once upon the general intra-ocular tension, heightening the latter, so that it acts more vigorously upon the retinal veins and compresses them. It does this most at the spot where the blood pressure in the veins is lowest, which is at their point of emergence upon the disc, since the blood pressure in the veins diminishes in proportion as we approach the heart and get farther away from the capillaries. The veins consequently are constricted at the spot where they dip down into the vascular funnel, while the portion of the veins directly adjacent swells up, because the blood is dammed up in it. But as a result of this damming, the blood pressure in the veins rises rapidly to the point where it is able to overcome the compression—doing this the more readily as now the diastole of the heart sets in, and with this the intra-ocular pressure diminishes. As above stated, moderate digital pressure on the eyeball causes pulsation of the retinal veins. With greater degrees of pressure the retinal arteries narrow and the veins are emptied. This effect is most marked in children; it is less in elderly subjects; and in old persons with arteriosclerosis digital pressure may not produce even a venous pulsation (Black).

An *arterial* pulse is present under pathological conditions only. In

order to produce it artificially in a healthy eye, no inconsiderable pressure must be exerted upon the eyeball. When this is done, the person under examination notices a simultaneous obscuration of the field of vision, amounting finally to complete abrogation of sight, as a result of the obstruction to the retinal circulation produced by the pressure. In like manner a pathological rise in pressure (glaucoma) sometimes causes an arterial pulsation.

The explanation of this is as follows: As a result of the increased pressure in the interior of the eye, the blood is able to enter the vessels of the retina only during the systole of the heart; during the diastole, when the pressure in the arteries falls somewhat, the pressure in the central artery of the optic nerve is no higher than the pressure in the interior of the eye, and hence the blood-wave cannot make its way into the latter. Such a disproportion between the intra-ocular pressure and the pressure of the blood in the central artery may also, of course, be produced by the fact that while the former remains normal, the latter is diminished. Accordingly, an arterial pulsation is observed in general anæmia and when syncope is imminent; also in local compression of the central artery within the optic nerve (e. g. in optic neuritis).

A *true arterial pulse* (i. e., one not due to a completely intermittent inflow, but to a translation of the variations of the pulse into the retinal vessels) occurs as one of the symptoms of an abnormally extensive diffusion of the pulse-wave all through the body, e. g., in insufficiency of the aortic valves or in Basedow's disease.

409. Appearance of Fundus; Macula.—Since in healthy living eyes the *retina* is transparent, we see no part of it with the ophthalmoscope except the blood vessels. At most we find the red fundus in the immediate neighborhood of the disc covered by a delicate gray veil which shows a radiating fine striation and which is the representative of the layer of nerve fibres of the retina, which in this situation is still quite pronounced. In children vivid reflexes often exist which are especially pronounced along the vessels, change their place with every movement of the mirror, and give the retina a lustre like that of watered silk. We must not regard these as pathological opacities. In old people, on the other hand, we find the retina less transparent and lustrous, the vessels narrower, and the disc paler (Leber).

The region of the retina that is most important for vision, the *macula lutea*, with the fovea centralis, is just the part that has very few distinctive ophthalmoscopic features. We find it with the ophthalmoscope if we go a distance of 1½ to 2 disc-diameters outward from the outer border of the disc. Here we come upon a region devoid of vessels which is somewhat darker than the rest of the fundus. Directly in its centre, corresponding to the situation of the fovea centralis, we see, when examining the eye with the direct method, a bright point or a small, crescentic spot (Fig. 190, *f*). In the inverted image the macula lutea is represented by a fine white, not very sharply-defined curved line which forms a horizontal oval of about the size of the disc. The region inclosed by the line is colored a dark brownish-red and sometimes has in its centre a little bright dot. These appearances are by no means constantly present. When the pupil is dilated they become less marked or disappear altogether.

If the fundus is examined by a light from which the *red rays have been excluded* by a suitable filter, the reflexes above described become very marked, the vessels appear

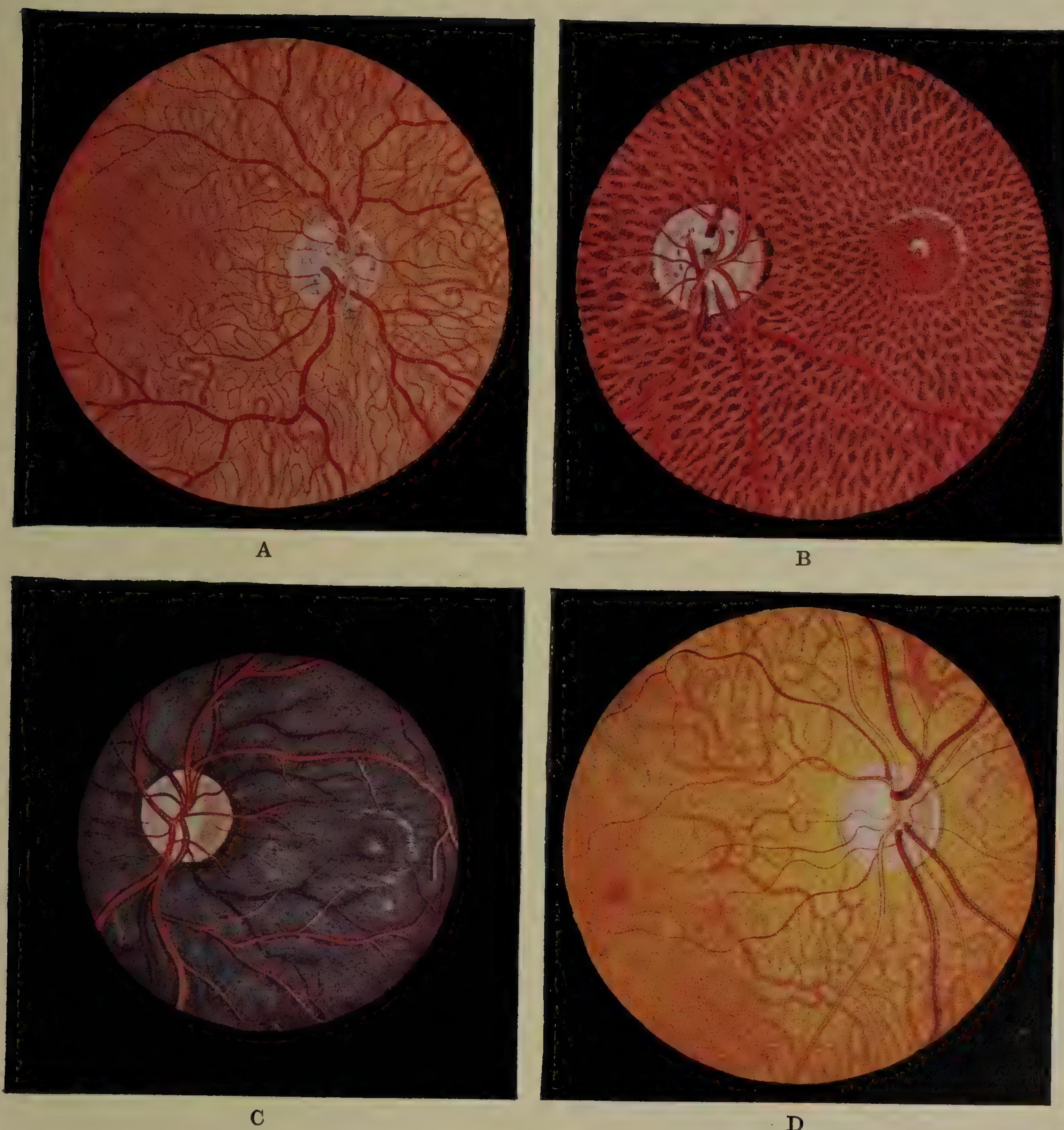


Fig. 194.—Varieties of the Normal Fundus. (After Würdemann in Posey and Spiller.)

A, albinotic fundus; albino and light blonde (after Greef, modified by Würdemann). B, the tessellated fundus; brunette (after Greef, modified by Würdemann). C, the negroid fundus; negro (Würdemann). D, the yellow fundus; Chinese (after Oeller, modified by Würdemann).

black on a greenish background, and the nerve fibres are clearly visible all over the fundus. The fibres going to the macular region run in a nearly straight line, those going more peripherally form elliptical arches surrounding the macular region and meeting and interlacing beyond it (cf. Fig. 40). The macular region, which is apparently one third the size of the disc, is a shining yellow and contains no pigment. (The dark color of the macula observable with ordinary light may be attributed to the intensification of the normal red color of the fundus by the yellow coloration, demonstrated by this method to be really present in the macula (Vogt, Von der Heydt).

410. Color of Fundus.—Since the purple color of the living retina is not visible with the ophthalmoscope, the *color* of the background on which the appearances above described are to be seen is produced by the structures lying behind the retina, namely, the pigment epithelium, the chorioid, and the sclera. The following varieties of coloration in the background of the eye are found, depending on the amount of pigment contained in the two structures first named:—

1. If the pigment epithelium is very uniformly and profusely pigmented, it conceals the chorioid completely, and the background of the eye appears of a uniform brownish-red (in very darkly pigmented men almost a dark gray—see Fig. 194 C). When the pigmentation is less uniform we observe in the erect image a finely granular appearance of the eye-ground which is caused by the cells of the pigment epithelium.

2. When the pigmentation of the pigment epithelium is less marked the chorioid shows through it and if this latter is profusely pigmented we can recognize the interspaces between the chorioidal vessels (the so-called intervascular spaces, Fig. 27, *H*) as dark elongated islands. The light red striæ running between the latter and anastomosing everywhere with each other correspond to the chorioidal vessels, which are chiefly veins. No sharp outlines of vessels, however, are visible, because the pigment epithelium spreads a veil over the chorioid. Such an eye-ground is said to be *tessellated* (Figs. 194 B, and 195); by beginners it is often confounded with chorioiditis.

3. The less pigment there is contained in the pigment epithelium and in the chorioid the more the white sclera shows through, and consequently the lighter red is the eye-ground as a whole. In individuals with little pigment —i. e., in blondes and to the greatest degree in albinos—the pigmentation in the fundus is so scanty that we can see the vascular network of the chorioid distinctly. In contrast with the tessellated fundus, the intervascular spaces are lighter than the vessels, because the white sclera shows through. Such a fundus is called *albinotic* (Fig. 194 A, and Fig. 196; see also § 770). The retinal vessels run over the chorioidal vessels, but are easy to distinguish from them. The chorioidal vessels are broader, less sharply defined, and look flat and ribbon-like; they lack the reflex streak. In opposition to the retinal vessels, which branch after the manner of a tree and do not anastomose, they form by their numerous anastomoses a dense network with elongated meshes.

411. Determination of Differences of Level in the Fundus.—As already indicated (§ 402), the ophthalmoscope may be used by the direct method to estimate the refraction of the fundus. It may also be used to estimate the refraction of elevations or depressions in the latter. If a point in the fundus

projects above its surroundings, as, for instance, the swollen disc in neuritis, the axis of the eye corresponding to this point is shorter—that is, there is a hypermetropia. By determining the degree of the hypermetropia we can compute the height of the prominence. Conversely, a point of the fundus which lies farther back (for example, the bottom of an excavation) is

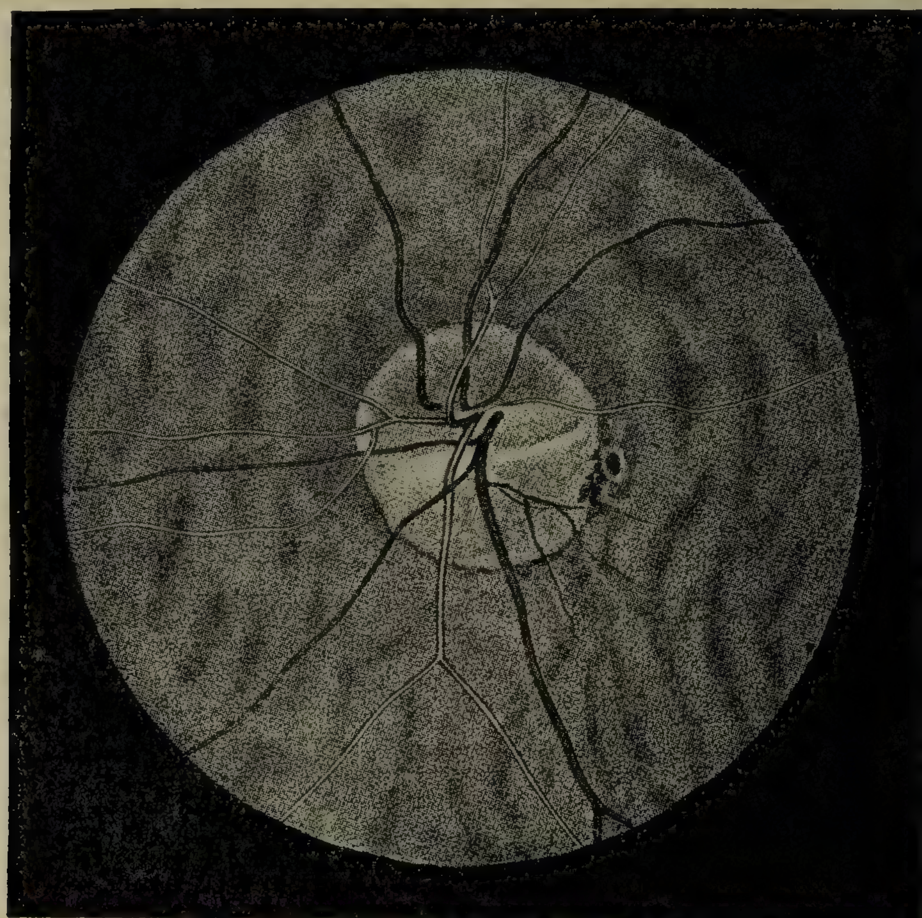
Nasal  *Temporal*

FIG. 195.—INFERIOR CRESCENT AND TESSELLATED FUNDUS.

The bright disc which at first glance might be taken for an enlarged papilla consists of two divisions. The upper, which is darker and of reddish hue, is the papilla proper, which has the form of an irregular oval. Its upper border is semicircular, its lower almost rectilinear, while its two ends are somewhat pointed. The orifice of exit of the vessels lies close to the lower border of the oval, and the vessels as they emerge are at first all directed downward. Hence those that are going to supply the upper half of the retina have to make a sharp bend in order to take the proper direction. Thus the whole arrangement of the vessels on the papilla has a peculiar appearance differing from the normal. The lower division of the light-colored area is formed by the crescent, which is separated by a still brighter rim from the lower border of the papilla. The crescent is unusually large in this case, and, in contradistinction to the reddish papilla, is partly gray, partly white. It is lined by a delicate rim of pigment, and a small patch of pigment also lies close to its temporal border. The fundus shows the pigmentation of a tessellated background.

myopic and from the amount of myopia the depth of the depression can be found. As the basis of this computation the rule holds that a difference of level of 1 mm. corresponds to a difference of refraction of 3 D(page 164).

Quite in the same way the *depth of opacities* in the vitreous may be measured by determining the difference in dioptrics between the glass with which they are clearly seen and the glass with which the fundus is visible. Thus if an opacity is seen with a + 8 D and the fundus with a − 4 D, the opacity is 12 ÷ 3 = 4 mm. in front of the retina.

Differences of level of the fundus are also made apparent by *parallactic displacement.* This is the displacement that under ordinary conditions of vision all objects show which do not lie in a plane parallel to the frontal plane. With the erect image we appreciate this parallactic displacement if during the examination we move a little from side to side. In making the examination with the inverted image we move the convex lens which serves for the production of the inverted image a little up and down during the examination. If the points of the fundus which we have fixed upon lie all in the same plane, they do not change their relative position to each other with the shifting of the convex lens. If, on the contrary, a difference of level exists between them, we notice a displacement with

relation to each other, so that they now come nearer together, now go farther apart. Figure 197 may elucidate what takes place. Let a be a point on the edge, b a point behind it on the bottom of an excavation of the optic nerve. If the convex lens stands at I, the images of the two points a_1 and b_1 fall behind each other and are superimposed. If the con-

FIG. 196.—FUNDUS OF AN ALBINOTIC LEFT EYE SEEN IN THE ERECT IMAGE. (After Jäger.)

The disc is surrounded by a light-colored scleral ring, and looks dark in comparison with the light hue of the rest of the fundus. The latter shows a thick network of chorioidal vessels and over them the retinal vessels, which are distinguished from the former by their better defined outline, their narrow calibre, and their straight course. Both the chorioidal and retinal vessels contrast by their darker red with the very light red of the background, the hue of which is produced by the white sclera shining through the chorio-capillaris. It is only at the macula lutea that the somewhat darker hue of the fundus gives evidence of the presence of a greater amount of chorioidal pigment.

vex lens is now brought to II, the image of the point a is reproduced at a_2, that of b at b_2; the points appear to have separated from each other. Had the convex lens been carried in the opposite direction, the apparent displacement of the two points would have

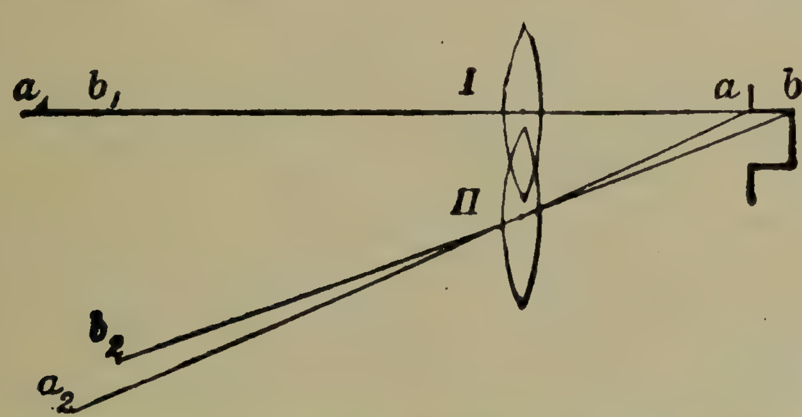


FIG. 197.—PARALLACTIC DISPLACEMENT OF THE INVERTED IMAGE OF POINTS OF THE FUNDUS, LYING AT DIFFERENT LEVELS.

taken place in the opposite sense; it would have looked as if the edge of the excavation had been drawn over the bottom of it. From the magnitude of the displacement the difference of level of the two points can be estimated, but cannot be exactly computed, as is possible with the aid of the erect image.

PART V

DISEASES OF THE EYE

DISEASES OF THE EYE

CHAPTER XIX

DISEASES OF THE LIDS

I. Inflammation of the Skin of the Lids

412. In the skin of the lids we find almost all those diseases which appertain to the skin in general. With regard to them, therefore, reference must be made to the text-books on skin diseases. In this place only such affections of the skin of the lids will be considered as are of comparatively frequent occurrence in the lids, or which, in consequence of the peculiar anatomical structure of the latter, present some special features in their course and their result.

1. *Exanthemata*

413. Erysipelas.—Among the *acute* exanthemata erysipelas requires special mention. If this attacks the skin of the face, the lids participate very markedly in the inflammation, so that they are very greatly swollen, and the patient for several days together can not open his eyes. When the swelling and infiltration are specially marked, the skin of the lids becomes gradually discolored and blackish, and at length to a large extent gangrenous (erysipelas gangrænosum). Not infrequently the erysipelatous process penetrates under the guise of a phlegmonous inflammation into the deeper parts, so that abscesses are produced in the lids or even in the orbit itself. In the latter case, implication of the optic nerve may occur, and, by transmission of the suppuration to the cranial cavity, meningitis may take place and lead to a fatal issue.

414. Herpes Febrilis.—Herpes febrilis forms on the lids small limpid vesicles which are usually situated in groups placed on a common, slightly reddened base. After a few days they dry up without leaving any scar. Herpes febrilis is usually unilateral and develops in consequence of febrile infectious diseases, most often those of the respiratory tract. It is oftener associated with an eruption of vesicles on the cornea (§ 565). Sprinkling the vesicles with a desiccant dusting powder suffices for the treatment.

415. Herpes Zoster.—Herpes zoster is an affection of the skin which consists in the formation of vesicles along the terminal expansion of a nerve. Of the cranial nerves the trigeminus is the one in whose area of distribution this affection occurs. The efflorescences are then found in the vicinity of the eye, for which reason herpes of the trigeminus is known as herpes zoster ophthalmicus or zona ophthalmica.

Violent neuralgic pains in the course of the trigeminus usually precede for some days the outbreak of herpes. Then the exanthem makes its appearance, with accompanying febrile symptoms—vesicles, which for the most part are arranged in groups, starting up upon the reddened skin. The

vesicles most frequently occupy the region of distribution of the first branch of the nerve, so that they are found upon the upper lid, upon the forehead as far as the scalp, and also upon the nose (Fig. 198). When the district supplied by the second branch of the trigeminus is affected, the vesicles are situated upon the lower lid, over the superior maxillary region as far down as the upper lip, and over the region of the malar bone. Sometimes the terminal expansions of both branches are affected simultaneously, while it is extremely rare for the region of the third branch to be involved. It is a characteristic feature of the exanthem, which is almost always confined to one side, that the affection of the skin is sharply delimited at the middle line.

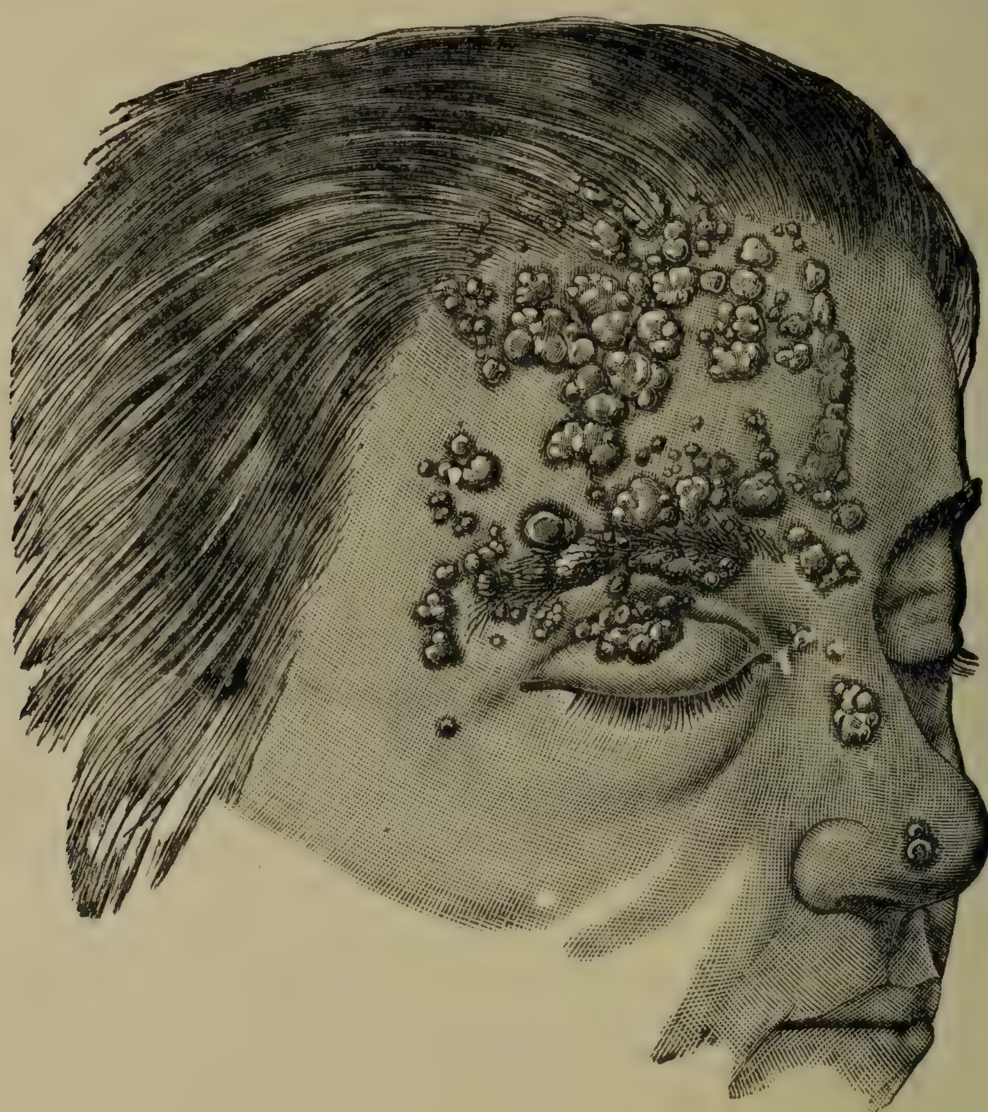


Fig. 198.—Herpes Zoster Ophthalmicus.

In a girl twenty years of age. Beginning of the disease five days previous. The vesicles occupy the region of distribution of the first branch of the trigeminus. This is the case even with those on the nose which extend down to the tip of the latter (corresponding to the distribution of the ramus naso-ciliaris of the first branch).

At first the vesicles contain a limpid fluid, which soon becomes cloudy and purulent, and finally dries up into a crust. If this is removed, an ulcer is found beneath it, a proof that the suppuration has penetrated into the corium. After the ulcer heals, cicatrices remain which are visible during the whole life, and by their characteristic arrangement render it possible to diagnosticate the previous existence of a herpes zoster even years afterward. By this formation of cicatrices the vesicles of herpes zoster are distinguished from those of herpes febrilis, in which the epidermis alone is detached by the fluid, so that they heal without leaving any trace of their existence behind.

The affection of the skin is very frequently complicated with an affection of the eye, consisting either of a keratitis or an irido-cyclitis. By the presence of such a complication the prognosis of herpes zoster is rendered essentially worse.

It is said that the eye is implicated only in those cases of herpes zoster ophthalmicus, in which the naso-ciliary branch is involved (Hutchinson).

The *cornea* may be implicated in various ways. Herpes vesicles may break out on it, from which quite large ulcers may develop (§ 566). In other cases deep infiltrates may form which do not undergo purulent disintegration, but are very slow in disappearing (keratitis profunda; see § 581). The cornea may also be affected indirectly, in that a paralysis of the trigeminus remains, and as a consequence of this a keratitis neuroparalytica is set up. Fuchs has seen two cases in which herpes was complicated with facial paralysis, and as a result of the latter, a keratitis e lagophthalmo developed. *Iritis* is often associated with these various diseases of the cornea, and iritis and irido-cyclitis may also develop as a result of herpes independently—i. e., without there being any simultaneous affection of the cornea at all (§ 705). Mydriasis and paralysis of the oculo-motor or abducens nerves also occur in consequence of herpes zoster.

After the subsidence of herpes zoster, *anomalies in the function of the trigeminus* often remain; anæsthesia or neuralgia, or both combined, persisting for a long time in the area supplied by the affected branches. The cornea, which even while the inflammation is still present is less sensitive than normal, usually remains undersensitive for a long time. The two following phenomena likewise must be referred to alterations in the nervous influence: The first is the abnormally low tension that the eyeball frequently shows when implicated in the inflammation; the second is the striking elevation of temperature of the skin upon the affected side, which not only is present while the inflammation is recent, but often lasts for quite a long time afterward.

The *cause* of herpes zoster ophthalmicus is an inflammatory affection of the trigeminus, which is located either in the trunk of the nerve itself or in the Gasserian or ciliary ganglion. By what means this inflammation is produced remains in most cases unknown. In some cases herpes has been seen to develop as a result of chilling of the body, head injuries, the use of arsenic, and poisoning by carbon monoxide gas.

The *treatment* of herpes zoster is purely symptomatic. We avoid opening the vesicles, as by doing so the raw surface in the skin would be exposed and pain would be excited. To prevent this we sprinkle the affected spots with dusting powder which cause the vesicles to dry up into crusts, beneath which the ulcers can heal undisturbed. The affection of the cornea or of the iris is to be treated according to the ordinary rules.

416. Eczema.—Eczema of the lids occurs both in an acute and a chronic form. *Acute* eczema is often artificial, i. e., caused by the application of substances which the skin does not tolerate, such as tincture of arnica, blue ointment, adhesive plaster, etc. At the outset, as long as the skin is simply red and swollen, the disease looks like erysipelas, but is distinguished from it by the fact that in erysipelas the skin is infiltrated throughout its entire thickness, and hence feels much thicker and firmer than in eczema.

Chronic eczema either develops from an acute eczema or is chronic from the start. It may be simply one of the evidences of an eczema that is distributed widely over the body, or it may be present in the lids alone. In the latter case it usually has a local cause, which is most often a wetting

of the skin of the lids with the tears in chronic conjunctivitis, ectropion, affections of the lacrimal sac, etc. In these cases often only the lower lid is attacked.

Acute eczema is treated with compresses of aluminum acetate, and washing with water is avoided. Afterward when the swelling of the skin has gone down ointments are indicated. Of these we select either diachylon ointment (Hebra) or ointments made with zinc oxide or white precipitate (1 or 2 per cent) or with ichthyol (5 or 10 per cent). The ointments are spread thickly on pledgets of linen, which are laid on the closed lids and retained there by a bandage. In extensive eczema the entire face is covered with a linen mask smeared with ointment on the inside.

In chronic eczema the application of ointments or pastes is likewise indicated, and the skin at the same time is protected by them from the tears. In squamous eczema we use tar ointments and a lotion of salicylic spirit.

Eczema of the lids is especially common in children, particularly under the form of moist eczema, which is called by the name of crusta lactea (milk crust). It forms the most frequent accompaniment of phlyctenular conjunctivitis. The connection between eczema and conjunctivitis is either this, that both owe their origin to the same causal disease, scrofula, or else the eczema is the result of the conjunctivitis. For, as the latter is associated with profuse lacrimation, the lids are constantly moistened with the overflowing tears, and hence become eczematous. Besides, children are in the habit of rubbing their eyes with their hands, so that all the parts about the eye are wet with the lacrimal fluid. The eczema requires treatment both on its own account and also because of any eczematous conjunctivitis that may chance to be present. The latter is cured much more quickly if at the same time the eczema of the skin is got out of the way—contrary to the popular belief which is disposed to take the opposite view. The treatment in this case too is performed by means of the ointments, above given. Another effective method of treatment consists in the application of a 5-to 10-per-cent solution of silver nitrate (see § 488).

Eczema at the border of the lid, being modified by the peculiar anatomical structure of the region, appears under a special form and will receive separate description later under the head of blepharitis ciliaris (§ 427).

2. *Phlegmonous Inflammations of the Lids*

417. Under this head belong: 1. *Abscesses of the Lids.* These originate most frequently after injuries. In other cases the affection starts from the bones; periostitis and caries of the margin of the orbit lying at the root of it. This is especially apt to be the case in scrofulous children, in whom, moreover, the carious diseases of the margin of the orbit are frequently referable to injury. Lastly, erysipelas not infrequently gives rise to abscesses of the lids, if the inflammation penetrates from the skin into the deeper parts. 2. *Furuncles,* which mainly occur in the region of the eyebrows. 3. *Anthrax Pustule* (malignant pustule). This arises through a transfer, by a process of inoculation, of the poison of anthrax (the bacillus anthracis) from animals affected with anthrax to man. It is hence most frequently found

in those persons who have to do with animals or the products obtained from them—e. g., in hostlers, shepherds, graziers, butchers, tanners, and furriers. The disease sometimes terminates fatally.

The symptoms of the phlegmonous processes in the lids are marked inflammatory œdema and indurated infiltration in the skin of the lid or beneath it. Associated with this are swelling of the lymphatic glands in front of the ear and near the lower jaw, and fever and prostration. In the after-course of the disease disintegration of the infiltrated portion of the skin takes place, or, if the case is one of abscess, softening of the infiltrate sets in, with escape of the pus externally by its breaking through the skin. Not infrequently extensive gangrene of the skin of the lids occurs. The result of this is cicatricial shrinking of the lid in the course of healing and its consequent contraction, so that lagophthalmus or ectropion is produced. In both erysipelas and malignant pustule it is not uncommon for both lids to be affected.

It is a peculiar feature of phlegmonous inflammation that the destructive process even when of great extent leaves exempt the free borders of the lids together with the cilia that they bear. This exemption is perhaps to be ascribed to the fact that of all portions of the lids the free border is most abundantly supplied with blood-vessels, and hence less readily falls a prey to necrosis. The preservation of the border of the lid is a very favorable circumstance for those cases in which a plastic operation upon the lids afterward becomes necessary, since the border of the lid can be used to skirt the edge of the implanted flap.

In cases of tuberculous disease of the orbital margin, the suppuration often occurs without inflammatory symptoms of any account and under the guise of a cold abscess.

Treatment follows the general rules of surgery. In abscesses of the lids an incision should be made as early as possible (that is, as soon as we are able to make the diagnosis), in order to prevent the extension of the suppuration into the deeper parts (orbit and meninges). However, in cold abscesses we may first make the attempt to cure the abscess together with the bone lesion by aspirating the pus with a syringe and then injecting some iodoform emulsion into the abscess cavity. When the skin of the lids is destroyed by inflammation, it is our business to strive to prevent as far as possible the subsequent contraction of the lids due to cicatrization. In large losses of substance in the lids it is best to refresh the edges of the two lids in isolated spots and unite them by sutures. As long as the palpebral fissure is kept closed in this way, lagophthalmus cannot develop, and the cicatrix that forms is broader. It is also advisable in such cases to graft bits of skin upon the granulating surface of the injured lids. In order not to endanger the result by a secondary shrinking of the newly formed cicatrix, the lids that have been thus artificially united are not separated again until some months after the cicatrization has been completed. If in spite of these measures, such a considerable contraction takes place that lagophthalmus or ectropion is caused by it, the skin which has been destroyed must be replaced by blepharoplasty.

418. Mortification of the Skin of the Lids.—This occurs under two forms, dry mortification (*necrosis*), and moist mortification associated with putrefaction (*gangrene*). Mortification develops from (1) intense inflammation of the skin of the lid itself. This is most frequently the case in erysipelas and also in anthrax; and, very rarely indeed, mortification occurs in variola. (2) Severe inflammation of the structures situated beneath the skin of the lids, such as the subcutaneous cellular tissue (in phlegmons) and even the conjunctiva itself (in gonorrhœal conjunctivitis and diphtheria). Cases of the latter kind are extremely rare, yet Fuchs saw a child with ophthalmia neonatorum in whom all four lids became gangrenous. The child not only escaped with its life, but even partly regained its sight. (3) Occlusion of the vessels of the lid by embolism (in pyæmia and sepsis) or thrombosis (in exhausting diseases). (4) Infection of peculiar character (hospital gangrene, noma). (5) Traumatisms, such as crushing, burning, corrosion and freezing (from too prolonged application of iced compresses).

3. *Ulcers of the Skin of the Lids*

419. Tuberculous Ulcers (Scrofula, Lupus).—Ulcers are produced partly as the result of injuries (burns, the action of caustic substances, and contusion), partly spontaneously. Of the latter kind are the tuberculous (including as special forms scrofulous and lupous) ulcers and syphilitic ulcers. The *scrofulous* ulcers develop generally as a result of caries of the orbital margin and, like those that originate in suppuration of the lymph glands, present an irregular shape and reddened or livid borders, either take a long time to get well or keep constantly recurring, and in their later stages are distinctly puckered. The outcome is a very indrawn, very disfiguring scar, which is adherent to the bone and produces ectropion (Fig. 249). *Lupus* also occurs frequently on the lids, to which it usually migrates from the neighboring parts, such as the nose or cheek. It is most easily recognized from the characteristic nodules or scars that are encountered in its vicinity. From the lids it may pass to the conjunctiva and even to the eye itself; and so, when lupus of the face has lasted for a long time, considerable changes in the lids and eyeball (ectropion, xerophthalmus) are often found, which may produce complete blindness.

420. Syphilitic and other Ulcers.—The syphilitic ulcers of the lids are either examples of initial sclerosis (hard chancre) or disintegrating gummata.

Chancre of the lid is usually found either in the intermarginal space or at the canthi or on the tarsal conjunctiva. Characteristic features are its hardness, its grayish-yellow lardaceous base, its chronicity, the attendant enlargement of the preauricular or submaxillary glands, the presence of the spirochætæ, and the positive Wassermann reaction (Alter, Salzmann). Occasionally the cornea may be seriously involved. Very rarely double or even triple chancres are found (Finlay). *Gummata* form indolent tumors which usually develop at the same time with gummata of the skin in other parts of the body (syphilitic rupia). At the onset they resemble a chalazion or hordeolum, but are distinguished by the fact that they break down into indurated, painless ulcers which have a lardaceous coating and tend to spread to the deeper parts, in some cases even perforating the lid. Occasionally they are absorbed spontaneously. Very similar tumors may be caused by excessive doses of the iodides (Stephenson). In the diagnosis of gummata the luetin test is particularly valuable, as it may be positive when the Wassermann test is not (Alter)

Soft chancre and *lepra* also occur in on the lids.

Vaccine ulcers may develop on the lids from the transfer to the latter of secretion from vaccine pustules. They form pretty large, very coated ulcers, situated on the edges of the lids, and accompanied by considerable œdema of lids and even conjunctiva.

To these symptoms are added swelling of the lymph gland in front of the ear, and sometimes even fever. They heal in one or two weeks, leaving no scars, but simply spots where the cilia are deficient. In some cases, however, the infection is transmitted to the cornea producing perforating ulcers and symblepharon (Bedell).

421. Blastomycetic Dermatitis.—This is a disease which so far has been observed only in America, and mainly in the Mississippi valley. It begins as a papule, changing speedily to a pustule and then to a spreading, tumor-like mass composed of irregular elevations and containing miliary abscesses, from which pus oozes spontaneously or under pressure. The disease has a serpiginous growth and may produce extensive destruction of the skin, but rarely, if ever, attacks the conjunctiva. It looks very like epithelioma, but is due to a peculiar yeast-like budding organism, which can be obtained from the abscesses. Potassium iodide up to 10 gm. three times a day is almost a specific (Bevan), and the application of the x-ray or radium is also beneficial (Fagin).

4. *Œdema of the Lids*

422. Œdema of the lids is of course not a disease but only a symptom yet as such it is so frequent and at the same time so conspicuous that it deserves quite a detailed description. Its development is favored to an unusual degree by the anatomical structure of the lid (see § 2): hence it is found not only in connection with every violent inflammation of the lids themselves or of the neighboring parts, but also in consequence of simple venous congestion or an altered state of the blood. In the former case we are dealing with *inflammatory œdema* (œdema calidum), in the second case with a *non-inflammatory œdema* (œdema frigidum). As long as the œdema is on the increase, the skin of the lid is found to be smooth and tense; but as soon as the œdema begins to decrease, this is at once made manifest by the formation of minute wrinkles in the skin of the lid—a symptom which is therefore of value as affording evidence that the process has already passed its acme.

Œdema of the lids often causes the patient more alarm than does the lesion which lies at the bottom of it, because he cannot open the swollen eye, and hence cannot see with it. And for the less experienced physician marked œdema presents difficulties, inasmuch as it hinders the accurate inspection of the eyeball. If, in consequence, the physician gets but a transient view of the eye, or no view at all, he may easily make a false diagnosis, and may cause the patient great anxiety for what is perhaps an insignificant affection. For the benefit of the general practitioner, therefore, those affections which are associated with œdema of the lids will be enumerated in the following lines, and at the same time the symptoms will be given from which the diagnosis can be made.

The first thing to be done is to open the lids sufficiently in spite of the œdema, for which purpose we may with advantage use Desmarre's elevator (see Fig. 388, *d*), especially in the presence of marked swelling or violent blepharospasm. We then see whether or not the conjunctiva is free from redness, and the eyeball itself is normal, not protruding, and freely movable.

(*a*) *Upon Separating the Lids the Deeper Parts Appear Normal*

423.—It is necessary first to determine whether what we have before us is an inflammatory or a non-inflammatory œdema. The former is distinguished from the latter by the redness, the increased temperature, and not infrequently also by the sensitiveness to touch. Let us assume that we are dealing with an *inflammatory œdema*. In order to find out what affection lies at the bottom of it, we now try to ascertain whether in palpating the swollen part we do not come upon some one spot which is distinguished by greater induration and special painfulness.

1. If such a spot is found close to the free border of the lid, we are usually dealing with a *hordeolum*. In the very inception of this affection nothing besides the above-

mentioned symptoms are noticeable. But, in the days immediately following, a yellowish point of discoloration is discovered either between the cilia, or, if we are dealing with a Meibomian sty, upon the inner surface of the lid.

2. If the indurated and sensitive spot occupies the internal angle of the eye, our first thought must be of an acute *dacryocystitis*. This diagnosis is confirmed, if, upon pressure in the region of the lacrimal sac, pus is evacuated from the puncta, or if the patient says that epiphora has for a long time preceded the inflammation. To be sure, a furuncle or a periostitis may also develop in the region of the lacrimal sac, but these cases, in comparison with the frequently occurring dacryocystitis, are extremely rare.

3. In œdema of the lids due to a *furuncle* or a *malignant pustule* there is felt, contrary to what takes place in erysipelas, a circumscribed, indurated, and painful nodule of considerable extent in the skin of the lid itself; while, if the infiltration lies deep in the tissues, we are dealing with a commencing *abscess* of the lid. In *periostitis* of the margin of the orbit the latter can be felt through the œdematous lid, and it is then found to be not sharp but thickened and enlarged, and tender to the touch.

4. In *erysipelas* the redness and swelling of the lid are uniform. The skin itself when grasped between the fingers feels thicker and harder; while, on the other hand, circumscribed infiltration is absent. The swelling, as a rule, occupies both lids and also extends to the neighboring parts; and when we have had the case under observation for some time we can see that the swelling migrates. If in the course of the inflammation an induration develops which can be felt to be deeply seated, it is a proof that the process has penetrated into the deeper tissues, and that an abscess of the lid is forming.

Cases of erysipelas sometimes occur which are very slight in intensity and extent, and present correspondingly insignificant inflammatory symptoms. Then only the lids themselves, and perhaps the dorsum of the nose, too, are swollen; these parts are not tense but of doughy consistence, and are scarcely reddened; and fever and pain are absent. The swelling disappears within a few days, and the skin then peels off, but it may also happen that the doughy swelling lasts for weeks or even months—a condition of things that the dermatologists call by the name of erysipelas perstans. Such light cases of erysipelas usually recur, and thus acquire a resemblance to the cases of—

5. *Recurrent neurotic œdema* of the lids. In this a very marked œdematous swelling of the lids—a swelling, however, which is usually free from redness—suddenly makes its appearance, and then very rapidly—often within a few hours—disappears again. Frequently there are associated with it similar œdematous swellings in other parts of the body—e. g., on the lips, the trunk, or the extremities, more rarely in the larynx or pharynx. These transient attacks of œdema are referred to temporary disturbances in the innervation of the vessels (angioneuroses), and are allied to urticaria. They occur most frequently in women, and especially at the time of the menses.

6. Acute *eczema* of the lids is often associated with marked œdema, and in the first few days of its development may be hard to distinguish from an erysipelas.

Chronic eczema squamosum often also causes a moderate œdema of the lids in old people the skin of whose lids is lax, and it is then readily overlooked because the skin affection often manifests itself only by an insignificant roughness of the surface when stroked with the finger—the roughness being due to the slight desquamation.

7. Œdema of the lid due to *traumatism* is almost always accompanied by extensive hæmorrhagic suffusion of the lid, and from this fact can readily be recognized. If œdema of the lid develops in consequence of the sting of an insect, it is easy to make the diagnosis when we are able to discover the site of the sting.

Non-inflammatory œdema is met with as one of the symptoms of general œdema, as, for example, in heart disease, in hydræmia, and in nephritis. Not infrequently the lids are the very first part of the body in which these varieties of œdema show themselves, and thus give warning of the causal disease. In such cases the œdema of the lids sometimes appears under the guise of flying œdema (œdema fugax)—i. e., it comes suddenly and disappears again within a few days or even a few hours, only to return after a short interval of time.

A variety of œdema holding an intermediate position between the inflammatory and the non-inflammatory kinds, is that which is observed in connection *with blepharo-*

spasm that has lasted a long time (especially in children with phlyctenular conjunctivitis). This chiefly affects the upper lid, and is mainly referable to the compression of the palpebral veins, by the contracted orbicularis (see § 485)

(*b*) *Upon Separating the Lids Changes are Found in the Conjunctiva or Eyeball*

424. 1. Among affections of the *conjunctiva*, those associated with œdema of the lids are acute blennorrhœa and diphtheria, less frequently a violent catarrh, or, as above stated, a phlyctenular conjunctivitis. The diagnosis is easily made from the appearance of the conjunctiva and from the character of the secretion.

2. Violent *inflammations in the interior of the eyeball* lead to œdema of the lids; severe irido-cyclitis and acute glaucoma doing so to a less extent, *panophthalmitis* to a more considerable degree. In the latter disease, as in acute blennorrhœa, chemosis is also present. A confusion between the two diseases can, however, be readily avoided; since in panophthalmitis the purulent secretion in the conjunctiva is wanting, while a purulent exudate is visible in the interior of the eye (in the anterior chamber or in the vitreous). An important differential sign is the protrusion of the eyeball and the consequent diminution in its mobility in panophthalmitis, symptoms which are never present in acute blennorrhœa.

3. *Tenonitis, orbital cellulitis*, and *thrombosis of the cavernous sinus* share with panophthalmitis the symptoms of œdema of the lids, chemosis, and protrusion and immobility of the eyeball. These affections might hence be confounded with each other and with panophthalmitis. From the latter, however, they are at once distinguished by the fact that in all three the eyeball itself, except for the œdema of the conjunctiva, looks normal in its anterior portion, while in panophthalmitis the suppuration in the interior of the eye is visible. For the differential diagnosis between the three affections first named, see § § 872 and 873.

A *tumor* developing in the depth of the orbit may also, along with the protrusion of the eyeball, cause œdema of the lid due to congestion. In this case, however, inflammatory concomitants are either slight or are absent altogether.

425. Dystrophy of the Skin of the Lids.—In old age the skin of the lids loses its elasticity and becomes wrinkled and lax. To a less extent this loss of firmness also affects the fascia tarso-orbitalis that lies beneath the skin. In that case this fascia and the skin with it are pushed forward by the orbital fat, especially in fat persons. Thus develops the well-known baggy bulging of the lower eyelids, seen in old people. In the upper lid it is especially the skin over the inner angle of the eye that protrudes in this hernia-like fashion.

A relaxed condition of the skin, which occurs in young persons as well as old is the *ptosis adiposa* of Sichel, which consists in the fact that the covering fold of the upper lid is of unusual size, so as to hang down over the free border of the lid in the region of the palpebral fissure. It was formerly assumed that this enlargement was caused by an excessive accumulation of fat in the covering fold for which reason the name of ptosis adiposa was given to it. Its true cause, however, is that the bands of fascia connecting the skin with the tendons of the levator and with the upper margin of the orbit are not rigid enough; consequently the skin is not properly drawn up when the lid is raised but hangs down in the form of a flabby pouch (Hotz).

A condition differing from ptosis adiposa is *blepharochalasis* (χάλασις, relaxation). In this the skin of the upper lid becomes so thin that it lies in countless little wrinkles, and looks like rumpled tissue paper. Moreover, owing to the dilatation of numerous small superficial veins, it acquires a red appearance. In consequence of its relaxation and the looseness of its attachment it hangs down like a pouch; while the border of the lid itself is scarcely lower than usual. This change occurs in cases in which there have been frequent antecedent œdematous swellings of the lid (e. g., it occurs after recurrent neurotic œdema of the lid, see § 423), as a result of which the skin is stretched and loses its elasticity.

Except for the disfigurement they cause, neither ptosis adiposa nor blepharochalasis entails any disagreeable symptoms. They can be remedied by ablating the excess of skin, and attaching the skin to the upper border of the tarsus by Hotz's operation, so as to prevent its drooping (see § 928).

426. Other Cutaneous Affections.—*Elephantiasis* affects the lids under the form of a monstrous thickening, especially pronounced in the upper lid which hangs down over the lower lid and upon the cheek, and which, on account of its weight, cannot be raised, and thus renders vision with the eye so covered impossible. Elephantiasis usually develops as a result of repeated inflammations of the lids. The treatment consists in excision of the skin to a sufficient extent for the lid to regain approximately its normal dimension.

Under the name of *chromidrosis*[1] is denoted that rare affection in which the sweat from the skin of the lids is colored. As a result of it blue spots come out upon the lids which can easily be wiped away with a cloth that has been dipped in oil, although in a short time they make their appearance again. This disease is said to occur especially in women. A large number of the known cases must probably be referred to simulation—i. e., to the intentional application of some blue coloring matter to the lids. For xeroderma pigmentosum, see § 446.

II. Inflammation of the Border of the Lids

427. Varieties.—The free border of the lid is simply a part of the skin of the lid, which, however, is distinguished by many anatomical peculiarities such as its cilia with their hair follicles and glands, its particularly abundant vascular supply, etc., so that its diseases bear a special stamp. Affections of the borders of the lids are among the most frequent of all diseases.

Hyperæmia of the border of the lid manifests itself by the reddening of it, so that the eyes look as if rimmed with red. It occurs in many people in consequence of insignificant injurious influences, such as prolonged weeping, great straining of the eyes, staying in vitiated air, a wakeful night, etc. This is especially true of persons with a delicate skin, who at the same time have a light complexion and blonde or reddish hair. In many of these persons the hyperæmia of the lids is present all the time, and sometimes lasts for their whole life. With respect to the troubles that it causes, and also with respect to its treatment, the same statements hold good that will be made in speaking of blepharitis.

Inflammation of the border of the lids (blepharitis ciliaris or blepharoadenitis[2]) appears under the two following principal forms:

1. *Blepharitis Squamosa.* In this the skin between the cilia and in their vicinity is covered with small, white or gray scales looking like dandruff or bran. If the scales are removed by washing, the skin beneath them is found to be hyperæmic but not ulcerated. Upon removing the scales, some cilia usually fall out—a proof that they are less firmly attached than usual; but, as their follicles are not injured, they grow again afterward.

A sub-variety of blepharitis squamosa, which is of less frequent occurrence, appears under the following form: The border of the lids is covered with yellow crusts, which are sometimes rigid, sometimes flexible and fatty (like wax or honey). When they are removed, no ulcers are found beneath them, but simply reddening of the skin of the lid. The yellow crusts are therefore not inspissated pus, but simply the excessively abundant secretion of the sebaceous glands, which has solidified in the air into yellow crusts.

2. *Blepharitis Ulcerosa.* In this form also the border of the lid is covered with yellow crusts; but, after washing them off, we find not merely a hyperæmia of the skin but ulcerative processes as well. Thus we see here

[1] From χρῶμα, color, ἵδρωσις, sweating.
[2] From βλέφαρον, lid, and ἀδήν, gland; i. e., inflammation of the glands of the lid.

and there in spots small yellow elevations, from the centre of which rises a cilium. These are little abscesses, which have originated from suppuration of a hair follicle and of the sebaceous gland belonging to it. With these we find little excavations—that is, ulcers which have been formed out of small abscesses that have opened. Again, in other spots we notice small scars, the remains of similar ulcers. At the site of the scars the cilia are permanently deficient, because their hair follicles have been destroyed by suppuration. Since new hair follicles are constantly being transformed, one after another, into abscesses, the row of cilia, when the process has kept up for a long time, becomes more and more thinned out; the cilia that are still present are arranged in separate groups, which for the most part are glued together into tufts by the dried secretion. Blepharitis ulcerosa, accordingly, is distinguished from blepharitis squamosa by its deeper situation and the purulent character of the inflammation. It is hence to be regarded as the more serious of the two forms, the one in which both the inflammatory symptoms are more pronounced, and permanent sequelæ, particularly destruction of the cilia, remain.

Since the border of the lids is simply a modified portion of the external skin, it would be proper to consider the affections of it from the *dermatological* standpoint—that is, to compare them with analogous affections of the skin. From this point of view blepharitis squamosa might be regarded as seborrhœa. That form which is associated with furfuraceous scales would correspond to seborrhœa squamosa or seborrhœa sicca of the skin, which we most frequently observe, under the guise of increased formation of scales, upon the scalp. The form of blepharitis squamosa which is characterized by yellow, fatty crusts, is probably identical with the seborrhœa oleosa, which is likewise found upon the scalp, particularly in children. In blepharitis ulcerosa it is a case of eczema, which, on account of the hairy character of the border of the lid, is associated with suppuration of the hair follicles, such as is also the case in other hairy portions of the skin. For the inflammation creeps from the epidermis into the hair follicle, and the staphylococci which enter the gland from its orifice cause the suppuration. If the hair follicle alone suppurates, the small pustules, such as are ordinarily found, develop on the margin of the lid. But if the inflammation makes its way from the interior of the hair follicle to the surrounding connective tissue, so that the latter also suppurates, a somewhat larger circumfollicular abscess is formed. This corresponds to the acne pustule of the skin and when occurring on the lid border is called hordeolum. A genuine sycosis of the lid border (trichophytia) also occurs, although very rarely (Herzog). (For sportrichosis, see § 477.)

For the correct *diagnosis* of blepharitis the crusts covering the border of the lid must be removed, in order to determine the state of the skin beneath them. If the skin beneath the crusts is normal, we are not dealing with blepharitis at all, but with a disease of the conjunctiva, the dried secretion from which forms the yellow crusts. In blepharitis the skin is at least found to be reddened (in blepharitis squamosa), or it is covered with ulcers (in blepharitis ulcerosa). In the vicinity of the ulcers the skin of the palpebral border not infrequently is thickened by hypertrophy of the papillæ so that warty excrescences are formed, which are painful and bleed readily, and which must be removed.

428. Symptoms and Course.—The annoyance suffered by the patient is slight in the lightest cases of blepharitis, so that many patients visit the physician more on account of the disfigurement due to the reddened border of the lids than on account of any distress they experience. But in most cases the patients are annoyed by the increased sensitiveness of the eyes

which water readily, especially during work and in the evening, are sensitive to light, heat, and dust, and tire quickly. In the morning the lids are stuck together.

Blepharitis is distinguished by its eminently chronic *course*, which often extends over a series of years. In young patients the disease often disappears of itself, when they grow up; in others it continues during the whole life. Proper treatment always produces considerable improvement, or even effects a cure, which latter, however, is in most cases not lasting, as after the discontinuance of the treatment the disease usually returns; a permanent cure is obtained in only a few cases.

After lasting some time blepharitis entails a series of *sequelæ*, which to a certain extent react upon the blepharitis and render it worse. These are—

1. *Chronic conjunctival catarrh.* This is the constant concomitant of blepharitis, the annoyance produced by which is in no small part due to it.

2. Blepharitis ulcerosa leads to permanent *destruction of the cilia.* This may go on till nearly all the cilia are lost. In that case there are found upon the border of the lid a few scattered, minute, and abortive hairs. This condition, called *madarosis,*[3] produces marked disfigurement. As soon as all the cilia have been destroyed, the blepharitis ceases of itself, since there are no longer any hair folicles to undergo suppuration.

3. By the traction produced by scars which remain after suppuration of the hair follicles, neighboring cilia may be given a false direction, so as to turn backward toward the cornea (*trichiasis*).

4. *Hypertrophy* of the border of the lid may develop in consequence of its being constantly congested and swollen by inflammation. The lid is then found to be thicker and more misshapen at its free border, and drooping in consequence of its weight (*tylosis*).[4] This change affects mainly the upper lid.

5. The lower lid very often undergoes a change of position under the form of *ectropion.* This develops in the following way: Owing to the formation of the scars, the conjunctiva is drawn a little forward over the border of the lid. The border of the lid then looks as if it had a rim of red conjunctiva about it, and the posterior margin of the lid, which before was sharp, is now rounded off, and can no longer be distinctly made out. In consequence of this change of form, the borders of the two lids no longer fit exactly to each other when the latter are shut together. Furthermore, on account of the absence of its sharp posterior margin, the lid is no longer perfectly applied to the eyeball, and a shallow groove remains between the eyeball and the border of the lid (*eversion of the border of the lid*). In common with the border of the lids, the puncta are also turned forward so as no longer to dip into the lacus lacrimalis (*eversion of the puncta*). Thus the conduction of tears into the lacrimal sac is interfered with, so that epiphora develops. A portion of the tears runs down over the lower border of the lid upon the skin of the latter which because of this continual wetting becomes reddened, excoriated, and even eczematous; consequently it loses its pliability and becomes gradually contracted. In this way the lower lid

[3] From μαδᾶν, to melt away, to fall off. [4] From τύλος, a callous spot.

is drawn farther and farther away from the eyeball, so that an ectropion of the entire lid is gradually developed from the eversion of its border. At the same time the lacrimation also constantly increases, and this in turn reacts injuriously upon the blepharitis, the lid border being irritated to the point of inflammation by the tears that constantly flow over it.

429. Etiology.—The causes of blepharitis are either general or local.

The *general causes* lie partly in the constitution of the patient, partly in external injurious influences. In the first category are to be mentioned anæmia, scrofula, and tuberculosis, which particularly in children and young people furnish a frequent cause of blepharitis. When with increasing age the constitution improves, the blepharitis also disappears. In many families blepharitis is hereditary, being a kind of family disease. Among external injurious influences are to be considered all those which are known to be also causes of chronic conjunctival catarrh (see § 456). Among these are vitiated air, smoke, dust, heat (e. g., in the case of stokers), late hours, etc. Blepharitis due to general causes is always bilateral.

Among the *local causes* of blepharitis the most frequent are chronic inflammations of the conjunctiva (chronic catarrh, phlyctenular conjunctivitis, and trachoma) and epiphora. The latter excites inflammation of the border of the lid by keeping it continually wet. The epiphora may be caused either by increased secretion of tears or by interference with their discharge into the nose. The former is the case, for example, in phlyctenular conjunctivitis, which is characterized by profuse lacrimation. Since in this case the inflammation of the conjunctiva and the scrofulous diathesis of the patient act both at the same time to favor the development of blepharitis, it is easy to understand why phlyctenular conjunctivitis should so exceedingly often be found combined with blepharitis.

Epiphora may, however, also be produced by interference with the conduction of tears, as a result, for instance, of an affection of the lacrimal sac; in this case the blepharitis is found only in that eye in which the affection of the lacrimal sac exists. Hence, the rule is in unilateral blepharitis to examine the lacrimal sac at once, just as, on the contrary, in bilateral blepharitis we shall first have to look for a general condition as the cause of it. Another cause of interference with the conduction of tears, and hence also of blepharitis, is imperfect closure of the lids due to ectropion, to paralysis of the facial nerve, to congenital and acquired contraction of lids, etc.

430. Treatment.—The treatment of blepharitis must take account both of the causal indication and also of the local changes. Regard to the causal indication requires, so far as we can effect it, the improvement of the patient's constitution and of the hygienic conditions under which he lives. Local causes of blepharitis, such as lesions of the conjunctiva and the lacrimal sac, lagophthalmus, etc., are to be removed as far as possible. In the treatment of the diseased border of the lid itself ointments play the chief part. Their action is to be principally attributed to the fat they contain. This softens the scales and crusts and thus facilitates their removal, and also prevents the occlusion of the orifices of the palpebral glands; it renders the skin more pliable, and protects it from being wet by the over-

flowing tears. We must, therefore, select as the basis of the ointment a soft pliable fat, which also should be somewhat hydrous so that by the evaporation of its water the ointment may produce a refrigerant action. Cold cream and lanolin fulfill these requirements; the latter should be mixed with an equal amount of vaselin to make it more pliable. Of medicaments added to the ointments the mildest are boric acid (2 per cent), white precipitate (1 or 2 per cent), and ichthyol (2 to 5 per cent); rather more irritant in action are salicylic acid, resorcin, and yellow precipitate (all in a strength of from 1 to 2 per cent). (One must not forget that if the excipient of the ointment becomes rancid, the latter itself becomes irritating.) The way to apply the lid ointment is for the patient to rub it with the finger on the closed interpalpebral fissure before going to bed. Before he does this the crusts and scales that stick to the edge of the lids should be removed as carefully as possible by washing with lukewarm water so that the ointment shall come in contact with the skin itself.

A serviceable way of doing this is the following. Close the eyes gently. Make a good lather with any good soap or shaving cream and fill the eyelashes with it as if to shave them, grasp the eyelashes with a damp cloth held between thumb and finger, and with this, stroke them out so as to remove all lather, scales and loose lashes. Crusts or scales may be also loosened and teased off by stroking the cilia with a cotton swab wet with hydrogen peroxide. When the lashes are clean, dry them, then rub a very little of the ointment (preferably a 1-per-cent ointment of yellow oxide of mercury) gently into the roots of the lashes and edges of the lids. Wipe off all excess of ointment. The cleansing is to be done again in the morning after washing off the remnants of the ointment.

The physician must insist very particularly upon the cleansing since it is often painful, and hence, especially in children, is frequently not performed with sufficient care. In blepharitis squamosa a tar soap or salicylic tincture or chlorine water (1 part of chlorine water to 64 of water—Wilmer) is serviceable for cleansing the lids each morning, and in this condition the application of tar ointments or an alcoholic solution of tar is helpful, although not always well borne. In all forms of blepharitis Wilmer finds 2 per-cent iodine helpful and he also uses 2-per-cent optochin. When we have finally succeeded in bringing the border of the lids back to their normal state, we continue the use of the salve for some time longer, as otherwise the blepharitis will very soon recur.

In blepharitis ulcerosa, in addition to the employment of the ointment, the abscesses which form must be opened every day, and the cilia that project from them must be epilated. For this purpose we use the cilium forceps—that is, a forceps with broad rounded ends (Fig. 199). The healing of the ulcers can be accelerated by touching them lightly with a pointed stick of nitrate of silver.

In the treatment of blepharitis the mistake is very frequently committed of prescribing highly *irritant ointments*, by which the inflammation is simply aggravated. In severe cases of blepharitis ulcerosa it is advisable to smear the ointment thickly upon a pledget of linen and apply it over night to the eyes by means of a bandage. In this way the ointment acts in a much more penetrating way than if it were merely smeared upon the borders of the lids. This procedure is particularly indicated when the blepharitis is caused by congenital shortness of the lids, because here the closure of the palpebral fissure during sleep is at the same time insured (see § 447).

In many cases of blepharitis ulcerosa, particularly if *tylosis* is present at the same time, we will fail to accomplish our object until we have epilated all the cilia. We do this in several sittings, and subsequently also remove the cilia that grow in afterward, keeping on in this way until the border of the lid once more looks perfectly normal. We need not fear that the cilia, even if we have epilated them ever so often will ultimately fail to grow in again. For the tylosis itself, massage of the lid, with the aid of the white-precipitate ointment, does good service. This acts partly by exciting resorption, partly because it helps to remove mechanically the contents of the palpebral glands, and thus prevents their occlusion.

Not to be confounded with blepharitis is the condition called *phthiriasis palpebrarum*—i. e., the presence of crab lice (phthirius inguinalis or pediculus pubis) upon the eyelashes. Here the borders of the lids look strikingly dark. Upon more careful inspection we discover as the cause of the discoloration the black nits of the crab lice sticking fast to the cilia; sometimes, too, a grown specimen is found lodged between the cilia. The

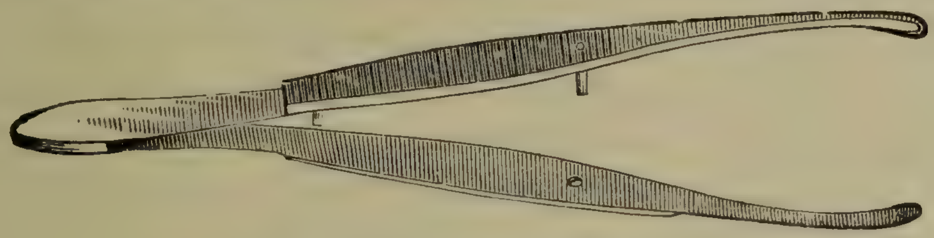


FIG. 199.—CILIUM FORCEPS.

disease, which is rare and which is found almost exclusively in children causes trouble only from the itching it produces. It is easily cured by means of blue ointment, which, when rubbed into the borders of the lids, kills the parasites.

431. Diseases of the Cilia.—Primary affections of the cilia are very rare. Loss of the eyelashes may occur as one of the symptoms of a general alopecia or as a result of an x-ray application to the eye. *White* cilia (poliosis or canities) occur as a congenital condition in albinism. An acquired blanching of the lashes is seen in different serious eye diseases, e.g. in trachoma and vitiligo, when it affects a few lashes, and in sympathetic ophthalmia, of which it is a rare manifestation, when it affects a larger number. In *trichorrhexis nodosa*, nodular thickenings form on the lashes, which then break off at the thickened points and become fibrillated. All these conditions may also affect the eyebrows (Salzmann).

III. DISEASES OF THE PALPEBRAL GLANDS

432. The glands which here come under consideration are the glands of the hair follicles of the cilia (Zeis's glands) and the Meibomian glands. The affections of the former have already been treated of in part under the head of blepharitis, a disease which presents a diffuse inflammation extending over the whole border of the lid, and in which the hair follicles are implicated. To be distinguished from this is the isolated inflammation which is limited to one or a few of these glands, and forms an independent affection. If such an inflammation develops as an acute affection, it is known as hordeolum; if as a chronic affection, it is known as chalazion. From these, again, are to be distinguished the cases of simple occlusion of the glands with inspissation of their contents but without inflammation—cases which form the starting point of the infarcts in the Meibomian glands.

433. Hordeolum[5] (Sty).—There are two varieties of hordeolum.

Hordeolum externum (or zeisianum) is produced by suppuration of one of Zeis's glands. There is first noticed an inflammatory œdema of the affected lid, which in severe cases may even extend to the conjunctiva

[5] From *hordeum*, barley. The proper classical form is *hordeolus*.

bulbi. Upon careful palpation there is discovered in the swollen lid a specially resistant and sensitive spot which lies near the border of the lid, and corresponds to the inflamed gland. In the next day or two the swelling at this spot increases, and the skin over it grows red, afterward shows a yellowish discoloration, and finally perforates near the border of the lids with a discharge of pus. After the evacuation of the pus the inflammatory symptoms rapidly abate, the small abscess cavity soon closes, and the entire process comes to an end. In spite of the fact that the disease lasts but a few days, it is still very burdensome to the patient on account of the often considerable pain in the tense and greatly swollen lids. Besides, many persons have a number of repeated attacks.

Hordeolum internum (or *meibomianum*) is much rarer than hordeolum externum. It consists in a suppuration of one of the Meibomian glands. The course of the disease is, on the whole, the same as that of the hordeolum externum; but as the Meibomian glands are larger than those of Zeis, and are enveloped in the firm connective tissue of the tarsus, the inflammatory symptoms are more violent, and it takes a longer time for the pus to be evacuated. The pus, as long as it is shut in in the affected glands, appears upon eversion of the lid as a yellowish spot shining through the conjunctiva. Afterward it breaks through the conjunctiva or is discharged through the orifice of the gland. Perforation through the skin occurs only as an exception, in contradistinction to hordeolum externum, in which this is the rule.

Hordeolum externum and internum are essentially the same process—i. e., they are both an acute suppuration of a sebaceous gland, for the Meibomian glands are nothing but modified sebaceous glands. A hordeolum is analogous to acne of the external skin. The violent inflammatory symptoms, and particularly the marked œdema, which distinguish a hordeolum from ordinary acne pustules of the skin, are caused by the peculiar anatomical structure of the lids, which predisposes the latter to inflammatory swelling.

Hordeolum is found principally in young people, particularly if they are of anæmic or scrofulous constitution and at the same time suffer from blepharitis. The latter, by causing accumulation of scales and crusts on the border of the lids, favors multiplication of the bacteria (staphylococci) that are always present there, which can thence readily penetrate into the orifices of the glands. Repeated crops of styes often indicate systemic disturbance, especially one due to digestive or menstrual disorders.

The *treatment* of hordeolum in the beginning of the disease consists in the use of moist warm compresses, which are applied to the lids in order to convert the hard infiltrate more rapidly into pus. When the yellow color of the pus is visible beneath the skin or the conjunctiva, the abscess may be opened by a small incision, and thus the duration of the inflammation may be shortened by several days. The prime means for avoiding a recurrence of the hordeolum is the treatment of any blepharitis that may be present. Yellow oxide ointment is, hence, a useful application for this purpose. Sometimes calx sulphurata given every three hours in doses of gm. 0.015 seems to abort a forming hordeolum. If there is a tormenting succession of hordeola, treatment with an autogenous vaccine prepared from the pus of the hordeola themselves may be tried.

434. Chalazion.—Chalazion [6] is a chronic affection of the Meibomian glands. It forms a hard swelling which develops very gradually in the lid. In many cases this occurs without any inflammatory symptoms whatever, so that the swelling is not noticed by the patient until it has become quite large. In other cases, however, there are moderate inflammatory concomitants, which, nevertheless, are insignificant in comparison with those which accompany a hordeolum. The tumor keeps constantly enlarging for months until it becomes as big as a pea or bigger; it then bulges the skin far enough forward to produce a perceptible disfigurement of the lid. Upon palpating the tumor we can make out that it is pretty resistant, and that it is intimately connected with the tarsus, while the skin lying over it can be displaced from side to side. Upon everting the lid we find the conjunctiva over the tumor reddened, thickened, and somewhat protruding. Later on, the tumor assumes a grayish look as seen through the conjunctiva, and ultimately the latter is perforated; then a viscid, rather turbid fluid flows out, which represents the central softened portions of the tumor. But the main portion of the latter, consisting of spongy granulations, remains, for which reason the tumor after it has opened does not at once disappear completely. On the contrary, it diminishes very gradually in size, and meanwhile it is not uncommon for the granulation masses to project like a fungoid growth through the perforation in the conjunctiva. It requires months more for the tumor to disappear completely.

Chalazion shares with the hordeolum internum its situation in the Meibomian glands, but is distinguished by the character of the process. Hordeolum is an acute inflammation, which goes on to suppuration and is over in a few days. Chalazion is a chronic disease, which does not lead to suppuration but to the formation of granulation tissue, and lasts for months or even years. It was formerly regarded as a cyst of the Meibomian glands, and analogous to the atheromata of the sebaceous glands. Such retention cysts do occur, but they are rare, and are essentially different from chalazia. In a chalazion there is a peculiar chronic inflammation, which produces not pus but granulation tissue, and which is probably caused by a micro-organism differing from the ordinary pus cocci (according to Del Monte by a protozoon). Horner has called attention to the analogy between chalazion and acne rosacea of the skin. In the latter sebaceous glands play the same part that the Meibomian glands do in chalazion.

Microscopic examination of a chalazion shows that first the epithelium of the acinus proliferates and an inflammatory infiltration is produced in the surrounding tissue of the tarsus. The latter process soon predominates, so that both gland acini and tarsus tissue ultimately are lost in the overgrowth of small cells (Fig. 200). This cell growth forms a soft tissue of the nature of granulation tissue, in which are found amorphous flakes representing remains of the inspissated contents of the acini; and it is inclosed in a connective-tissue capsule which is nothing but the connective-tissue in its vicinity, compressed and condensed by its growth. The granulation tissue also contains giant cells, which are probably of the same character as those which develop in a tissue in which epithelial structures or other, not directly absorbable foreign substances are imbedded. Finally, the central portions of the granulation tumor break down by a sort of mucilaginous softening, so that a cavity filled with a turbid liquid forms in the centre of the growth. Old chalazia, may thus be transformed into a cyst with thick walls and mucilaginous contents.

Chalazia which develop on the excretory duct of a Meibomian gland are situated near the free border of the lid, from which they project like a nipple, while on their posterior side they are flattened out by the counter-pressure of the eyeball.

[6] From χάλαζα, hail.

Sometimes a chalazion perforates the tarsus not behind but in front. Then the granulation tissue grows out into the subcutaneous cellular tissue and forms a soft flat tumor situated near the lid border and extending alongside of it. To this tumor (chalazion externum) the skin, which shows a reddish or bluish discoloration, is adherent. The tumor remains unchanged for months.

In specially bad cases of chalazia actual degeneration of the lids, especially the upper, takes place. The lids are thickened, so that they can hardly be everted. The skin of the lids forms nodular projections, but can be displaced on its bed and is not essentially altered. The conjunctival surface of the lids, on the contrary, appears uneven, nodular, and reddened, velvety in some spots, in others gray and translucent or perforated by sprouting granulations. In extreme cases of this sort we might at first be disposed to suspect tarsitis or a neoplasm. In operating upon such cases we see that the entire tarsus has disappeared in a spongy and partially softened granulation tissue.

Fig. 200.—Chalazion. Vertical Section through the Upper Lid. Magnified 4x1.

The chalazion has developed in about the middle of the highest point of the tarsus, and, as it projects farther forward than backward, it produces a crooking forward of the lid. The swelling consists of granulation tissue with giant cells, one of which is so large as to be actually recognizable with the naked eye in the section. It lies near the anterior wall of the larger one of the two cavities that have been produced by mucilaginous softening of the granulation tissue. The growth is surrounded by a thin capsule. Upon its anterior aspect it is covered by the skin of the lids, which is thinned, as can be seen from the way in which the cross section of the muscular bundles of the orbicularis is narrowed. The conjunctiva over the swelling, owing to papillary proliferation, is thicker and uneven. Above and below the swelling are unchanged acini of Meibomian glands.

Chalazion affects adults more frequently than children. Not infrequently several chalazia are found at once in the same patient and sometimes occur in crops, new chalazia developing at intervals of a week or so. A chalazion annoys the person who has it by the disfigurement it produces, and also by the condition of irritation which it keeps up in the eye. This condition of irritation is partly the result of the chronic inflammation of the lids, partly the result of the mechanical injury done to the eyeball by the uneven and bulging conjunctiva covering the inner surface of the tumor.

Treatment.—Quite small chalazia are best left alone. Larger chalazia are removed by an operation, in order to do away with the disfigurement and also with the irritation of the eye. (See § 921.)

435. Infarcts of the Meibomian Glands.—In elderly people we often see, upon everting the lids, small bright-yellow spots beneath the conjunctiva. These are the inspissated contents of the Meibomian glands which accumulate in their excretory ducts or their acini and distend them. These infarcts usually cause no trouble. But sometimes they are transformed by the deposition of lime salts into hard, stony masses (lithiasis conjunctivæ[7]). These bulge the conjunctiva forward and even perforate it with their sharp edges, which

7 From λίθος, a stone.

then cause mechanical injury to the eye. In this case they must be removed through an incision in the conjunctiva.

The *infarcts* of the Meibomian glands should not be confounded with the much more frequently occurring concretions that develop within new-formed glands in the conjunctiva of the tarsus. These also appear under the guise of yellow spots, but are more superficial (see § 7).

436. Affections of the Tarsus.—The tarsus is implicated not only in diseases of the Meibomian glands, but also in those of the conjunctiva. This is particularly true of trachoma and of amyloid degeneration of the conjunctiva. In the former we can often feel, when we evert the upper lid, that the tarsus has become thicker and more unshapely. This depends upon an inflammatory infiltration, which subsequently leads to atrophy and distortion through cicatricial contraction, and which must therefore be looked upon as the chief cause of trichiasis. In tarsi that have undergone this change, the Meibomian glands are also in great part found to be destroyed (see § 468). In amyloid degeneration of the conjunctiva the tarsus undergoes a like degeneration, and is transformed into a large, unshapely, friable structure (see § 494).

There is a primary affection of the tarsus (*tarsitis syphilitica*) which occurs as a consequence of syphilis. This develops very gradually, and as a rule without any notable pain. When it has reached its acme we find one or both lids of the same eye greatly enlarged and the skin of the lid tense and reddened. Upon palpation we can convince ourselves that the cause of the enlargement lies in the tarsus, which can be felt through the skin as a thick structure of cartilaginous hardness and ungainly form. The swelling of the tarsus is usually so great that the lid can no longer be everted. The enlarged tarsus consists, as is evident when it is cut into, of a lardaceous, bloodless tissue. The cilia upon the affected lid fall out, and the lymphatic gland in front of the ear upon the same side swells up. After the swelling has been for weeks maintained at the same height, it disappears very slowly again until the tarsus has reached its former volume, or has even in consequence of atrophy, fallen somewhat below it. It takes several months for the disease to run all through its course. Tarsitis makes its appearance in the third stage of syphilis, and is accordingly to be looked upon as a gummatous infiltration of the tarsus (cf. § 420).

IV. Anomalies of Position and Connection of the Lids

437. Trichiasis and Distichiasis.—Trichiasis [8] consists in a distortion of the cilia, which, instead of looking forward, are directed more or less backward so as to come into contact with the cornea. This anomaly of position either affects all the cilia or only those which jut out furthest back; it may also extend over the whole length of the border of the lid, or be present over a part of it only (total and partial trichiasis). The inverted cilia are seldom normal, being for the most part stunted, and consisting of short stumps or of minute, pale, and scarcely visible hairs.

Trichiasis causes a continual irritation of the eyeball, due to the action of the cilia; there are photophobia, lacrimation, and the feeling as if a foreign body was in the eye. The cornea itself suffers more considerable injury. Superficial opacities are produced in it, since the epithelium, in consequence of the constant irritation, undergoes a sort of callous thickening, and thus in a way protects the cornea against the effect of the external injury. In other cases deposits like pannus, or ulcers of the cornea are formed. It not infrequently happens that persons are tormented by frequent recurrences of corneal ulcers, until at length the physician discovers

[8] From θρίξ, hair.

one minute cilium which is directed against the cornea, and which has been the cause of the formation of the ulcers.

The most frequent *cause* of trichiasis is trachoma. The conjunctiva, which in the regressive stage of trachoma undergoes cicatricial shrinking and contracts, tends to draw the skin of the lid backward over the free border of the latter and thus displaces the cilia more and more. At first the most posterior cilia, afterward the anterior rows, too, are turned backward. The distortion of the tarsus acts to produce the same effect. Owing to this distortion, that portion of the tarsus which adjoins the free border of the lid bends off at an angle from the rest of the cartilage and is turned backward (t_1, Fig. 207 B), and in so doing it draws with it the covering of the free border of the lid which is firmly attached to it.

Partial trichiasis, in which only some of the cilia are turned backward, develops in consequence of scars left upon the free border of the lid or in the conjunctiva by blepharitis, hordeolum, diphtheria, burns, operations, etc.

Distichiasis.[9]—This is the condition in which, the lids being normal in shape, there are two rows of cilia, one of which looks forward, and the other, which is usually less developed, is situated directly at the posterior edge of the lid and is directed backward. This condition occurs as a rare congenital anomaly, and is sometimes present in all four lids. In this disease the normal Meibomian glands are wanting, being replaced by ordinary, irregularly distributed sebaceous glands.

The term distichiasis is employed by most authors not only for the condition in which there are congenitally two rows of cilia, but also the acquired anomaly of position due to trachoma, in which the posterior rows of cilia are set backward, while the anterior are still directed forward. But this condition is identical in its nature with trichiasis proper, and differs from it only in degree. At the commencement of the cicatricial contraction the rows of cilia are first drawn apart; then the posterior, and last of all the anterior row are turned backward. Accordingly, distichiasis is developed first and trichiasis afterward, and a sharp line of distinction cannot be drawn between the two. It is hence better to use the expression trichiasis for this condition in all its phases, and to confine the term distichiasis to the congenital cases in which two regular series of cilia are present. Then trichiasis and distichiasis really denote two perfectly distinct conditions.

Treatment.—When only a few of the cilia have an improper position they can be removed by epilation. Inasmuch as they grow again, epilation must be repeated at intervals of a few weeks—a thing which can very often be attended to by the patient himself or his relatives. It is still better to employ a method in which, with the performance of epilation, the follicle of the cilium is at the same time destroyed, so that the cilium does not grow again. The best procedure for this purpose is electrolysis (see § 926). When quite a large number of the cilia or all of them are directed toward the eyeball, epilation is not suitable; in that case these methods are indicated by means of which the cilia are brought to their proper position by a shifting of the place in which the hair bulbs are implanted (see §§ 927-929).

438. Entropion.—Entropion[10] is a rolling inward of the lid. Trichiasis affects simply the lid border, of which both the shape and the position are faulty. In entropion the shape of the lid border may be normal, but the

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[9] From δίς, double, and στίχος, a row.
[10] From ἐν, in, and τρέπειν, to turn.

lid border as a whole is revolved back so as not to be visible at all when the open eye is seen from in front. Hence, to get a view of the border of the lid we have to roll the lid out again by pulling it toward the margin of the orbit. However, the dividing line between trichiasis and entropion is not a sharp one, and the evil consequences of the two are the same.

According to their etiology we distinguish the two following varieties of entropion:

(a) *Entropion Spasticum* is that form which is produced by the contraction of the orbicularis. The fibres of the palpebral portion of the orbicularis (see § 3) describe arcs having a curvature in two different directions. One sort of curvature is due to the fact that the muscular fibres encircle the palpebral fissure; the concavity of these arcs accordingly looks toward the palpebral fissure and is directed downward in the upper lid, upward in the lower. The second variety of curvature is caused by the fact that the muscular fibres in conjunction with the lids are molded to the anterior convex surface of the eyeball; the concavity of these arcs looks backward in both lids. When the fibres of the orbicularis contract, they tend to shorten from the form of an arc to that of its chord. In so doing they exert a double action: by the flattening out of the first set of curves they narrow the palpebral fissure; by the flattening out of the second set they press the lids against the surface of the eyeball. Either component may lead to inversion of the tarsus, if the character of the subjacent structures is such as to give rise to this condition.

By the action of the orbicularis in the sense of the first component, the lids are forcibly apposed to each other by their narrow edges when the palpebral fissure is being closed. We may conceive of the two tarsi as represented by two visiting cards, standing one over the other in the same vertical plane, so that the lower edge of the upper rests upon the upper edge of the lower, and the edges of the two are forcibly apposed. Then slight pressure with the finger upon the line of contact of the two edges suffices to produce inversion of the cards in a sense opposite to that in which the pressure is made. In like fashion the apposed tarsi bend forward or backward, according as the subjacent structures either tend to press them forward or, on the other hand, fail to afford them a sufficiently firm support. Much more important, however, for the production of a change of position in the lids is the second component, in accordance with which the fibres of the orbicularis press the lid against the eyeball; for the lids are in perfect contact with the eyeball only so long as the latter furnishes them a uniform bed to lie upon. When then, owing to lack of uniformity in the bed, either the free or the attached border of the tarsus receives insufficient support posteriorly, a bending of the tarsus results in the sense either of an entropion or an ectropion.

The stronger the contraction of the fibres of the orbicularis in general, the more readily will the mechanical causes mentioned lead to an alteration in the position of the lids. And from what has been said it is easy to understand that there is a spastic ectropion as well as entropion; it depending upon the mechanical conditions above described and also upon other circumstances (especially upon the character of the skin of the lids) whether blepharospasm causes the lids to bend forward or backward.

For an entropion to develop, two conditions are necessary: Deficient support of the free border of the lid, and an abundant amount of extensible skin upon the lid. The former condition obtains when the eyeball is wanting; entropion spasticum, therefore, develops above all when the orbit is empty. For entropion to develop, however it is not necessary for the eye-

ball to be entirely absent; it is sufficient that it should be diminished in size, or should simply be situated more deeply in the orbit, as it is in the old and lean. Again, when the border of the lid is rolled inward, the skin of the lid is drawn after it. If this is prevented an entropion cannot develop. If the entropionized lid is put back in place, and then the skin of the lid is drawn a little up toward the margin of the orbit and fixed there by pressure, the rolling in again of the lid is rendered impossible. Entropion spasticum, therefore, does not usually, develop in persons having firm and elastic skin upon the lids, but requires the presence of a large amount of wrinkly, readily displaceable skin, such as we meet with in old persons.

It is clear that the rolling in of the lids is favored when the fibres of the orbicularis are very forcibly contracted, as in blepharospasm, and also when the pressure with which the muscular bundles of the orbicularis are squeezed against the border of the lid is aggravated by an external pressure—e. g., by a bandage. Similarly blepharophimosis acts to favor the development of entropion, since it draws the skin toward the free border of the lid.

If we summarize what has just been said, it follows that entropion spasticum develops mainly in elderly people with flabby lids, and that its production is favored by deep placing, diminution in size, or absence of the eyeball, by the existence of a blepharospasm or a blepharophimosis, and by the wearing of a bandage. For the last-named reason entropion is a frequent and unpleasant complication during the after-treatment of cataract operations where the patients are elderly people whose eyes have to be kept bandaged for quite a long time.

Entropion spasticum is almost invariably restricted to the lower lid.

(*b*) *Entropion Cicatriceum* is caused by the cicatricial contraction of the conjunctiva by means of which the free border of the lid is drawn inward. It may be said to form one step further on the way upon which trichiasis has started. Like the latter, this condition is observed after trachoma, diphtheria, pemphigus, burns of the conjunctiva, etc.

Treatment.—In entropion spasticum which has formed beneath a bandage, the discontinuance of the latter is often all that is required. If we are compelled by important reasons to continue the bandage, we place upon the lower lid in the neighborhood of the margin of the orbit a roll of adhesive plaster, which is kept pressed against the lid by the bandage. This procedure depends upon the observation that the entropionized lower lid takes a correct position spontaneously when we press back with our fingers that portion of it which lies next to the margin of the orbit. Another method of exerting a constant pressure of this sort consists in fastening a narrow strip of a good sticky adhesive plaster to the lid throughout its entire length; the strip being placed about in line with the lower margin of the orbit and being drawn quite tight. We may also evert the lid and try to hold it in place with collodion.

If entropion is due to absence of the eyeball, we enjoin the wearing of an artificial eye.

If we cannot attain our object by bloodless measures, we must try an operation (see §§ 927–929).

439. Ectropion.—Ectropion consists in the revolution of the lid outward, so that its conjunctival surface looks forward. It is, therefore, the opposite of entropion. There are different degrees of ectropion. The lowest degree is that in which the internal margin of the lid stands off a little from the eyeball (*eversion* of the border of the lid). Even this very slight degree, however, bears within itself the conditions for its own increase. With the eversion of the border of the lid there is also associated eversion of the puncta, in consequence of which epiphora develops, and by this a contraction of the skin of the lower lid and hence an increase of the ectropion are produced. Ectropion may present all degrees up to complete eversion of the entire lid. The consequences of ectropion are epiphora and also redness and thickening of the conjunctiva wherever it is exposed to the air. The hypertrophy of the conjunctiva may reach such a high degree, especially if this membrane has already been considerably altered (by gonorrhœal conjunctivitis or by trachoma), that the conjunctiva looks like exuberant "proud" flesh (wound granulations). In marked ectropion the cornea is incompletely covered by the lids, so that keratitis e lagophthalmo is set up.

According to differences in their etiology the following kinds of ectropion are distinguished:

(*a*) *Ectropion Spasticum.* It was shown, in speaking of entropion, that the lids can be bent by blepharospasm, and that the direction in which they are bent depends upon the mechanical relations of the individual parts. In ectropion these conditions are the direct opposite of those which we have found to be the causes of entropion. They consist partly in the displacement forward of the tarsal portion of the lid, and partly in a firm, elastic character of the skin of the lid, by virtue of which the palpebral border is drawn toward the margin of the orbit. We often have the opportunity to observe the effect of a traction of this sort when we try to open the palpebral fissure in a child with swollen lids and with blepharospasm. As soon as we draw the lids apart they become spontaneously everted, and in such cases if we should not carefully put the lids back in place we might readily set up a permanent spastic ectropion; for the peripheral bundles of the palpebral portion of the muscle contract spasmodically behind the everted tarsal portions of the lid, and maintain them in their faulty position. Then the everted lids swell up, because they become congested and this swelling renders their reposition the more difficult the longer the condition lasts. Inasmuch as a certain degree of tension of the skin of the lids is requisite for the development of spastic ectropion, this condition is found principally in children and young persons.

The second condition mentioned above for the development of ectropion is the forcing of the edge of the lid away from the eyeball so that eversion of the tarsus is facilitated. It occurs mostly as a result of thickening of the conjunctiva, particularly when due to gonorrhœal conjunctivitis and trachoma. However, the eyeball itself may force the lids so far forward that they become ectropionized, as is the case in enlargement or protrusion of the eye.

The two predisposing causes above mentioned will be the more likely to induce eversion of the lid, the greater the blepharospasm present.

From what has been said, it follows that ectropion spasticum is particularly apt to occur in young persons who at the same time suffer from inflammation of the conjunctiva with swelling of the latter and with coincident blepharospasm. Ectropion spasticum frequently affects the upper and lower lids simultaneously.

(*b*) *Ectropion Paralyticum* arises as a result of paralysis of the orbicularis. The lids are then no longer kept pressed against the eyeball by the contraction of the fibres of the orbicularis, and thus the lower lid sinks down of its own weight. For this reason ectropion paralyticum is found only in the lower lid; the upper lid, because of its weight, remaining applied to the eyeball even when there is no action of the muscle. In conjunction with drooping of the lower lid there is inability to lift it. Consequently the palpebral fissure cannot be perfectly shut (lagophthalmus).

(*c*) *Ectropion Senile* also is found only in the lower lid (Fig. 202). The way it develops is that in old people the lower lid is relaxed in all its parts and is pressed with insufficient force against the eyeball by the enfeebled fibres of the orbicularis. Another factor that here comes into play is the chronic catarrh of the conjunctiva (catarrhus senilis), which is so frequent in old people.

Likewise due to enfeeblement of the action of the orbicularis is the ectropion of the lower lid, that occurs when the lid has been divided in a vertical direction at any spot, or when the external commissure has been destroyed, so that continuity of the orbicularis is interrupted somewhere.

(*d*) *Ectropion Cicatriceum* develops when some part of the skin of the lid has been destroyed and has been replaced by scars so that the lid is contracted. Injuries (particularly burns), ulcers, gangrene, excision of the skin in operations, etc., may give rise to it. Ectropion frequently develops as a sequel to caries of the orbit in scrofulous children (Fig. 249). Here, in addition to the contraction of the skin of the lid, its fixation to the osseous cicatrix upon the margin of the orbit is of moment, and with this there is associated marked retraction of the skin. So also ectropion is set up by the contraction of the skin and the loss of its elasticity that result from the eczema which occurs in connection with the continual wetting of the skin of the lids by the tears or from various other causes. Ectropion is therefore frequently found in conjunction with a blepharitis of long standing and with disorders of the conjunctiva and the lacrimal sac.

Non-operative *treatment* is successful mainly in ectropion spasticum. It consists in putting the lid back in place and keeping it pressed against the eyeball by a well-fitting bandage. In ectropion paralyticum we must employ, besides the bandage, the remedies indicated for the cure of facial paralysis, particularly electricity. Ectropion senile is curable without an operation only as long as it has not reached any very high degree. For quite a long time the eye must be bandaged at night, and the patient must be told, when wiping away the tears that flow down over his cheek, to apply his handkerchief from below upward, and not, as is ordinarily done, from

above downward, a procedure by which the lid is drawn down only so much the more. It is, furthermore, advisable to slit up the inferior canaliculus in order to diminish the epiphora due to the eversion of the punctum. The higher degrees of ectropion, and particularly ectropion cicatriceum, require treatment by operations of one kind or another (see §§ 930–933).

440. Ankyloblepharon and Symblepharon.—Ankyloblepharon[11] consists in an adhesion of the upper to the lower lid along the palpebral margin (Fig. 201). It is either partial or total, and is very often combined with an adhesion between the lid and the eyeball, or *symblepharon*. It also has a common etiology with the latter; i. e., it occurs when, as a result of burns, ulcers, etc., the borders of the two lids are converted into raw surfaces at opposed points and so become adherent. (For symblepharon see § 505.)

Through ankyloblepharon the palpebral fissure is diminished in size and the movements of the lids are hindered; in total ankyloblepharon there is complete occlusion of the palpebral fissure. The treatment, when we are dealing with simple ankyloblepharon without coincident symblepharon, consists in separating the adherent lids by an operation. If the adhesion extends as far as the angle of the lid, the latter must be supplied with a lining of conjunctiva, as otherwise the adhesion would form again, starting from the angle. When symblepharon is present along with the ankyloblepharon, it depends mainly upon the extent of the former whether an operation is practicable at all or not.

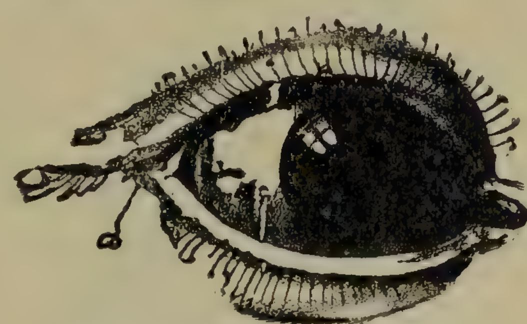

FIG. 201.—ANKYLOBLEPHARON.

This occurred as the result of an injury from a splinter of glass. After the injury, the patient without calling in any medical aid kept the eye bandaged for some time. At *a* the margin of the upper lid is adherent to the margin of the lower, and over an area corresponding to this adhesion the cilia are absent. To the temporal side of the site of adhesion, a small portion of the palpebral fissure has been preserved as far as the external angle of the eye. Hence in this case simple division of the bridge would suffice to remove the ankyloblepharon.

441. Blepharophimosis.—In blepharophimosis[12] the palpebral fissure appears to be contracted at the external angle of the eye (Fig. 202). Upon drawing the lids apart we see that the contraction is produced by a fold of skin which extends in a vertical direction at the external angle of the eye and juts out in front of it like a sliding screen. If we draw the fold of skin outward, we disclose behind it the normally formed external palpebral angle with the delicate ligament uniting the borders of the two lids. The distinction between ankyloblepharon and blepharophimosis, two conditions which are commonly confounded, is therefore as follows: In ankyloblepharon the borders of the lids are adherent to each other, but in blepharophimosis they are normal, and the contraction of the palpebral fissure is only apparent, being caused by the drawing of a fold of skin over its outer extremity.

Blepharophimosis is most frequently found in persons who suffer from epiphora and blepharospasm of long standing—that is, it is especially met with in chronic inflammations of the conjunctiva. It originates in a contraction of the skin of the lids due to their being frequently wet with the

[11] From ἀγκύλη, a stiff limb (or a thong), and βλέφαρον, eyelid.

[12] From βλέφαρον, lid, and φίμωσις, contraction, from φιμός, a muzzle.

tears or with secretion. If this contraction is particularly marked in a horizontal direction, the skin from the adjacent part is drawn up so as to project over the palpebral fissure on the temporal side like a sliding screen. This effect is re-enforced by the action of the fibres of the orbicularis, which in blepharospasm draws the skin on the outer side of the face in toward the external angle of the eye. We can artificially imitate blepharophimosis by pushing the skin from the temple over the palpebral fissure with our fingers; and conversely can make an existing blepharophimosis disappear by drawing the skin out toward the temple. Blepharophimosis is ordinarily not found at the inner angle of the eye, because the adjoining skin of the bridge of the nose is not so readily displaced, although in old persons with thin skins a projecting fold is sometimes formed here too.

FIG. 202.—BLEPHAROPHIMOSIS AND ECTROPION SENILE.

The ectropion as is usually the case, is more marked in the nasal than in the temporal portion of the lid, because in the former the contact of the lower lid with the upper is interrupted by the horseshoe-shaped notch at the inner angle of the eye. Moreover in the present case the blepharophimosis counteracts the drooping of the lid at the outer angle.

Blepharophimosis accordingly, like that form of ectropion which occurs in connection with chronic catarrh, epiphora, etc., owes its origin to a contraction of the skin of the lid. The difference between the two lies in the fact that, in the first case, the contraction makes itself apparent chiefly in the horizontal direction; in the second case, in the vertical direction. Blepharophimosis and ectropion, therefore, as originating from the same cause, may be both present at the same time (Fig. 202). That this in general is but rarely the case is due to the upward traction which the vertical fold of the skin forming the blepharophimosis exerts upon the lower lid and which opposes the eversion of the latter. For this reason, indeed, blepharophimosis really favors the development of an entropion, which in such cases can often be cured simply by the abolition of the blepharophimosis. Another consequence of blepharophimosis is the contraction—to be sure, an apparent one only—of the palpebral fissure, which consequently cannot be opened as wide as usual.

Blepharophimosis, at least in young persons with elastic skin, may gradually disappear of itself, provided its causes (epiphora, blepharospasm) have ceased to act. If it does not appear spontaneously and if it causes any trouble, it may be removed by canthoplasty (see § 923).

442. Lagophthalmus.—By lagophthalmus[13] is meant an incomplete closure of the palpebral fissure when the attempt is made to shut the lids together. In the lesser degrees of lagophthalmus complete closure of the palpebral fissure is still possible by squeezing the lids tight; but since during sleep there is no such squeezing of the lids, but only a gentle closure of them, such patients sleep with their eyes open, and from this the disease derives its name. In the higher degrees of lagophthalmus it is no longer possible for the patients to bring the lids into contact even by forcible squeezing.

[13] I. e., hare's eye, from λαγώς, hare, because it was believed that hares sleep with their eyes open.

The evil consequences of lagophthalmus depend upon the harm which the eyeball suffers from being insufficiently covered. What part of the eyeball is it that remains uncovered by the lids in lagophthalmus? If we tell a patient with a slight lagophthalmus to shut his lids tightly together, we see that the borders of the lids remain separated some millimetres from each other, and that between them there lies the part of the sclera that is below the cornea, but not the cornea itself. The reason for this is that at the same time that the lids are shut the eye is turned upward, so that the cornea is concealed beneath the upper lid (§ 66). The same is the case during sleep. Hence the only part of the conjunctiva scleræ that is constantly exposed to the air is that situated below the cornea. As a result of this exposure it is injected, and the patient suffers from the symptoms of a chronic conjunctival catarrh. In the higher degrees of lagophthalmus the cornea, too, is seen to lie in the slit which remains open when the lids are closed together; and, because the cornea is turned upward, it is the lower part of it that is found there. Lagophthalmus but seldom reaches a degree such that the cornea remains completely uncovered.

The cornea may suffer in two ways from being covered insufficiently; either its surface dries up wherever it is constantly exposed to the air, and keratitis e lagophthalmo ensues (see § 558 and Fig. 249), or the cornea protects itself against the exposure by a change in its epithelium which becomes thicker and epidermoid, so that the deeper layers of the cornea are preserved from desiccation (xerosis of the cornea, see § 507). But as opacity both of the epithelium and of the cornea itself is associated with this process, the sight is thereby prejudiced. In any case, therefore, vision is endangered in lagophthalmus if the latter is so considerable that the cornea is no longer sufficiently covered. Another result of lagophthalmus is epiphora, since complete closure of the lids is requisite for the normal conduction of tears into the nose.

The causes of lagophthalmus are: 1. Narrowing of the lids. This is in most cases caused by the loss of a portion of the skin of the lids in consequence of burns, ulcers (particularly lupus), operations, etc. Less frequent are the cases of congenital narrowness of the lids. These are characterized by the fact that the palpebral fissure still remains open a distance of some millimetres when the lids are shut tightly together, and that, nevertheless, no signs whatever are present of loss of the skin of the lids under the form of scars. In these cases the symptoms of a chronic blepharitis ulcerosa are often present. 2. Ectropion. 3. Paralysis of the orbicularis. 4. Constant patency of the eyes, occurring in persons who are very ill or who are unconscious, and due to a reduction of the sensitiveness of the cornea, so that the reflex acts of winking and of shutting the eyes are no longer initiated. 5. Enlargement or protrusion of the eye, so that the lids, in spite of being normal in size and mobility, are unable to cover it completely. Under this head should be specially mentioned exophthalmic goitre, in which the lagophthalmus is bilateral, so that bilateral blindness sometimes occurs from it.

The *treatment* of lagophthalmus consists first of all in regarding the

causal indication—i. e., in the removal of those conditions which prevent the complete closure of the lids. Under this head belong the remedying of contraction of the lids by blepharoplasty, the cure of ectropion, the treatment of facial paralysis, etc. Until we have succeeded in getting rid of the lagophthalmus, for which often quite a long time is required, the eye must be protected from its ill effects. This is accomplished by closing the palpebral fissure artificially with a bandage. For this purpose we first bring the borders of the lids into perfect apposition, and keep them so by strips of sticking plaster which are attached vertically across the lids; over this is applied an ordinary protective bandage with dry cotton. In the lighter cases it is sufficient to apply this bandage at night, since then the danger of desiccation of the cornea is the greatest, and during the day the act of winking suffices to keep the cornea moist. But in the higher degrees of lagophthalmus, or in cases in which the cornea is already attacked, the bandage must be worn constantly. When the lagophthalmus is so great that the margins of the lids no longer come near touching and cannot be kept in contact by a bandage, we may give up the attempt to cover the cornea and simply keep it from drying by making a moist chamber. For this purpose we cover the eye with a watch-glass, the edges of which are fastened down tight by plaster to the circumference of the orbit. At some point in the concavity of the watch-glass is placed a little cotton wet with water. Because of the warmth of the eye the water evaporates, so that the air between the watch-glass and the eye is kept constantly moist.

In those cases in which the removal of the cause of the lagophthalmus is impossible, or is likely to require a very long time (as, for example, in a case of exophthalmic goitre), it would be disagreeable for the patient to have to wear a bandage for so long a time—a year or so. For these cases tarsorrhaphy (see § 924) is advisable. By this the palpebral fissure is abbreviated and the borders of the lids are brought nearer each other, so that the closure of the eye is facilitated.

For ptosis and other disorders of the lid muscles, see §§ 360 et seq.

V. Injuries of the Lids

443. Injuries of the lids of all kinds, including simple contusions, incised, lacerated, and contused wounds, burns by heat or caustics, etc., are very frequent. A peculiarity of these injuries that needs to be emphasized is that, because of the great elasticity of the skin of the lids and their loose attachments to the subjacent parts, both ecchymosis and œdema in the injured lids are usually much more considerable than after a similar injury in other parts of the body. Accordingly, we must not allow ourselves to be frightened merely by the great swelling and bluish-black discoloration of the lids, as these appearances are often enough produced by comparatively slight contusions. On the contrary, the diagnosis and prognosis should not be pronounced until after a careful examination. In this, three points are chiefly to be considered: Solutions of continuity of the skin of the lids, injury to the subjacent bones, and injury of the eyeball.

Solutions of continuity of the skin of the lids present a varying aspect

according to their direction. Those which run horizontally—i. e., parallel to the line of fibres of the orbicularis—gape but little, so that the lips of the wound often lie in apposition spontaneously. But if the cut or rent runs in a direction perpendicular to the fibres of the orbicularis, the wound gapes widely in consequence of the retraction of the divided bundles of the muscle. Consequently, the cicatrices after horizontal wounds of the skin of the lids are scarcely visible, while those after vertical incisions are conspicuous and disfiguring. Hence, in operations upon the lids the rule is laid down that all incisions should, wherever possible, be made parallel to the course of the fibres of the orbicularis. The worst wounds are those which sever the lid in a vertical direction through its entire thickness. If these do not unite by first intention, there remains an indentation of the border of the lid, or even a deep triangular incision in it (coloboma palpebræ traumaticum). By this the complete closure of the lid is rendered impossible, so that, in addition to the disfigurement, a permanent epiphora results.

The presence of an *injury of the subjacent bone* is determined by palpating the margin of the orbit with the finger through the swollen lid. A fracture of the orbital margin is manifested by unevenness and special sensitiveness at some spot, symptoms to which in many cases is added distinct crepitation. A certain sign of injury of the bone is *emphysema of the lids.* This consists in the entrance of air into the cellular tissue beneath the skin of the lid. The lids then have a peculiar soft feeling like a feather bed, and at the same time we get in the palpating finger a sense of crepitation due to the displacement of bubbles of air beneath the pressure of the finger. The air comes from the cavities surrounding the orbit—most often from the ethmoid cells. Its presence in the subcutaneous cellular tissue of the lids hence presupposes some abnormal communication between the latter and these cavities, such as can have originated only through a fracture of the bone. When then, by blowing the nose, straining, or coughing, the air in the nose and its accessory cavities is put under greater pressure, it is forced into the subcutaneous cellular tissue, and emphysema is produced (§ 876).

The mutilation of the lids due to injury may cause danger to the *eye* itself. Through cicatricial contraction of the lids, or the formation of fissures in them, lagophthalmus and consequently inflammation of the cornea may be set up.

The *treatment* of injuries of the lids is carried on according to general surgical rules. In simple ecchymosis we apply cold compresses with lead water. In emphysema of the lids the air contained in the tissues usually undergoes resorption without causing any ill results. To accelerate absorption a compressing bandage is indicated; at the same time the patient must avoid straining, blowing the nose, etc., in order not to drive fresh charges of air into the tissue. Recent wounds, the edges of which are not too greatly contused, are best united at once by sutures. In wounds the edges of which are destroyed by contusion and the like, we apply an antiseptic dressing and wait for the necrotic portions to be cast off. The same rule holds good for burns and injuries caused by caustic substances. After the elimination of those portions of the skin that have been destroyed, granulating raw

surfaces are presented to view which cicatrize over and thus lead to a contraction of the lids. To combat this, we use the measures laid down for the treatment of destruction of the skin by inflammation (§ 417).

444. Ecchymoses of the Lids.—*Ecchymoses* are usually pretty sharply limited at a line corresponding to the margin of the orbit, because the skin is attached to this by rigid connective tissue which prevents the further advance of the blood. On the other hand, the blood not infrequently travels beneath the skin of the dorsum of the nose over to the other side. An ecchymosis is then found in the lids of the other eye also. As the skin upon the dorsum of the nose is thick, it frequently does not permit the blood to be seen through it, so that we do not notice the bridge connecting the ecchymosis of one eye with that of the other. We might then easily be induced to believe that the injury had affected the other eye also, a thing which however, can be excluded with certainty in many cases—e. g., when suggillation of the lids of one side sets in after enucleation of the other eye.

A similar migration of extravasated blood is observed in *fractures of the base of the skull.* The blood oozes forward from the site of the fracture and travels along the floor of the orbit. It then makes its appearance some time after the injury under the form of an ecchymosis in the lower part of the conjunctiva of the eyeball, and also on the lower lid close to the margin of the orbit, particularly in the region of the inner angle of the eye. This symptom, although it is not present in all cases, is of great importance for the diagnosis of fractures of the base.

Spontaneous ecchymoses sometimes occur in the lids, in the same way as they do in the conjunctiva, from violent straining, excessive coughing, and the like; also in infantile scurvy (Stephenson).

Extravasations of blood into the lids, instead of disappearing by resorption, may go on to *suppuration,* so that an abscess of the lids develops. This is particularly to be apprehended when there is at the same time a solution of continuity of the skin of the lids, through which infectious germs may penetrate into their tissue.

VI. Tumors of the Lids

445. Benign Tumors.—*Xanthelasma*[14] is a flat tumor of a dirty sulphur-yellow color and projecting but little above the skin of the lid. It is found most frequently on the upper and lower lids in the neighborhood of the inner angle of the eye. In this situation the tumors are often located symmetrically on the two sides of the eye, like the yellow spots above the eyes of the dachshund. Xanthelasmata occur in elderly persons, particularly of the female sex. They grow very slowly, and have no bad results besides the disfigurement they cause, which, moreover, affords the only reason for their removal by operation, as is sometimes done.

Molluscum contagiosum is a small, rounded tumor, the surface of which is somewhat flattened and has an umbilicated depression in its centre. From this a substance resembling sebum is discharged upon pressure. This form of molluscum is contagious.

Molluscum simplex (fibrous molluscum) is a tumor of the skin which is attached by a pedicle to the skin of the lids and hangs down like a pouch.

Warts and cutaneous horns are also observed on the lids.

Among *cysts,* milia, atheromata, and dermoid cysts occur. The latter, which may attain pretty large dimensions, will receive a more detailed description under the head of affections of the orbit (§ 879). On the borders

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[14] From ξανθός, yellow, and ἔλασμα, plate. It is also called xanthoma.

of the lids small, transparent cysts are frequently met with (Fig. 228), which have developed from occluded sweat glands in the border of the lid (glands of Moll).

The *vascular tumors* (angiomata) are found in the lids under the two forms of telangiectases and of tumores cavernosi. The former are bright-red spots situated in the skin of the lid itself, and are composed of dilated and tortuous blood-vessels. The latter lie beneath the skin of the lid, which they bulge forward and through which they can be seen shining with a bluish lustre. They consist of closely aggregated, large, venous cavities, which can be felt and compressed through the skin; the arteries running to the tumors are dilated. Vascular tumors are usually congenital, but they develop still more extensively after birth, and sometimes attain such a size that they cover a great part of the face, and are also continued backward into the conjunctiva and the tissues of the orbit. Hence they should be removed as early as possible. In doing this our principal care must be to destroy the skin of the lids over as small an area as possible, as otherwise we might get shrinking of the skin, with ectropion and lagophthalmus. If the angioma is so small that we can remove it without sacrificing too much skin, it is best to extirpate it (§ 922); or we can destroy such angiomata with fuming nitric acid or with the thermo-cautery or the galvano-cautery. In very superficial angiomata carbon-dioxide snow may be applied; but treatment with radium or x-ray seems to give the best results and leaves very little scar. In the case of large angiomata radium or electrolysis (§ 922) may be used with advantage.

Other tumors which occur very rarely in the lids are lymphomata, fibromata, enchondromata, myxomata, lipomata, cavernous lymphangiomata, plexiform neurofibromata (§ 836), and adenomata of the sebaceous glands, Meibomian glands, sweat glands, and glands of Krause and of Moll.

446. Malignant Tumors.—Carcinomata occurring in the lids are, as a rule, epitheliomata, which start from the skin of the lid, particularly from that of the border. Subsequently they pass over upon the eyeball and even penetrate into the depth of the orbit. *Sarcomata* develop from the connective-tissue portion of the lids. The pigmented melano-sarcomata most often develop from a congenital nævus of the skin or conjunctiva. With malignant tumors we find an enlargement of the neighboring lymphatic glands, occurring first in the gland in front of the ear, afterward in the glands along the lower jaw and in the neck.

Epitheliomata, particularly basal-celled epitheliomata, not spreading rapidly and not involving the bone can be completely cured by x-ray or radium in large, rapidly repeated doses (New and Benedict, Janeway). Squamous-celled epitheliomata and melanotic sarcomata are not so amenable to this treatment, and require extirpation (§ 922).

The *flat cancers* of the skin that not infrequently occur upon the lids require particular mention. In these cases there is found a shallow ulcer with an uneven floor and irregular rather hard walls. The infiltration of the walls of the ulcer is the only characteristic sign, for there is no tumor in the proper sense of the word, and for this reason

inexperienced observers readily mistake the true nature of the lesion, which is nothing else than an epithelial carcinoma. The ulcer advances in one direction while it cicatrizes on the side opposite, for which reason it has been called by the name of *ulcus rodens.* Its progress, however, is extremely slow, so that such tumors often last many years before attaining any great size.

Glandular carcinoma is also occasionally found in the lids.

Xeroderma pigmentosum is a condition developing in early childhood, in which pigmented spots resembling freckles form on the lids. These later become more deeply pigmented, take on a warty look and then ulcerate. They may cause deformations of the lid and keratitis, and tend to undergo malignant degeneration so that ultimately they cause death.

Blastomycetic dermatitis (§ 421) may simulate a malignant tumor of the lids.

VII. Congenital Anomalies of the Lids

447. Under the name of *epicanthus* is denoted a fold of skin which juts out on both sides of the dorsum of the nose and projects over the inner angle of

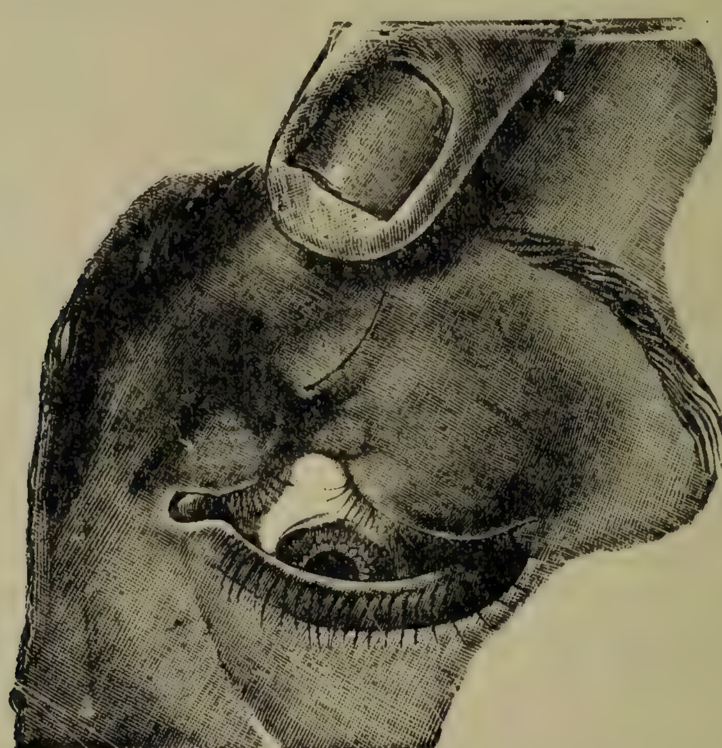

Fig. 203.—Congenital Coloboma of the Upper Lid.

the eye so as partly to cover it. In the Mongolian race a moderate degree of epicanthus is the rule, and produces the characteristic appearance of the palpebral fissure in these people. In the Caucasian race, not infrequently, there is observed in children a slight degree of epicanthus, which disappears again when in the process of growth, the dorsum of the nose becomes more prominent. Higher degrees of epicanthus, which persist all through life, must be regarded as a malformation, and are sometimes found in connection with other congenital defects (e. g., ptosis). The fold of skin forming the epicanthus disappears if with our fingers we pick up the skin upon the dorsum of the nose into a vertical fold and thus shorten it horizontally. Upon this observation depends the operation for epicanthus recommended by Ammon, which consists in the excision of an elliptical piece of skin upon the dorsum of the nose. We may also excise the projecting fold of skin itself.

In the rare *epiblepharon* a similar fold stretches along the border of the lower lid, so as to cover the greater part of the latter and press the lashes against the eyeball.

A *coloboma* of the lids is a fissure having the shape of a triangle with its base at the border of the lids. Congenital coloboma (Fig. 203) is rather rare and is usually found in the upper lid—sometimes in conjunction with a dermoid tumor of the cornea (§ 511) or with remains of the amnion attached to the skin near by. A gap resembling congenital coloboma may be produced by injury (coloboma traumaticum).

Complete absence of the lids and the palpebral fissure (*ablepharia*) occurs in *cryptophthalmus*, in which the skin passes unbroken over a rudimentary eyeball.

In *anophthalmus* (§ 769) the lids are extremely small and the palpebral fissure, although of fair size, originally, is reduced by adhesion of its borders (ankyloblepharon, § 440) to a minute chink. Another form of congenital anklyloblepharon, occasionally found, is that in which a fine filiform bridge connects two lids that are otherwise normal (*ankyloblepharon filiforme adnatum*).

In simple microphthalmus the palpebral fissure is shorter and the lids, which in other regards are of normal shape, are correspondingly smaller (*microblepharia*), In high degrees of microphthalmus with cyst formation the lower lid is so protruded by the subjacent cyst that the rudimentary eyeball is scarcely visible.

An anomaly of peculiar sort is the *congenital shortness* of the lids. A normal upper lid can be stretched a good deal in a vertical direction. A lid, congenitally short, can scarcely be stretched at all, and there is a slight degree of lagophthalmus, especially in sleep, which produces epiphora and an obstinate blepharitis, but rarely any more serious troubles.

For congenital distichiasis see § 437, and for congenital ptosis see § 362.

CHAPTER XX

DISEASES OF THE CONJUNCTIVA

I. HYPERÆMIA OF THE CONJUNCTIVA; CONJUNCTIVITIS

448. Hyperæmia.[1]—The conjunctiva more than any other mucous membrane, is exposed to exterior influences, and hence in every man in the course of his life hyperæmias occur in the conjunctiva which may ultimately set up permanent changes in the membrane.

The same influences probably accounts for the development of papillæ and adenoid structures—a development that takes place in very varying amount in different persons. Repeated irritation of this sort also causes the multiplication of plasma cells and nodules composed of lymphocytes, as well as of Henle's glands and the similar tubular glands of the retrotarsal fold. For this reason in the conjunctiva more than anywhere else the line between the normal and pathological state is hard to draw.

The effect of exterior injuries is most clearly seen in the formation of the pinguecula (§ 9).

The injection of the conjunctiva that occurs so frequently in inflammations of the eyes varies greatly in situation and degree.

In the *conjunctiva of the tarsus* we distinguish two sorts of injection. In the less intense, *reticular* injection the separate vessels are still distinguishable and the Meibomian glands are visible through the reddened conjunctiva. Reticular injection is the expression of a simple hyperæmia unassociated with changes in the conjunctival tissues. In the more intense *diffuse* injection the conjunctiva has a uniformly red color and the Meibomian glands are no longer visible. The conjunctiva in this case is always thickened and generally shows a papillary hypertrophy.

In the conjunctiva of the eyeball we have mainly to consider the situation of the injection. Since this area is supplied by vessels from two sources there are two kinds of injection. Injection of the *posterior* conjunctival vessels occurs in those varieties of inflammation which especially affect the retrotarsal fold and is marked by the fact that it diminishes from the periphery toward the cornea. Injection of the *anterior* conjunctival vessels occurs in inflammations located in the limbus and the conjunctiva of the cornea. It is often combined with injection of the marginal network of vessels, is most intense at the margin of the cornea, and diminishes toward the periphery. In both of these forms of injection (*conjunctival* injection) the separate vessels are very clearly visible as vividly red, tortuous lines, which branch in arborescent fashion or unite in a network and which can be pushed back and forth with the conjunctiva.

Since the system of anterior conjunctival vessels is supplied by the anterior ciliary arteries, injection of this system is always associated with what is called *ciliary injection*, i. e., a diffuse rose-red or violet coloration of

[1] This section is from Salzmann.

the sclera about the cornea. When of slight degree this injection is 3 or 4 mm. broad and fades off quite gradually toward the periphery. When it is more marked it extends further and at its periphery forms a network of indistinctly visible violet-red vessels which cannot be displaced with the conjunctiva. Ciliary injection is not seated in the conjunctiva, but consists in a hyperæmia of the episcleral network of capillaries and veins. In its pure form, i. e., without an associated conjunctival injection, ciliary injection occurs in the deep inflammations of the cornea and inflammations of the iris and ciliary body. Though it is by no means rare in any severe conjunctival inflammation, its presence should always be the signal for a careful examination of the cornea and iris.

Injection of the conjunctiva occurs not only in inflammations of the conjunctiva itself, but also in inflammation of other parts of the eyeball. This *symptomatic* injection predominantly affects the bulbar conjunctiva but it also affects the conjunctiva of the tarsus. By beginners it is almost always regarded as a simple conjunctivitis, so that the primary condition, e. g., an iritis, is altogether overlooked. It is, however, readily distinguishable from true conjunctivitis, since in symptomatic injection the retrotarsal conjunctiva is comparatively unaffected. If we evert the lower lid, so as to see all three areas of the conjunctiva at once, this comparative pallor of the retrotarsal fold comes out quite clearly.

449. Inflammation of the conjunctiva—conjunctivitis—comprises many forms, some etiologically distinct, like gonorrhœal and diphtherial conjunctivitis, others clinically well defined but of complex or uncertain etiology. The clinical classification is followed here.

II. Conjunctivitis Catarrhalis

(*a*) *Conjunctivitis Catarrhalis Acuta*

450. Symptoms.—Acute conjunctival catarrh, in the lighter cases, chiefly affects the conjunctiva of the lids and of the retrotarsal fold. The conjunctiva of the lids presents a vivid redness and is relaxed. The injection is usually reticular; it is only when the injection is especially dense that the conjunctiva acquires a uniformly red appearance. The surface of the conjunctiva is smooth; catarrh is thus distinguished from some other forms of inflammation of the conjunctiva in which the latter is infiltrated and subsequently hypertrophied, as shown by the unevenness of its surface. The retrotarsal fold (as well as the plica semilunaris) is likewise greatly reddened and is somewhat swollen, while the conjunctiva bulbi shows little or no change.

The *severe* cases are distinguished from the lighter ones by the fact that the process invades the conjunctiva bulbi and sometimes the cornea, too. These severe forms occur especially in epidemics of conjunctivitis. The redness and swelling of the palpebral conjunctiva are greater, and moderate œdema of the lids is often present at the same time. The conjunctiva of the eyeball shows both a dense reticular reddening and a slight degree of swelling. Very frequently, especially in the form caused by the

Koch-Weeks bacillus, we find in the midst of the reticular injection red-colored spots—i. e., small hæmorrhages,(ecchymoses of the conjunctiva) produced by the rupture of small vessels. The severer cases, in which the conjunctiva is affected throughout its entire extent, are designated under the name of *ophthalmia catarrhalis,* to distinguish them from the lighter forms, which are named simply conjunctivitis catarrhalis. In some of these cases there is a considerable swelling of the retrotarsal fold. This occurs particularly in the epidemic form and, according to Butler, is specially marked in cases due to the pneumococcus. Moreover, in severe cases of pneumococcus infection and sometimes also in other forms of infection, if specially virulent, there may be a formation of false membranes.

Inflammation of the conjunctiva is accompanied by increased conjunctival *secretion.* This secretion appears under the form of flakes of mucus, swimming in the abundant lacrimal fluid. The more intense the inflammation the greater the secretion, and the more the character of the latter changes from mucous to purulent. Violent cases of ophthalmia catarrhalis, therefore, are in their inception often hard to distinguish from a gonorrhœal conjunctivitis of slight intensity, although, of course, the subsequent development of the case makes the diagnosis clear. The secretion which exudes from the palpebral fissure dries at night upon the edges of the lids and glues them together.

The *subjective* symptoms consist of photophobia, and of itching and burning of the eyes. The intensity of the annoyance given depends naturally upon the degree of inflammation. Violent pain, however, is but rarely present, and then, as a rule, is excited, not by the catarrh itself, but by its complications (especially ulcers of the cornea). The same is true of photophobia, which occurs most frequently in cases with corneal involvement and as a result of improper treatment, such as bandaging the eye. A very troublesome sensation frequently present is that of a foreign body in the eye, and is caused by flakes and filaments of tough mucus in the conjunctival sac. If such filaments lie upon the cornea, they produce the disturbances of sight of which the patients sometimes complain. These are distinguished from visual disturbances of more serious character by the fact that clear vision is immediately restored by brushing the mucus off with the lids. It is a characteristic feature of catarrh that all its disagreeable characters are least marked in the morning, and afterward gradually increase until they reach their highest point in the evening.

451. Course and Prognosis.—The prognosis is favorable in uncomplicated cases, the inflammation disappearing spontaneously after from eight to fourteen days. Not infrequently, however, there remains a condition of chronic inflammation (chronic conjunctival catarrh), which, to be sure, causes less annoyance than the acute stage but, on the other hand, is protracted over a long time. In the majority of cases acute conjunctival catarrh attacks both eyes, and either both simultaneously or one eye a few days after the other.

The *complications* which are observed in catarrh are corneal ulcers and iritis. The development of corneal disease is manifested by an increase in

the pain and photophobia. At first we recognize, in the neighborhood of the corneal margin, small gray points, which are arranged in a row concentric with the corneal margin. The next few days, these punctate infiltrations of the cornea become more numerous and at length confluent, so as to form a small gray crescent. By a process of superficial disintegration an excavation is produced, so that finally a crescentic ulcer is formed, situated very near the corneal margin and concentric with it. Such ulcers are characteristic of conjunctival catarrh and are hence called *catarrhal ulcers*. Ordinarily the ulcer heals quickly leaving behind it a slight arcuate opacity which if large looks like an arcus senilis. In cases of special intensity, perforation of the cornea may occur. (For other varieties of corneal ulcers occurring in conjunctival disease see § 537.)

From the clinical picture of acute conjunctival catarrh, as sketched above, we sometimes find variations, forming what are described as special varieties of catarrh. Among these variations belongs the development of so-called *follicles*, which will be described more precisely in § 458. Another variety of catarrh is *vesicular catarrh*, in which the conjunctiva of the tarsus is covered with numerous minute elevations, looking as if fine sand had been scattered over a moist glass plate (Arlt); according to Mayweg what we have to do with here is very small follicles. A third variety of catarrh is that to which is given the name of the *pustular* form. In this, flat elevations develop upon the conjunctiva bulbi, mostly near the margin of the cornea. These break down into pus on their surface, and in this way are formed grayish or yellowish ulcers with somewhat elevated base and of the size of a millet seed or more. These have a great resemblance to the efflorescences occurring in phlyctenular conjunctivitis (§ 482). The distinction between the pustular form of catarrh and phlyctenular conjunctivitis consists in the fact that in the former the phenomena of catarrhal inflammation are present in the conjunctiva of the lids and of the retrotarsal fold, while in phlyctenular conjunctivitis these divisions of the conjunctiva take little or no part in the inflammation. Many regard this form as a mixture of conjunctivitis catarrhalis and phlyctenular conjunctivitis.

The three varieties of catarrh just named are seen chiefly in children or in adolescents. In adults, on the contrary, we encounter much more frequently the crescentic ulcers of the cornea which result from catarrh, but which are rarely observed in children.

In Europe and America the *course* of conjunctivitis is that described above. In the Near East, where conjunctivitis is extremely prevalent, untreated cases are apt to drag on for months, and cases due to pneumococcus or mixed infection resist treatment. Cases due to the Koch-Weeks bacillus are readily cured, but tend to relapse; those due to the Morax-Axenfeld diplobacillus tend to become chronic, but are amenable to treatment (Butler).

The *complications* above mentioned are observed only in severe cases—that is, only in ophthalmia catarrhalis. They very often owe their existence to faulty treatment of the catarrh. Among the laity, all sorts of household remedies are in use for inflammation of the eyes, such as the application of raw meat, or of bread soaked in milk, or of cooked onions, or a bathing with urine, etc. Such remedies are well adapted to increase the inflammation and produce complications. Involvement of the cornea is specially common in infection by the pneumococcus and in certain mixed infections. Pneumococcus infection quite often produces the very serious ulcus serpens corneæ (see § 550).

452. Etiology.—Acute conjunctival catarrh in the majority of cases is certainly produced by bacteria, whose multiplication in the conjunctival sac induces the inflammation of the mucous membrane. The source of the morbid germ varies. They may be transferred from a diseased to a healthy eye; that is, the catarrh may be produced by contagion. This is eminently probable in the case of epidemics occurring in the

same family or the same house. Infected handkerchiefs, towels, etc., may spread the disease; in other cases the transfer may be effected by flies. At certain times, especially in the spring, when so many people are attacked by catarrhs of the air passages, by coryza, coughs, etc., conjunctival catarrhs, too, are generally present in specially great number and many patients with conjunctival catarrh also have at the same time a coryza, in the secretion from which not infrequently the same bacteria are demonstrable as in the conjunctival secretion. In that case the disease, apart from direct transfer, can be spread by particles thrown out in sneezing, coughing, etc. At such times real epidemics of conjunctival catarrh occur, and under these circumstances it is the violent form (ophthalmia catarrhalis) which is prevalent. In Palestine, conjunctivitis, which occurs sporadically during the winter months, assumes in the summer an epidemic form with marked aggravation of the symptoms. Both forms seem to be produced by the same causal agents (Koch-Weeks bacillus, pneumococcus, gonococcus), which, however, in summer receive an access of virulence (Butler). It is further possible that pathogenic germs which are sometimes already present in the normal conjunctival sac may spontaneously multiply so greatly as to excite inflammation.

Acute catarrhal conjunctivitis, also called conjunctivitis simplex, may be caused by different kinds of bacteria (§ 94). These are specially demonstrable in the early stages when they can sometimes be obtained in a pure culture. In the later stage it may be impossible to find any pathogenic organisms. Mixed infections are frequent.

The *Koch-Week's bacillus* (Fig. 204) often excites a severe epidemic conjunctivitis, the acute stage of which lasts from 2 to 4 weeks and leaves a hypertrophy of the conjunctiva that may last for weeks longer. The disease may be associated with coryza and with the formation of phlyctenules in the limbus and of marginal infiltrates in the cornea. But the infection may also run a mild and uncomplicated course (cf. § 451). The secretion in Koch-Weeks' conjunctivitis is extremely contagious, but drying soon destroys its infective power. Cell inclusions (§ 477) are sometimes found in this condition.

The *Morax-Axenfeld diplobacillus* (Fig. 205), which usually causes a chronic conjunctivitis (see § 456), may produce an acute inflammation of mild type, particularly marked by reddening at the angles of the lids (blepharitis angularis). Petit's *diplobacillus liquefaciens* is very similar morphologically, but differs in biologic reactions and in causing a primary involvement of the cornea (Chaine), while with the Morax-Axenfeld bacillus involvement of the cornea, if it occurs at all, is secondary.

The *pneumococcus* may cause an acute inflammation, which lasts only 8 to 10 days and abates so quickly that one might almost speak of a decline by crisis. These cases are usually uncomplicated. In the Near East infection from this cause is more frequent and apparently more severe.

The *micrococcus catarrhalis*, which resembles the gonococcus in appearance and staining properties, occurs in some cases especially of epidemic conjunctivitis. In others the *staphylococcus* seems to be the exciting cause.

Other bacteria are less common. The streptococcus has been found in cases of conjunctivitis with simultaneous disease of the tear sac; the influenza bacillus in epidemics of influenza; and the meningococcus when there is a coincident cerebrospinal meningitis. In some cases the colon bacillus, the bacillus subtilis, the pneumobacillus, and sarcinæ have been found. The diplococcus samoensis is said to cause *Samoan conjunctivitis*, an acute infectious disease, marked by rapid onset, severe pain, photophobia, intense conjunctivitis soon becoming purulent, and a tendency to corneal destruction (Ely).

To the fact that the flora of the diseased conjunctiva is so manifold must be added the further fact that the bacterial finding and virulence also vary greatly with the time and place. For example, the Koch-Weeks bacillus is frequent in New York, and apparently much less so in Chicago, where, on the other hand, the Morax-Axenfeld bacillus

appears to be common. Moreover, even the normal conjunctival sac not infrequently contains pathogenic germs (§ 94).

In some cases a poisonous principle *circulating in the blood* causes the conjunctival inflammation. This is the case in the conjunctival catarrh which accompanies measles, and indeed often forms the first prominent symptom of it (see § 489). Compare also remarks on metastatic gonorrhœal conjunctivitis (§ 460).

In a high degree dependent upon atmospheric influences is that form of acute conjunctivitis which accompanies *hay fever*. This affection, which is pretty frequent in certain countries, attacks individuals who are predisposed to it in the beginning of the summer, and makes itself apparent by fever and also by violent catarrhal inflammation of the conjunctiva and of the air passages. It is probably the result of the direct action of a poisonous substance which is contained in the pollen grains of many gramineæ.

453. Therapy.—By suitable treatment the duration of a conjunctival catarrh can be considerably shortened and the development of a chronic

FIG. 204.

FIG. 205.

FIG. 204.—KOCH-WEEKS BACILLUS. (After Weichselbaum-Müller.) Magnified 925×1. The bacilli are very small, as is shown by comparison with Fig. 205, which is drawn under a magnification only half as great. For the most part the bacilli lie upon the pus corpuscle; at a distance from this can be seen a chain of four bacilli.

FIG. 205.—MORAX-AXENFELD'S DIPLOBACILLUS. Magnified 480×1. In the coagulated secretion lies an epithelial cell and the nuclei of two pus corpuscles. The bacilli are long and thick and are connected either in pairs or in fairly long chains.

catarrh prevented. The sovereign remedy in all the more intense cases of catarrh especially when the secretion is profuse and tends to become purulent, is one of the preparations of silver, either the nitrate or one of the organic salts. In applying the nitrate, we evert the lids so that their conjunctival surface looks forward. This we brush with a camel's hair brush or better with a cotton swab dipped in a 1- or 2-per-cent solution of silver nitrate, taking care that the solution does not come in contact with the cornea; then quickly wash the excess of the solution with lukewarm water or with a weak solution of salt. We now find the surface of the conjunctiva covered with a delicate bluish-white pellicle. This is the superficial slough which the solution has produced. If we use the nitrate, the immediate result of this procedure is violent burning and marked irritation of the eye; an increase, in short, of all the inflammatory phenomena (stage of exacerbation). After this has lasted from a quarter to half an hour, according to the energy of our application, improvement gradually sets in. An examination of the eye at this time shows that the thin slough is separating and is being thrown off in the form of shreds. When this is completed, we find the eye paler and the patient feels relieved and much less annoyed by his catarrh than was the case before the application (stage of remission). This improvement lasts from half a day to a day, according to the intensity of the catarrh. Then the troubles gradually return. This is a signal for

repeating the application. As a rule, it is sufficient to make the application once a day, and best in the morning.

The silver nitrate solution finds an extensive application not only in catarrh, but also in other affections of the conjunctiva. In regard to it the following hints may be laid to heart: (*a*) Many physicians apply weaker or stronger solutions according to the effect which is to be obtained, but we can always succeed with a 2-per-cent solution, since we have it in our power to regulate the effect by making a light or penetrating application. The best applicator is a wooden toothpick wound at one end with cotton. This is thrown away, after being used once. (*b*) We must avoid making the application too energetically. If this has been done, the pain that follows the application lasts unusually long (for hours), and we find that even after a pretty long time, indeed even on the following day, the slough is still adherent in places. This is a proof that the sloughing process has penetrated too deeply. If, in spite of this, we should repeat the application, we would produce a progressively deeper and deeper sloughing of the tissue, and increase the inflammation instead of curing it. We must omit the application, therefore, as long as the slough is still adherent to any part of the conjunctiva. In infants too frequent or too energetic applications may cause profuse and persistent hæmorrhage. (*c*) The application should not be made at night, because the secretion, which is poured out more abundantly after the application, would be retained in the conjunctival sac by the closure of the lids in sleep. For the same reason the eye ought not to be bandaged immediately after the application. (*d*) Corneal ulcers do not constitute a contraindication for making the application; on the contrary, they furnish a direct indication for it, in case they prove to be catarrhal ulcers. Only still greater care than would otherwise be necessary must be taken to prevent the caustic from coming into contact with the cornea. (*e*) If the treatment of the conjunctiva with the silver solution is kept up too long (for some months or a year), there is produced little by little a dirty-gray coloration of the conjunctiva, which never afterwards disappears. This phenomenon, called *argyrosis* or argyria[2] is caused by the fact that silver is deposited in the form of an oxide or an albuminate in the tissues of the conjunctiva (in its elastic fibres), and can never afterwards be removed. Argyrosis is produced even more readily by the constant instillation of the silver solution than by the application of the brush, since in the former case the excess of the solution is not removed by being washed off but remains in the conjunctival sac. This coloration of the conjunctiva is also observed when the conjunctiva is constantly exposed to the action of silver dust, as occurs, for example, in many of those who work in silver.

Nitrate of silver acts as an escharotic on all organic matter, so that it kills all germs with which it comes in contact. At the same time it destroys the superficial layer of epithelium, which in fact forms the superficial slough produced by the application. But it is in this very fact that the value of the remedy consists, since it kills not only the germs on the surface but also those that have penetrated into the epithelium that is cast off.

For silver nitrate may be substituted with advantage one of the organic salts of silver, especially argyrol and protargol. *Argyrol* is said to have no bactericidal action, yet clinically it has proved its efficacy, particularly when used frequently and in strong solutions. The surgeon may apply it with a dropper or with a swab in 30-or even 50-per-cent strength, and the patient may drop in a 10-to 20-per-cent solution 2 to 6 times daily. Some have thought that the best results are secured by keeping the conjunctiva continually flooded with the solution (immersion method—Bruns). The secretions mixed with argyrol form coagula, which act like foreign bodies. Accordingly, the translator in a case of conjunctivitis first flushes out the eye with an indifferent (saline or boric) solution, then instills the argyrol,

[2] From ἄργυρος, silver.

waits a few minutes to allow the latter to penetrate to all the recesses of the conjunctival sac, then washes the eye out again to remove the coagula, and finally instills another drop of argyrol which is allowed to remain. So far from having a cauterant or even irritative effect argyrol is usually distinctly sedative, relieving irritation in acute conjunctivitis. If, however, its use is kept up too long, especially after secretion has stopped, argyrol seems to cause congestion and irritation.

Protargol is more irritating than argyrol, but not as a rule excessively so. The surgeon may apply it once a day in 10-to 33-per-cent strength, and in addition a 5-per-cent solution may be instilled from 2 to 5 times a day. Clinical tests show that protargol is more efficient than argyrol and when applied in strong solution is more efficient than even nitrate of silver (Butler). It is distinctly bactericidal.

One advantage of both argyrol and protargol over silver nitrate is that, being non-irritating, they can be applied at any stage of a conjunctival inflammation—for example before suppuration has set in—and that their administration can be largely intrusted to the patient himself. Another advantage over silver nitrate and also over indifferent detergent solutions is that both argyrol and protargol penetrate into all the recesses of the conjunctival sac, lodge in them a long time (so as to produce a continuous action), and as they gradually exude drive before them the secretion with its contained bacteria. They also force their way down the nasal duct, and help to keep the latter pervious, thus preventing stagnation of secretions and consequent multiplication of bacteria in the conjunctival and lacrimal sacs.

The organic silver salts may cause argyrosis in the same way as the nitrate if their use is kept up too long (two weeks).

In conjunctivitis due to the pneumococcus and in some cases due to the streptococcus, *ethylhydrocupreine* (*optochin*) acts well. A freshly prepared 1-or 2-per-cent solution of the hydrochloride may be applied with a swab or a 1-per-cent solution may be dropped into the eye every hour or two. The drug must be used with caution as it sometimes causes destruction of the cornea.

Mercurochrome, a compound of mercury and fluorescein, has been used in acute conjunctivitis in 1-or 2-per-cent solution (Lancaster).

For less severe cases of acute catarrh (especially for those produced by the Morax-Axenfeld diplobacillus) *zinc sulphate* is used. This is instilled once a day in ½-per-cent solution. It is sometimes effective in ¼-or ⅕-per-cent solution, and is often ordered to be instilled two or three times a day. On the other hand, not a few cases require the use of 1-or occasionally even a 2-per-cent solution. In this strength it is almost a specific for cases of diplobacillus infection.

Many acute cases with mild symptoms and scanty secretion and running a rapid course, require nothing more than a solution of boric acid instilled three or four times a day.[3]

When the acute stage of catarrh has started to decline, we change to the use of *astringent collyria,* such as are employed for chronic catarrh, for which see § 457. For the agglutination of the lids during the night we

[3] For these cases the translator in common with Bruns prefers a solution containing 1 gramme each of boric acid and sodium borate dissolved in 15 grammes each of camphor water and distilled water.

order an ointment (of boric acid or of white precipitate) to be rubbed at bedtime upon the borders of the lids—the eyes being closed.

Corneal ulcers complicating conjunctivitis are treated like corneal ulcers in general, but if there is much secretion a bandage should be avoided, as, in fact, it should be in conjunctivitis under any condition.

In addition to our medicinal treatment of conjunctival catarrh, we must not forget to enjoin upon the patient general *hygienic* rules; telling him to keep the eye clean by washing it with lukewarm water, and to avoid smoke, dust, and bad air in general, and recommending him instead to pass his time in the open air. He must also refrain from straining the eyes much, especially in the evening by artificial light. In consideration, too, of the possibility of spreading the disease by contagion, the patient must take care not to use the same wash basin, towels, etc., as other people. Physicians and nurses handling many cases of acute conjunctivitis should be careful to keep their hands clean and protect their own eyes from any germ-carriers (soiled handkerchiefs, flies). Where but one eye is affected it is well, following Butler's suggestion, to drop a 5-per-cent solution of protargol into the other as a prophylactic.

(*b*) *Conjunctivitis Catarrhalis Chronica*

454. Symptoms.—In chronic conjunctival catarrh the changes *objectively* perceptible are on the whole but slightly pronounced. A moderate degree of redness of the conjunctiva is present either over the tarsus alone or in the retrotarsal fold also. The conjunctiva is smooth and not swollen; it is only in old cases that hypertrophy with thickening and a velvety appearance of the conjunctiva is developed. The secretion is scanty and makes itself chiefly apparent by a gluing together of the lids in the morning. The whitish scum often found at the angles of the lids is produced by the lacrimal fluid being beaten up with the secretion of the Meibomian glands into a sort of a foamy emulsion, as a result of the frequent blinking of the lids. The constant moistening of the skin at this spot often leads to the formation of excoriations. In many cases the secretion, instead of being increased seems even to be diminished. In view of the fact that there is little or no increase in the secretion, several authors call many of these cases not by the name of chronic catarrh, but by that of hyperæmia of the conjunctiva.

In proportion to the insignificance of the objective symptoms, the greater is the attention that has to be paid to the complaints made by the patient—in fact, the *subjective* symptoms are generally so characteristic that the diagnosis of chronic conjunctival catarrh can easily be made from them alone. The discomfort of the patient is usually greatest at night. The heaviness of the lids, scarcely noticeable in the daytime, becomes at night so marked that the patient has difficulty in keeping the eyes open; he has the feeling of being sleepy. An annoying sensation of there being a foreign body—like a speck of dust—in the eye, is produced by the scanty secretion which remains in the conjunctival sac in the form of mucous filaments, and if these filaments lie upon the cornea the sight is interfered with, or rainbow colors make their appearance about a candle flame when looked

at. Further unpleasant sensations of various kinds are described, as, for example, that the eyes burn and itch; that they are dazzled by the light; that, moreover, they are tired out quickly by working; that they blink often, etc. In the morning the lids are somewhat stuck together, or a little yellowish dried secretion is found to have collected in the inner angle of the eye. In other cases there is an annoying sensation of dryness, and the eyes can be opened only with difficulty, the patient, at the same time, having the feeling as if the lids were stuck to the eyeball because of the lack of moisture (*catarrhus siccus*). These troubles, so various in their nature, do not always by any means bear any definite relation to the objective conditions. We see the conjunctiva quite intensely reddened in many people without their complaining in the least; while in others, who do nothing but annoy the physician with their expressions of discomfort, there are often scarcely any changes perceptible in the conjunctiva.

455. Course.—Chronic conjunctival catarrh is one of the most frequent of ocular diseases, chiefly affecting adults, and especially persons somewhat advanced in age. In old people it is almost the rule to find a light grade of chronic conjunctival catarrh, which is denominated senile catarrh. The duration of conjunctival catarrh is ordinarily a long one; many people suffer from it for a great part of their lives. The disease can lead to *complications* which in part produce irreparable changes. Among the most frequent complications is inflammation of the edges of the lids (blepharitis) resulting from the frequent wetting of the palpebral margins by the copiously secreted tears. As a further consequence of this wetting with the tears, the skin of the lower lid is attacked with eczema or becomes rigid and contracted, so that the free edge of the lid is no longer in perfect apposition with the eyeball. As a result of this the punctum lacrimale no longer dips into the lacus lacrimalis, so that the transportation of the tears into the lacrimal sac is impeded, the epiphora increased, and thus again a still further injurious reaction upon the character of the skin is produced. In this way there is formed a vicious circle, which leads to a constantly increasing depression of the lower lid (ectropion). This outcome is still further promoted by the circumstance that the patient keeps wiping away the overflowing tears, and thus makes with the handkerchief stroking movements from above downward, by which the lower lid is drawn down. If the contraction of the skin of the lids thus moistened by the tears is more marked in the horizontal direction, blepharophimosis develops (§ 441). Lastly, small ulcerations of the cornea are a frequent product of catarrh.

456. Etiology.—The causes which lie at the foundation of chronic catarrh are: 1. A preceding acute catarrh, which, instead of healing completely, passes into the chronic stage. 2. General injurious influences of various kinds. Chief among these is bad air, vitiated by smoke, dust, heat, the presence of many people, etc. Workers in factories where there is a great deal of dust, waiters in inns that are filled with smoke, etc., very frequently suffer from chronic conjunctival catarrh. Going late to bed, staying awake at night, and the immoderate use of alcoholic beverages are additional predisposing factors. Persons who already suffer from chronic

conjunctival catarrh find that the latter is made considerably worse after the action of any injurious influence of this sort—for example, after an evening spent at the theatre or in a smoky place. So also the constant action of the wind and bad weather frequently causes catarrh in farmers, coachmen, etc. For the same reason, too, eyes which are very prominent (goggle eyes), or whose lids are retracted (lagophthalmus), are attacked by catarrh, because they are too little protected against the air. The effect which constant contact with the air exerts upon the conjunctiva is best shown in ectropion, in which the conjunctiva tarsi, where it lies bare, becomes very much reddened and thickened, and velvety or even covered with large prominences. The conjunctiva bears continued exclusion from the air as little as it does constant contact with it, on which account chronic catarrh sets in when bandaging of the eye is kept up for a long time. 3. Excessive straining of the eyes, especially in hypermetropic or astigmatic persons, can result in chronic catarrh. 4. Local injurious influences. Here belongs irritation of the conjunctiva by foreign bodies lodging in the conjunctival sac, among which, using the term foreign bodies in the wider sense of the word, are to be reckoned cilia which are turned in toward the eye. In most cases the local injurious influence consists of some other disease of the eye, that induces catarrh as a sequela, as, for example, blepharitis or infarction of the Meibomian glands. Accumulation of the tears, as a result of disease of the tear sac, or because the punctum lacrimale does not dip properly into the lacus lacrimalis, is a frequent cause of catarrh, so that we should never forget to look for an infection of the tear passages in unilateral catarrh. We say unilateral, for catarrh produced by local causes is distinguished from that due to general injurious influences in this respect, that the former is very frequently unilateral, while in the latter, from the nature of the case, both eyes are generally affected.

In many cases of chronic catarrh there is found in the scanty secretion the diplobacillus of Morax-Axenfeld, which, in § 452, was noted as a cause of acute conjunctivitis, but which much more often produces a catarrh that is chronic from the start. These cases are often distinguished by a marked redness of the skin at the angles of the eye (whence the name of *angular conjunctivitis, angular blepharitis*). The symptoms are persistent itching and burning and sometimes an obstinate asthenopia.

457. Therapy.—It is clear that the treatment must first of all pay regard to the causal factor by regulating in a suitable way, as far as is compatible with the patient's calling, the general conditions under which he lives, and by removing all local causes of catarrh that may be present, etc. For the treatment of the conjunctiva itself we first employ, as we do in acute catarrh the *nitrate of silver*, which is applied by the brush in 1-or 2-per-cent solution. We make use of it in those cases only in which the catarrh is accompanied by rather abundant secretion and by relaxation of the conjunctiva—that is, in periods of acute exacerbation, such as frequently occur in the course of every chronic catarrh—and use it, furthermore, when hypertrophy of the conjunctiva has already set in. Otherwise we succeed better with *astringent collyria*, which the patient can instil himself. The most usually

employed of these is zinc sulphate, which is instilled in ½ or 1-per-cent solution once or twice a day, and is especially indicated in cases of diplobacillus conjunctivitis. In the chronic catarrh of elderly people with marked reddening of the conjunctiva but no secretion, the most suitable remedy is the collyrium adstringens luteum,[4] which is not ordinarily prescribed undiluted but mixed with an equal quantity of water. Other astringents are lapis divinus [5] (½ per cent), alum [6] or tannin (1 per cent), boric acid (3 per cent).

The order in which these collyria are here arranged about corresponds to their gradation in activity from the strongest to the mildest. They should be instilled once or twice a day, but not at night. So many of them are enumerated, because it is good to have a pretty large number to select from, since as the catarrh is of long duration, a change will have to be made pretty often in the remedies. Every remedy, if too long applied, loses its activity, since the conjunctiva grows accustomed to it. For the sticking together of the lids, as well as for any excoriations that may be present, an ointment of boric acid (2 per cent) or of white precipitate (½ to 1 per cent) may be rubbed upon the closed lids at bedtime.

(c) *Conjunctivitis Follicularis*

458. Follicular catarrh is characterized by the presence of *follicles.* These are small round granules of about the size of a pin's head which lie in the retrotarsal fold. They are of a pale, translucent aspect and puff up the conjunctiva in the form of small eminences. Either only a few follicles or many are present; in the latter case they are ordinarily arranged in rows like a string of beads. The upper retrotarsal fold usually contains fewer follicles than the lower. Microscopic examination shows that the follicles, like the trachoma granules, consist of a circumscribed accumulation of adenoid tissue (Fig. 210, *T*).

Follicles are most frequently observed in youth, and especially among pupils in day schools, boarding schools, etc. In many school children the disease exists in a perfectly latent form, as, in spite of there being a considerable number of follicles, the conjunctiva is not reddened and causes no symptoms of any kind, so that the trouble is discovered only by medical examination. In such cases the follicles often persist for years.

In other cases the disease has an acute character; a conjunctivitis sets

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[4] This collyrium, called also Horst's eye water, is of the greatest service and in many cases can be replaced by no other. It is prepared in the following way:

Take of ammonium chloride 50 centigrammes and zinc sulphate 125 centigrammes, dissolve in 200 grammes of distilled water and add a solution of 40 centigrammes of camphor in 20 grammes of dilute alcohol and 10 centigrammes of saffron. Digest for twenty-four hours with frequent agitation, and filter.

The translator has found very useful a preparation made by mixing 13 to 25 centigrammes of zinc sulphate, 1 gramme, each, of boric acid and sodium borate, and 30 grammes of distilled water, and filtering.

Rather stronger in action is the tinctura opii crocata, diluted with an equal amount of water and filtered. (The tinctura opii crocata is a solution of opium and saffron, about 9 per cent each, in cinnamon water.)

Romershausen's eye water, which is also frequently employed in chronic ophthalmic catarrh, consists of a mixture of aqua fœniculi and tinctura fœniculi.

[5] Or aluminated copper; a preparation made by fusing together 32 parts each of copper sulphate, potassium nitrate, and alum, and adding a mixture of 2 parts each of camphor and alum.

[6] Alum is more often applied in the solid form, as a sharpened crystal (*alum stick*) with which the conjunctiva is stroked. *Gallicin* is also employed, being sprinkled in fine powder with a brush upon the conjunctiva.

in with the symptoms and disagreeable accompaniments of a violent catarrhal ophthalmia and with the simultaneous development of numerous follicles. This acute form often occurs epidemically and is more obstinate than a catarrhal ophthalmia that is not complicated with follicles. On the other hand, the follicles do not last as long as in the chronic form, but soon disappear when the conjunctivitis abates. Intermediate between the very chronic and the acute form are the particularly numerous cases in which the presence of follicles is combined with slight irritation of the conjunctiva and with correspondingly mild symptoms such as obtain in a chronic catarrhal conjunctivitis.

The *treatment* of follicular catarrh is directed mainly at the accompanying inflammatory symptoms on the part of the conjunctiva. If these are altogether absent, no treatment at all is needed, as ultimately the follicles disappear of themselves, leaving no trace of their presence. If there are very many follicles ointment of copper sulphate (1–1,000) may be used. If the follicles are associated with inflammatory changes in the conjunctiva the latter are to be treated according to the principles laid down for catarrhal conjunctivitis—that is, by applying 2-per-cent nitrate of silver or a 10- to 25-per-cent solution of protargol or argyrol if the inflammation is an active one, and in the milder cases by dropping in a solution of zinc sulphate (½ per cent). As in catarrh of all kinds, so particularly in follicular catarrh, living in fresh, pure air is to be enjoined.

The *prognosis* is altogether good, as the disease leads to no complications and leaves no permanent changes whatever; in this being opposed to trachoma, which it otherwise somewhat resembles (§ 473).

The fact that follicles may at one time be associated with intense inflammation of the conjunctiva and at another time may not cause the slightest evidences of irritation is probably to be explained as follows: The follicles are a circumscribed new formation of lymphoid tissue, this new formation being the way in which the conjunctiva reacts to certain irritants. Lymph follicles occur in many mucous membranes, and even in the conjunctiva they are normally present in many of our domestic animals, though not in man. The injurious influence which induces the formation of follicles in man is not necessarily of a bacterial nature, for in many men we see a conjunctival catarrh with numerous follicles develop after the continued instillation of an atropine solution even when it has been carefully sterilized (*atropine catarrh*) (see § 126). In the chronic cases of follicular catarrh the irritation probably originates in the impure air of the school room or in similar injurious influences and causes simply a gradual development of follicles, but not any inflammatory changes in the conjunctiva. In that case, to be sure, the follicles, if they are numerous, may in their turn induce slight evidences of irritation in the conjunctiva. In the cases of acute follicular catarrh, however, there is probably a bacterial infection, such as would under other conditions simply cause a catarrhal ophthalmia but in this case produces in addition a rapid development of follicles.

Follicular conjunctivitis is said to be rare in the negro (Calhoun).

Follicles are also found in trachoma, inclusion conjunctivitis, and other conditions (see §§ 473, 477).

III. Conjunctivitis Gonorrhoica

459. Conjunctivitis gonorrhoica, also called acute blennorrhœa[7] or gonorrhœal ophthalmia, is an acute inflammation of the conjunctiva, which

[7] From βλέννα, mucus, and ῥέω, I flow.

originates in contagion from gonorrhœal virus, and whose copious purulent secretion is likewise contagious in its action. The carriers of the contagion are gonococci, which are found both in the pus secreted by the conjunctiva and also in the most superficial layers of the conjunctiva itself. They are mostly arranged in pairs, as diplococci, and as a rule lie together in heaps. Fig. 206 shows a specimen taken from the secretion of gonorrhœal conjunctivitis. In it are seen the heaps of gonococci, partly free (*a*), partly upon and within the cells, which are either pus cells (*b*) or cast-off epithelial cells (*c*).

Gonorrhœal conjunctivitis occurs in new-born infants or may develop later in life. The following description refers to the latter form; the disease as it occurs in the new-born will be treated of under the head of ophthalmia neonatorum:

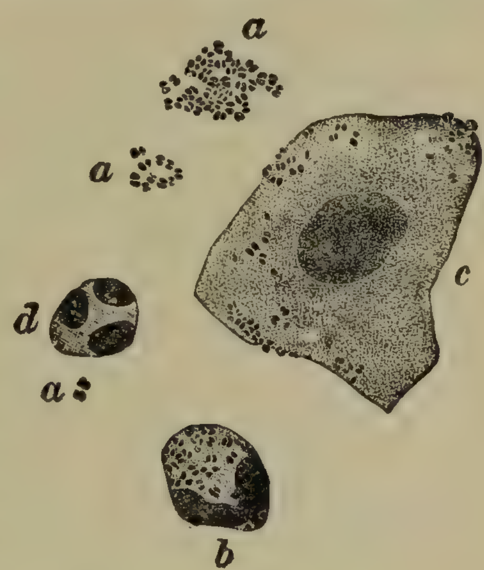

Fig. 206. Secretion of Acute blennorrhœa with Gonococci.

In Europe and America postnatal gonorrhœal conjunctivitis is not a very common affection. According to White, it occurs but once in every seven or eight hundred cases of gonorrhœa. In Egypt and Palestine it is exceeding frequent, most of the cases occurring in children of from one to five years of age.

It must be observed that the micrococcus catarrhalis and the meningococcus, which resemble the gonococcus in morphology and staining, and differ only in biological characters, can also cause an acute purulent conjunctivitis. Since these organisms cannot be differentiated in the smears, but only in cultures, it is possible that some cases of conjunctivitis have been classed as gonorrhœal, which were not. Cf. remarks on Extragenital Gonorrhœa, § 463.

In some cases of gonorrhœal conjunctivitis cell inclusions are found, which, however probably denote a mixed infection (see § 477).

460. Symptoms and Course.—When infection has taken place, the disease breaks out after a certain period of incubation, the duration of which varies, according to the intensity of the contagious action, from a few hours up to three days. The lids grow red, become hot, and are swollen with œdema, generally to such an extent that the patient can no longer open them, and even the physician often has trouble in separating them far enough from each other to bring the cornea into view. The conjunctiva of the lids and of the retrotarsal fold is intensely reddened and greatly swollen. The swelling is produced by an abundant cellular infiltration of the conjunctiva, which is consequently tense, and has a granular, uneven surface. This feature of acute blennorrhœa serves to distinguish it from catarrh, in which even in the severe cases the swelling is rather of a serous nature, and hence the conjunctiva is yielding and has a smooth surface. The conjunctiva of the eyeball shows a like tense swelling, which stops short at the corneal margin, so that a raised wall is thus formed about the more deeply placed cornea (chemosis). The secretion produced by the conjunctiva is like meat juice—that is, it is a serum which is colored red by admixture with blood, and in which float some flakes of pus. The eye is uncommonly sensitive to contact, the lymphatic gland in front of the ear is swollen, the patient has slight fever.

Ordinarily it takes from two to three days for the disease to mount from its initial point to the pitch just described, and at this pitch it is maintained for two or three days more. This period is designated as the first stage, or *stage of infiltration.* Succeeding this as a second stage is that of *pyorrhœa.* The swelling of the lids gradually diminishes, a fact which we recognize principally by means of the return of the small wrinkles of the skin of the lids, and the tense infiltration of the conjunctiva slowly retrogrades. Simultaneously with this there begins a very profuse secretion of pus, which trickles out continually from the palpebral fissure; hence the name of pyorrhœa, or flow of pus. In the further course of the disease the conjunctiva keeps getting less and less swollen all the time, and in many cases returns by degrees to the normal within four or six weeks. In most cases, however, a condition of chronic inflammation of the conjunctiva remains, which is designated as the third stage of the disease, the stage of *chronic blennorrhœa.* In this period the lids are no longer swollen. The conjunctiva is reddened and thickened, especially upon the tarsus, where its surface looks uneven, granular, or velvety. The retrotarsal fold forms an ungainly swelling; the conjunctiva of the eyeball, which shows hyperæmia only, is the least changed. After this state of conjunctival hypertrophy has abated, a process which usually takes months for its accomplishment, there usually remain slight, but permanent cicatrices of the conjunctiva.

The description here given corresponds to the cases of most frequent occurrence, which are those of medium intensity. In addition, both light and also very severe cases of the disease come under observation which exhibit rather different features. In the light cases, which we are accustomed to call *subacute blennorrhœa,* all the inflammatory changes are less, and the changes are limited chiefly to the conjunctiva of the lids. Frequently such cases are not to be distinguished with certainty by their external aspect from severe catarrh. The diagnosis can be rendered certain by the microscopic examination of the secretion, since by it the presence or absence of gonococci is demonstrated. Such subacute and also chronic forms are found particularly in Egypt, where also the disease shows a seasonal incidence, being especially prevalent in spring (Beaton).

In the *severest* cases, the infiltration of the conjunctiva is so great that the latter in places appears no longer red, but grayish-yellow, because, as in diphtheria of the conjunctiva, the vessels are compressed by the bulky exudation, and the conjunctiva is thus rendered anæmic. The conjunctiva forms about the cornea a tense grayish-red wall. Quite often the surface of the conjunctiva is found to be covered with a clotted exudate, or croupous membrane.

Quite a different picture is presented by the not very infrequent *metastatic gonorrhœal conjunctivitis* (Byers). This occurs as an acute, usually bilateral catarrhal conjunctivitis, with slight and mucoid, rarely purulent secretion and without infiltration of the conjunctiva. Uncomplicated cases run their course in about two weeks, but corneal and other complications (see infra) often prolong the disease, and relapses also may occur.

461. Complications.—The most dreaded complication of acute blennorrhœa is the *involvement of the cornea,* by which, in many cases, incurable blindness is produced. At first the cornea becomes dull upon its surface and covered with a slight diffused opacity. Then circumscribed infiltrations of grayish color make their appearance, which soon become yellow and break down into ulcers. These infiltrations may be situated at the margin of the cornea, and give rise to speedy perforation of the latter. This is a comparatively favorable result, as, after the perforation has taken place, the purulent infiltration of the cornea not rarely is brought to a standstill, and so a portion of the cornea is preserved. But it can also happen that the marginal infiltrations become rapidly confluent, and unite into a yellow ring surrounding the entire cornea (a so-called annular abscess). In that event the cornea is lost, for this ring soon spreads over the entire cornea and destroys it. In other cases the purulent disintegration occurs first in the middle of the cornea. When, in one way or another, the cornea has gone either entirely or in part to destruction, the outcome is either a formation of cicatrices with incarceration of the iris, or it may even be a panophthalmitis. Since these sequelæ are observed after every destruction of the cornea, even when due to other causes, they will find detailed description under the diseases of the cornea.

Involvement of the cornea is the more certain to take place, the severer the conjunctivitis, and, in particular, the more pronounced the participation of the conjunctiva bulbi in the inflammation. In the severest cases with tense chemosis the cornea is alway affected, and is, as a general thing, irretrievably destroyed. In the cases of moderate severity when the chemotic swelling of the conjunctiva is less pronounced and especially is less hard, it is usually possible to preserve the cornea, either entirely or in great part, inasmuch as the ulcers that develop, even if they are attended with perforation, are of but small size. In the lightest cases, where the process is limited to the palpebral conjunctiva, there is, on the whole, little danger to the cornea. This statement, however, does not apply to the metastatic form, which, though comparatively mild, is often complicated by a keratitis, usually bilateral and symmetrical, with multiple superficial infiltrates, or by irido-cyclitis (Byers).

The severer the course of the inflammation, the earlier the involvement of the cornea sets in; in bad cases, the cornea is already clouded by the second or third day. Sometimes corneal ulcers are not developed until late in the disease, when the conjunctivitis is already well on the retrograde path. These late affections of the cornea are not very dangerous, and it is generally possible to check them readily.

The *inflammation of the cornea* is to be referred to infection of the latter by the secretion which constantly bathes it and macerates its epithelium. Since the secretion collects chiefly in the gutter lying at the rim of the cornea, between the latter and the steep slope of the chemotic conjunctiva, the purulent infiltration most frequently begins here, too. The tense infiltration present in this chemotic wall of conjunctiva is to be regarded as a second factor in the production of corneal trouble. This leads to obstruction of the circulation in the marginal loops of the cornea, and thus interferes with the nutrition of

the latter. Hence, in cases where the chemosis is unequally great, we often see the involvement of the cornea take place first at that portion of the corneal rim where the chemosis is the greatest.

Since infection of the cornea is certainly very greatly favored by the existence of gaps in the epithelium of the latter, we must avoid injuring the epithelium of the eye by carelessness in cleansing.

If gonorrhœal conjunctivitis happens to affect an eye which is covered with *pannus* the latter will afford the cornea a secure protection against suppuration. Nay, more, it is often apparent, after the violence of the inflammation has passed, that the pannus has cleared up considerably, so that in cases of old pannus inoculation with gonorrhœal conjunctivitis has been designedly performed. This procedure has now been abandoned in favor of the treatment with jequirity.

Suppuration of the swollen *preauricular gland* is sometimes observed.

As gonorrhœa of the urethra can by metastasis excite conjunctivitis, so also conversely cases have been observed in which a gonorrhœal arthritis, where gonococci have been demonstrated to exist in the pus, has arisen by way of metastasis from a blennorrhœa of the conjunctiva. Cases of this sort have been known both in adults and in new-born children affected with blennorrhœa (Deutschmann and others).

462. Prognosis.—The prognosis is determined essentially by the condition of the cornea. This is dependent upon the intensity of the inflammation of the conjunctiva bulbi, and on this, therefore, the prognosis must be based. The earlier corneal ulceration sets in, the severer it is, so that the longer we can keep the cornea normal, the better the prognosis. The conjunctival disease itself gets well without complications.

The prognosis in all genuine cases must be regarded as serious, although with improved methods of treatment it is better than it used to be. According to Hirschberg, 11 per cent of present-day cases retain fair vision and only 17 per cent become blind (as against 50 per cent formerly). Young children respond better to treatment than do adults, and this perhaps explains why the progress is better in the Egytian form of the disease, which is so frequent in the very young. In the metastatic form, if uncomplicated, the prognosis is good.

463. Etiology.—Acute blennorrhœa is produced by infection. The poison can be introduced into the eye from the genitals directly, generally because an individual (whether man or woman) affected with gonorrhœa touches the eyes with unclean fingers after these have been in contact with the genitals. The infection, however, can also come from an eye affected with blennorrhœa. If, for instance, one eye is already diseased and is affected with profuse suppuration, the other eye can also be infected by a transfer of the secretion to it. An individual with an eye diseased with blennorrhœa can infect the persons who are nursing him or any others who may share his room. This sort of transfer is the rule in the cases observed in Egypt (Beaton).

Metastatic conjunctivitis is produced by the passage of gonococci or the toxin that they produce from a remote focus in the body to the eye by way of the blood. The corneal and other complications in this case may be associated manifestations of the same metastasis that causes the conjunctivitis or may be secondary to the latter.

In Palestine and Egypt a good many cases of acute conjunctivitis of virulent character, and often causing destruction of the cornea, are due to diplococci which in smears appear identical with the gonococcus. As genito-urinary gonorrhœa is extremely rare

in these countries, direct infection from the urethra or vagina is unlikely, and these cases have been regarded as due to *gonococcus infection of extragenital origin* (Butler, Ticho, and others). Since, however, the gonococcus cannot be differentiated in smears from other diplococci (micrococcus catarrhalis, meningococcus), which may cause acute conjunctivitis, this diagnosis has been regarded as doubtful. But recent researches seem to show its essential correctness (Beaton).

The secretion containing gonococci is usually brought into the eye by means of dirty fingers or by the use of a contaminated sponge, towel, or handkerchief. Sometimes, however, a *direct transfer* from the diseased mucous membrane to the sound one is observed; for example, when a drop of secretion spurts into the eye of the physician or the attendant while cleansing genitals that are affected with gonorrhœa, or even when cleansing the eye of a patient affected with blennorrhœa.

If one eye is already infected, the transfer to the other is often brought about by secretion of the diseased eye flowing over the bridge of the nose into the sound eye during sleep.

We sometimes also observe *acute purulent conjunctivitis in small girls* of the age of two to ten years, who at the same time are troubled with a vaginal discharge (Arlt). These cases are not always gonorrhœal. A simple catarrhal secretion of the vagina may excite an inflammation of the conjunctiva, which in this instance runs a less severe course than a gonorrhœal conjunctivitis, from which it can be distinguished by a microscopical examination of the secretion, which proves the absence of gonococci.

Piringer's inoculation experiments have shown the *relation between the infective material and the ophthalmia produced by it.* He found that the more violent the blennorrhœa that serves as the source of the material inoculated, the shorter is the period of incubation. The infective power of the secretion is weakened by various influences, as by dilution with water—by dilution to the one-tenth strength any secretion can be rendered inert—or by drying. Secretion that has been dried upon a piece of linen loses its activity after thirty-six hours. Preserved like vaccine, it remains infective for sixty hours. In proportion as the virulence of the infecting secretion is weakened, the period of incubation increases in length and the inflammation excited becomes milder. The differences that we observe in the grades of gonorrhœal conjunctivitis can therefore be referred to the fact that the source of infection supplies secretion of different degrees of virulence, and this virulence is, moreover, still further modified by the immediate circumstances attending the process of infection and by the susceptibility of the conjunctiva, which varies greatly in different persons.

464. Prophylaxis.—By proper prophylaxis infection by gonorrhœal conjunctivitis can be prevented, a matter to be so much the more borne in mind because, when the disease has once broken out, an unfortunate result cannot always be averted. It is the physician's duty to call the attention of every man with gonorrhœa, and also of every woman with a vaginal discharge, to the danger of infecting the eyes, and to urge upon them strenuously the requisite cleanliness. If an eye is already attacked with acute blennorrhœa, care must be taken to keep the other eye from being infected by it and also to keep the disease from being transferred to persons in the vicinity. The protection of the second eye which has not yet been involved in the disease is best effected by a bandage which is applied in the following manner: The palpebral fissure is first closed by means of some narrow strips of sticking plaster applied in a vertical direction. Then the hollow about the eye is filled up with cotton, and the whole is covered by a strip of plaster (zinc adhesive plaster, zinc mull, etc.), which is cut to the proper shape and is carefully attached all round the margins of the orbit. In order to secure it better, the edges of the flap and the adjacent skin may further be coated with collodion. It is well to have the dressing include a

transparent covering (watch crystal, Buller's shield) hermetically fastened over the eye. The eye can then be inspected without removing the dressing. If there is reason to think that infection of the eye apparently healthy has already taken place, we may try to prevent the outbreak of the disease by instilling a 2-per-cent solution of silver nitrate or 5-per-cent protargol before applying the bandage.

To prevent the spread of the disease to those in the neighborhood of the patient, the greatest cleanliness must be inculcated both upon him and upon the persons attending him; they must always cleanse the hands after touching the affected eye, and must remove, or, best of all, burn, all materials that have been used for cleansing the eye (pieces of linen, cotton, etc.). The physicians and nurses should use protective (large, colorless, coquille-shaped) eye guards. If in spite of this protection secretion spurts into the eye, the latter must be very thoroughly washed out; then a couple of drops of 2-per-cent silver nitrate should be instilled, and subsequently for some hours cold compresses placed on the eye.

465. Treatment.—The treatment of the disease itself consists primarily in careful, repeated *cleansing* of the eye from its profuse secretion (by night as well as day). This is done with a weak lukewarm solution of potassium permanganate (1–5,000), with which the conjunctival sac is washed out thoroughly three or four times a day (Kalt) or with warm boric acid solution used even more frequently. The irrigator used had best have a flattened spout so as to be introduced readily between the lids and eyeball. Between each irrigation the eye is cleansed assiduously with cotton pledgets moistened in the same solution; the lids being drawn gently apart when this is done. Specially valuable, according to Kirkpatrick are hypertonic solutions of magnesium sulphate (10-per-cent or stronger) used as an eye-bath for 5 minutes every two or three hours. This reduces the chemosis and in the majority of cases prevents destructive inflammation. Parenteral *injections of milk* (repeated twice or thrice, if necessary) are said to cause disappearance of the gonococci and rapid subsidence of the chemosis (Pillat and others).

If the great swelling of the lids does not permit the palpebral fissure to be properly opened, and thus makes cleansing impossible, the palpebral fissure must be fully widened by a section made with the scissors at the external angle of the lids (*canthotomy;* see § 923). This section has the further beneficial effect of diminishing the pressure which the much-swollen lids exert upon the eyeball.

In the first stage of the disease we combat the inflammation by *iced compresses*, applied continuously or at short intervals, according to the effect they produce on the swelling of the lids, and also by the application of leeches (six to ten in number) to the temple. In the second stage brushing the conjunctiva with *nitrate of silver* is the best means for making the swelling of the conjunctiva and the profuse secretion rapidly disappear. The application of the brush must not, however, be begun until the tense swelling of the conjunctiva has given place to a soft, succulent condition; there should no longer be any membranous deposit, nor any grayish infil-

trated spots upon the conjunctiva. The application should be made with a 2-per-cent solution, but quite freely, and must be repeated twice a day as long as the profuse secretion is still present. The presence of ulcers of the cornea furnishes no contraindication to the use of nitrate of silver.

As soon as, in the third stage, the inflammatory symptoms and the secretion also have nearly disappeared and the thickening of the conjunctiva is the only thing that still remains to be removed, we exchange the silver solution for *copper sulphate.* This is applied by whittling a crystal of the substance down to a smooth, rounded extremity (copper pencil or bluestone) and stroking with it once or twice the conjunctiva of the everted lids. Then the lacrimal fluid, which is tinged blue by the copper salt dissolved in it, is dipped up from the conjunctiva with a pledget of cotton; otherwise the pretty concentrated copper solution would come into contact with the cornea and irritate it greatly. The application of the bluestone is much more painful than that of the silver solution, but acts more energetically, and hence we get quicker results with it; but this treatment is permissible only if the cornea is either quite sound or has ulcers already in process of cicatrization, and not if there are fresh ulcerations of the cornea, which are still coated with pus.

The treatment of complications involving the *cornea* is conducted according to the rules (§ 541 et seq.) for purulent keratitis. In very severe cases all treatment proves powerless to preserve the cornea, so that our efforts must be confined to the prevention of the more remote evil consequences of destruction of the cornea, like panophthalmitis or the formation of staphyloma, and to the securing of a flat cicatrix.

IV. Ophthalmia Neonatorum

466. We designate by this name the inflammations of the conjunctiva occurring in the new-born. They do not constitute an etiological unit, being produced by a variety of morbid germs; yet on practical grounds it is advisable to retain the group-name of ophthalmia (or blennorrhœa) neonatorum.

The majority of cases, and particularly almost all the severe cases, owe their origin to the gonococcus, and hence are in the same category as the gonorrhœal conjunctivitis of adults. The infection that gives rise to this *ophthalmia neonatorum gonorrhoica* occurs as a rule during parturition. In the passage of the child's head through the vagina, the eyelids are covered with the secretion contained in the latter, and this either penetrates immediately into the conjunctival sac through the palpebral fissure, or does so as soon as the child first opens his eyes. Under these circumstances the disease breaks out as a rule on the second or third (rarely on the fourth or fifth) day after birth. In those cases in which the disease makes its appearance still later than this, the infection can not any longer be referred to the act of birth. It has then been brought about through subsequent infection by the vaginal secretion of the mother (as is readily possible, particularly if the child sleeps in bed with the mother) or the child has been infected by another child, as, for instance, not rarely happened in former times in lying-in establishments and foundling asylums.

The *symptoms* of the disease are the same as in the gonorrhœal conjunctivitis of adults, except that they are in general less severe. For even when there are great swelling of the lids and very profuse purulent discharge, the part which the bulbar conjunctiva takes in the process is comparatively small, and we rarely find great chemosis. Hence also the danger of suppuration of the cornea is not so great. It does indeed occur, and that often enough too, but only in those cases which are treated badly or not at all. If a case comes under treatment in season—that is, while the cornea is still intact—the latter can almost to a certainty be maintained in a healthy state. The prognosis, therefore, can be stated as correspondingly favorable.

Conjunctivitis of the new-born may be produced by other morbid germs than the gonococcus (pneumococcus, colon bacillus, etc.). In these cases, too, the infection generally occurs during birth; but the inflammation usually breaks out rather later and runs a milder course than in the gonorrhœal form. It presents the symptoms of a more or less intense catarrhal ophthalmia. But since gonorrhœal cases are occasionally of a milder character, a sure distinction between the gonorrhœal and the non-gonorrhœal forms can be made only by bacteriological examination of the secretion. In making this it must be remembered than an acute conjunctivitis may be caused by organisms, which in smears are apparently identical with the gonococcus, (page 449) so that the differentiation can be made only in cultures. In some cases cell inclusions are found (§ 477).

The *treatment* in the first stage consists in diligent cleansing of the eye (see § 465); when suppuration commences we begin with the application of a 1-or 2-per-cent nitrate-of-silver solution to the conjunctiva. In cases with profuse secretion this must be done twice a day. The application should be continued until the cure is complete, as otherwise the process may readily recur to a moderate degree. It is particularly important that the application should not be made too often nor too vigorously (see § 453). In making it, injury to the child's eye should be avoided by fixing the child's head in the manner described in § 387 and by separating the lids with Desmarre's elevators (Fig. 388, *d*) instead of with the fingers, which may cause too much pressure on the eyeball.

In ophthalmia neonatorum, as in purulent conjunctivitis in general, both argyrol and protargol are used in place of silver nitrate, and with satisfactory results if applied sufficiently often and in sufficiently strong solution (see § 453).

In the conjunctivitis of the new-born *prophylaxis* plays a still greater part than in gonorrhœal conjunctivitis of adults. There is perhaps no other eye disease in which the rigorous carrying out of prophylactic treatment would afford more gratifying results than in the conjunctivitis of the new-born, which might by means of it be made to disappear almost entirely. Credé's method has proved the best. While the first bath is being given the child's eyes should not be wet with the water of the bath but should be cleaned with extreme care with pure water and cotton. Then as soon as

the child has been put on the dressing table to be wrapped up, a drop of a 1- or 2-per-cent solution of nitrate of silver is instilled into each eye.

Ophthalmia neonatorum is one of the most frequent of diseases; before the introduction of the prophylactic treatment it attacked from 1 to 20 per cent of children in the various lying-in establishments. Since children of the poorer class who were born outside of the institutions and were affected with gonorrhœa often were brought under medical treatment either too late or not at all, many of them went blind. Before the introduction of prophylaxis, ophthalmia neonatorum caused more than a tenth of all cases of blindness. Even today among a large number of blind or partially blind children examined, in 9 per cent the disability was found to be due to this disease (Harman). In consonance with its usual etiology (genital gonorrhœa), ophthalmia neonatorum is unknown in Palestine, where also gonorrhœa practically never occurs (Butler, Friedenwald).

That prophylaxis, as introduced into practice by Credé, is actually efficient, is proved by the following data: Credé formerly had in the Leipsic Lying-in Asylum an average of 10.8 per cent of cases of blennorrhœa neonatorum in the whole number of new-born; after the introduction of his prophylactic method the number sank to 0.1 to 0.2 per cent. Others have similar favorable results to record. Hence, the endeavor should be made to have this form of prophylaxis carried out not only in lying-in establishments and by physicians, but also universally by mid-wives. The majority of pregnant women have catarrh of the vagina with a mucous or purulent discharge. In the greater portion of these cases we have to do with a benign vaginal catarrh, in a smaller portion with a virulent catarrh (gonorrhœa). In individual cases the distinction between benign and virulent is difficult or impossible, for which reason prophylactic treatment ought to be carried out in all cases.

Credé's prophylactic treatment acts as a protection against infection during parturition only. Infection can, however, take place even earlier; children have been known to come into the world with a blennorrhœa already fully developed—in fact, with the cornea already destroyed. Furthermore, against subsequent infection by the vaginal secretion of the mother, or by other children, other measures will have to be adopted, among which great cleanliness is to be assigned the first place. In foundling asylums, infants infected with blennorrhœa should be isolated from the rest, as otherwise infection will frequently take place.

In infants after the subsidence of the acute inflammation, a chronic hypertrophy of the conjunctiva (chronic blennorrhœa) develops much less frequently than in adults. On the other hand, even after an ophthalmia neonatorum, if severe, a mild cicatricial condition of the conjunctiva, especially of the retrotarsal folds, often remains for life. Perhaps, too, the change described by Schapringer under the name *epitarsus* and considered by him to be congenital, should in many cases be attributed to a cicatricial formation of this sort. This formation consists in a flat fold of conjunctiva which runs down from the convex border of the tarsus over the conjunctiva of the lid and, growing constantly narrower, finally becomes attached again to the conjunctiva of the lid near the free border of the latter. As in the case of a pseudopterygium (§ 504), we can generally pass a sound between the fold and the palpebral conjunctiva.

V. Conjunctivitis Trachomatosa

467. Nature and Symptoms.—Trachoma is an inflammation of the conjunctiva, which originates by infection, and produces an infectious purulent secretion. It is distinguished principally by its chronic course, in which is developed a hypertrophy and consequent roughening of the conjunctiva, which forms the most characteristic symptoms of the disease and from which the latter has, in fact, received its name.[8]

[8] Trachoma, from τραχύς rough.

The disease almost always attacks both eyes, but cases do exceptionally occur in which the disease, even after it has lasted for years, remains confined to one side.

The patients complain of sensitiveness to light, of lacrimation, and of sticking together of the lids; pain and visual disturbances are also often present. Examination shows that the eye is less widely opened, partly because of photophobia, partly because the upper lid droops.

The *ptosis* which almost always accompanies trachoma and which gives trachomatous patients their characteristic appearance is in many cases attributable to the fact that the lid droops because it is heavy. Ptosis, however, is observed even when the thickening of the conjunctiva is inconsiderable, or indeed not present at all, and sometimes patients come to a physician solely on account of the ptosis, without having experienced any other trouble from their trachoma. In such cases the ptosis is probably due to a paralysis of the tarsal muscle, which is involved in the infiltration and later in the cicatricial contraction.

After everting the lids, we see the conjunctiva of the tarsus and also that of the retrotarsal fold diffusely reddened and thickened; its surface at the same time has become uneven to a varying degree. These changes are to be referred to a hypertrophy of the mucous membrane, which occurs under two different forms.

The *first form* consists in the development of the so-called papillæ. These are elevations newly formed on the surface of the conjunctiva, which consequently appears velvety, or, if the papillæ are large, appears studded with coarse granules, with small nodules, or even with raspberry-like projections, the thickening of the conjunctiva being so great that the subjacent Meibomian glands are no longer visible through it. This kind of hypertrophy, which is called the *papillary form*, if found exclusively in the tarsal conjunctiva (*t* Fig. 207 A). It is always most marked on the upper lid, which therefore must be everted in making the diagnosis of the trachoma.

The *second form* of hypertrophy is characterized by the presence of the trachoma granules. These are gray, translucent, roundish granules, showing through the most superficial layers of the conjunctiva, which they push up to form hemispherical swellings. On account of their translucent, seemingly gelatinous character, they have been likened to the eggs of frog-spawn or to grains of boiled sago. They are found principally in the retrotarsal folds (*f*, Fig. 207 A), in which they are imbedded in such numbers that, when the lower lid is drawn down, the fold projects as an elastic swelling, at the summit of which we sometimes see the granules arranged in rows like a string of pearls. To bring into view the granules in the upper retrotarsal fold, we push the fold down (§ 388). In the conjunctiva tarsi the trachoma granules are less readily visible. They are smaller in this situation, and cannot push the conjunctiva up because the latter is very closely adherent to the tarsus. Here, therefore, they generally appear as small, bright-yellowish points, which are situated deeply in the mucous membrane. Quite often, though, they are hidden from sight altogether by the development of the papillæ. Trachoma granules are often found in the semilunar fold, more rarely in other parts of the conjunctiva of the eyeball. This variety of proliferation of the conjunctiva is called the *granular* form.

The two forms of proliferation of the conjunctiva sometimes occur separately. In the great majority of cases, however, both are found at the same time in the same eye, and so distributed that in the conjunctiva of the lids the most prominent feature is the proliferation of papillæ, in the retrotarsal fold it is the formation of trachoma granules (Fig. 207 A). The conjunctiva of the eyeball is, in light cases, unaltered, but when the irritation is more intense shows a coarsely reticulate injection. In the fresh cases and in those attended with marked symptoms of irritation there is a purulent secretion. In older cases and in those which run a more sluggish course the secretion is very scanty.

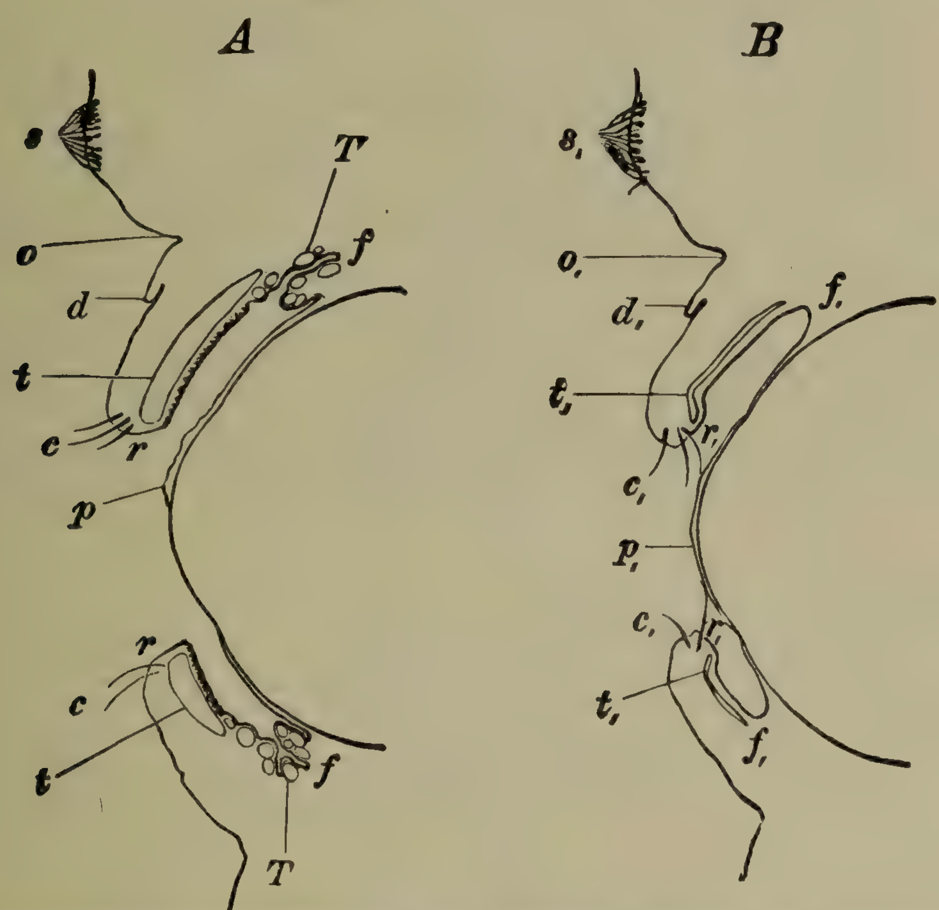

Fig. 207.—Schematic Section through the Lids and Eyeball (*A*, in Recent, *B*, in Old Trachoma).

A shows the way in which the two forms of hypertrophy of the conjunctiva are distributed among the separate divisions of the latter; *B*, the stage of sequelæ of trachoma; *s*, s_1, eyebrows; *o*, o_1, furrow between the brow and the lid (sulcus orbito-palpebralis); *d*, d_1, covering fold; *c*, cilia in their proper position; c_1, cilia turned toward the cornea; *r*, free border of the lid, with the borders of the upper and lower lids running parallel and the posterior margins of the lids sharp-edged, r_1, free border of the lid, looking backward, and with its posterior margin rounded; *t*, tarsus thickened by infiltration and covered with the velvety conjunctiva tarsi; t_1, tarsus thinned (atrophic), bent at an angle near its free extremity, and covered with smooth conjunctiva; *f*, fornix with numerous trachoma granulations, *T*, in the folds of the conjunctiva; f_1, fornix smooth, without folds (symblepharon posterius); *p*, thick pannus covering the upper half of the cornea; p_1, a shrunken pannus extending over the whole cornea.

The form with papillary hypertrophy (trachoma papillare, chronic blennorrhœa, ophthalmia purulenta chronica) and that with the formation of granulations (trachoma granulosum, trachoma verum, trachoma Arlti) were formerly regarded as distinct diseases.

The condition in which both changes were found was called trachoma mixtum.

The *papillary* growths which impart to the conjunctiva its velvety or raspberry-like appearance are caused by an increase in size of the surface of the hypertrophic conjunctiva. The latter is thrown into folds, between which correspondingly deep clefts are formed; then on cross section the folds appear under the form of papillæ (Fig. 208, *P* and P_1). The connective tissue forming the papillæ is stuffed full of round cells; the surface of the papillæ is covered with a very much thickened epithelium (*e*, *e*), which is continued on into the depressions (*t*, *t*) that exist between the papillæ. These depressions hence have in microscopical cross section the appearance of a narrow canal coated with

epithelium, and might easily be regarded as tubular glands. And in fact real, repeatedly branching glandular tubes do grow out from them and extend into the tissue of the conjunctiva.

Papillary hypertrophy of the conjunctiva, however, is by no means a characteristic feature of trachoma, in the sense of being limited to it alone. In a less marked degree it is found in connection with every long-continued irritation of the conjunctiva; as in chronic catarrh, in phlyctenular conjunctivitis that has lasted a long time, in ectropion upon the portion of the conjunctiva that is exposed to the air, after acute blennorrhœa when the so-called chronic blennorrhœa has developed from it, etc. Papillæ that are large but compressed and flat are the distinguishing mark of spring catarrh (see Fig. 215).

The *trachoma granulations* appear in microscopical cross sections under the form of rounded accumulations of cells, between which a very delicate connective tissue framework can be made out. The cells in the marginal portions of the granulations are lym-

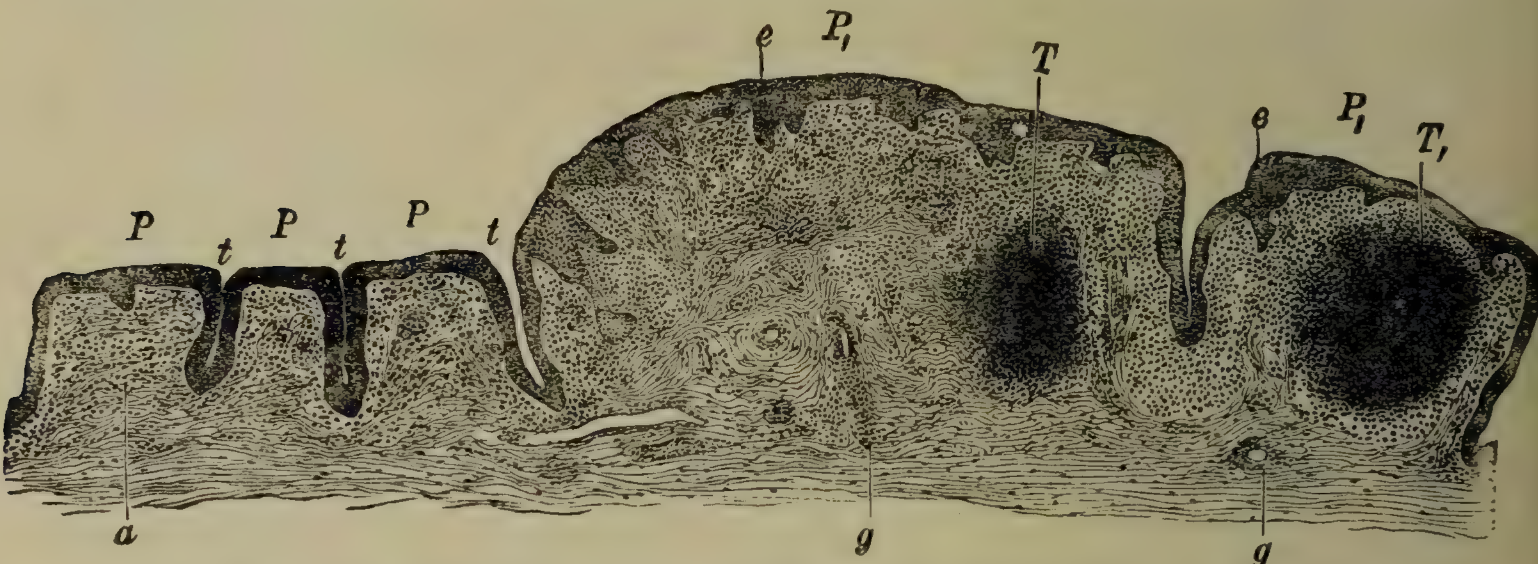

FIG. 208.—CROSS SECTION THROUGH THE TRACHOMATOUS CONJUNCTIVA OF THE UPPER LID. Magnified 24×1.

Both small papillæ, P, P, P, and large ones, P_1, P_1, are found. The former stand side by side like the pickets of a palisade; the depressions, t, t, t, lying between them and coated with epithelium, look like the tubules of glands. The large papillæ contain trachoma granules, T, T_1, which are not sharply limited and do not possess a capsule. The epithelium of the conjunctiva is in many places, e, e, thickened. The mucous coat is in a condition of cellular infiltration, a, which is especially marked in the vicinity of the blood-vessels, g. g.

phocytes; in its interior parts are predominantly uninuclear leucocytes (epithelioid cells), between which lie a few particularly large cells (phagocytes) which contain in their protoplasm small deeply-staining corpuscles; and in the connective tissue surrounding the granulation are found plasma cells. The trachomatous granulation either passes without any sharp line of demarcation into the surrounding tissue, which is also very rich in cells (Fig. 208, T and T_1), or there is, especially in the case of the older granulations, a sort of incomplete capsule of connective tissue (Fig. 210, k). In the later stages of trachoma these granulations disappear (§ 468).

The so-called *trachoma mixtum*, which clinical observation has already shown to be the most frequent form, is proved by microscopical examination to be almost the only form that occurs. That is, even in those cases in which to the naked eye papillæ alone appear to be present, trachomatous granulations are found in cross sections examined under the miscroscope lying in the tarsus either within the papillæ themselves or imbedded in the deeper portions of the mucous membrane. In the former case the papillæ have a particularly broad or even knob-shaped appearance (Fig. 208, P_1). In the second case the trachomatous granulations are concealed by the papillary bodies, beneath which they lie; then we often see them coming into view afterward when, as the result of prolonged treatment, the papillary growths have disappeared.

The *gelatinous trachoma* of Stellwag represents a later stage of mixed trachoma, in which a more uniform lymphoid infiltration exists in conjunction with superficial cicatricial changes. We have in that case a conjunctiva which is thickened, smooth on the surface, yellowish, and of gelatinous translucency. In this there is a more uniform lymphoid infiltration with superficial cicatricial changes.

468. Stage of Cicatrization.—In the subsequent course of the disease the hypertrophy of the conjunctiva gradually increases, growing steadily greater, until it has reached a certain height, which is not the same in all cases. Then

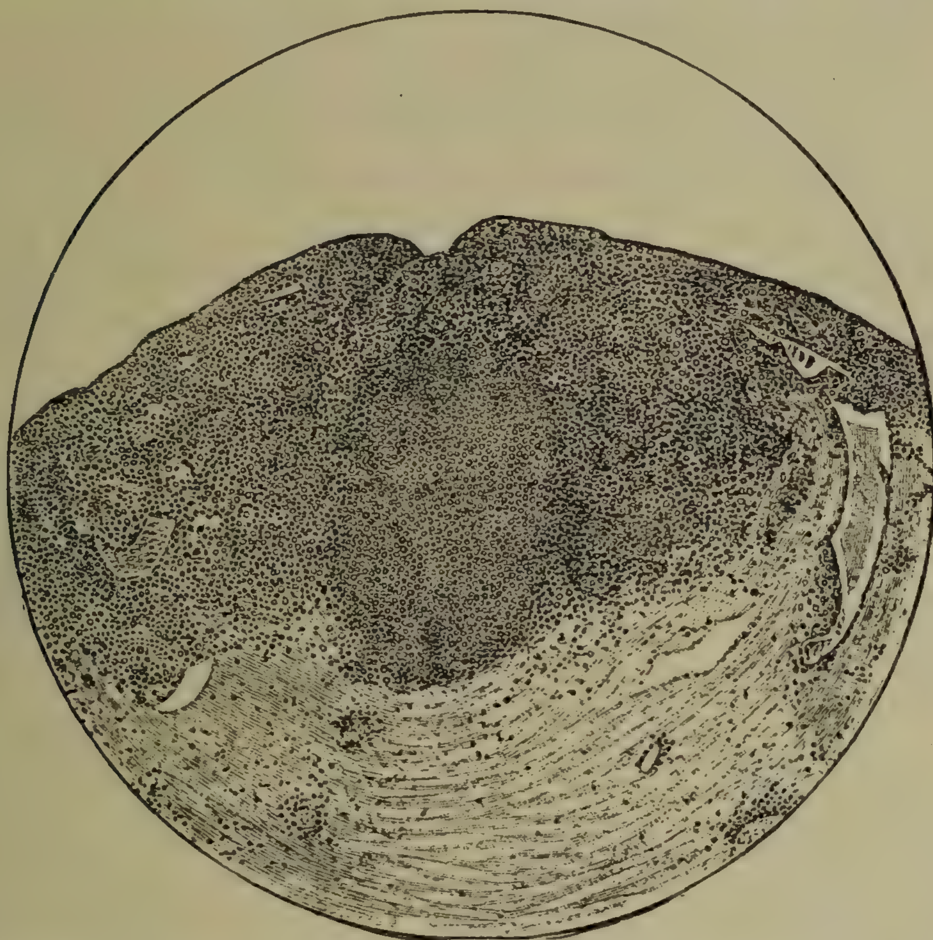

FIG. 209.—GRANULATION FROM THE RETROTARSAL FOLD OF A MONKEY. Magnified 52×1.

The monkey 32 days before had received inoculation from a case of ophthalmia neonatorum, the secretion of which contained no gonococci but did contain the inclusions which are like those occurring in trachoma and are the mark of inclusion conjunctivitis, (see § 477.) The granulation shows the same histological picture as a trachoma granulation in man. It is still recent and hence is not yet shut off by a capsule from its surroundings. The central lighter portion, which in the section forms a vertical oval, consists almost exclusively of epithelioid cells, while in the peripheral portions of the granulation the lymphocytes predominate. The epithelium of the conjunctiva over the granulation has been exfoliated, and in this spot there has been produced by disintegration of the tissue a depression, through which the granulation would perhaps have been evacuated later on.

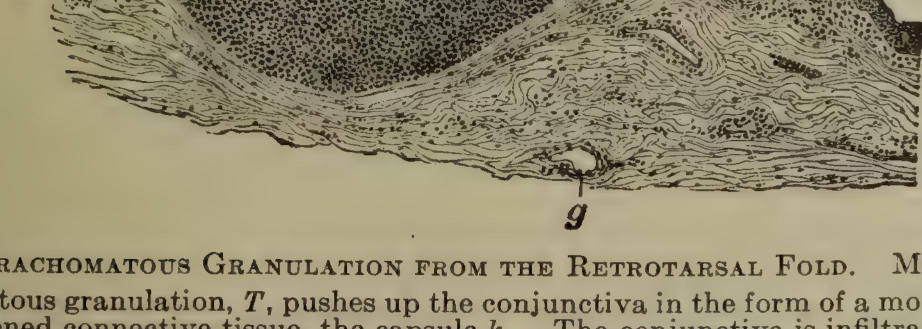

FIG. 210.—TRACHOMATOUS GRANULATION FROM THE RETROTARSAL FOLD. Magnified 24×1.

The trachomatous granulation, *T*, pushes up the conjunctiva in the form of a mound, and is inclosed by a layer of thickened connective tissue, the capsule *k*. The conjunctiva is infiltrated with cells, both in its upper layers, *a*, and along the vessels, *g*; the epithelium, *e*, shows, above the place marked *a*, bright spots which correspond to the goblet cells; at *d* it lines one of Henle's glands.

it disappears again, step by step, while a cicatricial state of the conjunctiva with contraction takes its place. In this way the trachoma is cured in the sense that the specific morbid process has come to an end. Nevertheless, the conjunctiva has not become normal again by any means; on the contrary, it

bears upon it lasting marks of the disease that has passed, namely, the signs of a cicatricial contraction which, in many instances entails other, additional, consequences, such as we shall group together under the phrase "the stage of sequelæ." The more considerable the hypertrophy of the conjunctiva the longer is the duration of the disease, which in most cases is counted by years, and the greater and more striking also is the subsequent contraction. The object of the treatment, therefore, must consist in checking the hypertrophy of the conjunctiva while it is developing, as thus both the duration of the disease is shortened and its evil consequences also are diminished.

In the *conjunctiva tarsi*, the beginning of the *formation of cicatrices* is betokened by a few narrow, whitish striæ (fine cicatrical bands), which we see emerging in the midst of the reddened and thickened conjunctiva. These striæ gradually become more numerous and unite to form a delicate network, the meshes of which are occupied by red islands—that is, by those portions of the conjunctiva which are still hyperæmic and hypertrophied. Little by little the cicatricial lines grow steadily broader and the islands that they inclose steadily narrower, until at length that condition is produced in which the conjunctiva of the tarsus has become perfectly pale, thin, and smooth. The cicatricial condition of the conjunctiva corresponds in extent and intensity to the amount of hypertrophy that has preceded it. In those cases in which the hypertrophy of the conjunctiva has attained a considerable height in certain spots only, it is also only at these spots that deep cicatrices remain after the trachoma has run its course, while those parts of the conjunctiva which were simply infiltrated, or were hypertrophic to only a very slight degree, return to the normal state.

In the *conjunctiva of the fornix* the same process occurs, but presents a somewhat different aspect. Here we do not see any whitish bands but we find that the thick swellings which are formed by the hypertrophic retrotarsal fold are becoming gradually thinner and flatter. Associated with this process, and proceeding with it step by step, is a condition of contraction taking place in the conjunctiva, a condition which steadily increases until even the folds that in the normal eye are present in the fornix are smoothed out and disappear (Fig. 207 B, at f_1). The conjunctiva has grown pale, and a delicate bluish-white coating is witness to the cicatricial character of its superficial layers.

The transformation of the conjunctiva into *cicatricial tissue* proceeds as follows: A part of the numerous cells which are contained in the conjunctiva, and which are either uniformly scattered through it or occur in circumscribed accumulations (trachomatous granulations), disappears by resorption; another part, owing to rupture of the granulations, empties externally; and still another part gradually grows into spindle-shaped cells, and finally into connective-tissue fibres. This new-formed connective tissue shrinks extremely, so that the conjunctiva contracts and becomes thinner and of tendinous character. We have here a process similar to that wich occurs in cirrhosis of the liver—i. e., the shrinking of a new connective tissue which has developed out of an inflammatory infiltration. It would be a mistake to suppose that in the trachomatous conjunctiva there are raw spots which become covered with a cicatrix—a mistake into which we might be more apt to fall because of the term granulations. What we call granulations in trachoma have nothing at all in common with the granulations of wounds, except their external appearance.

In the cicatricial trachoma, a linear cicatrix which runs parallel to the lid margin is often apparent on the conjunctiva of the upper lid. This has a varying significance. In the majority of cases it lies quite close to the margin of the lid (Fig. 211 B); it then corresponds to the sulcus subtarsalis where the infiltration has gone more into the deeper parts (§ 471). In the case figured there was also cicatricial formation at another place of predilection, namely, at the convex border of the tarsus. In less frequent cases the cicatricial stria lies higher up (Fig. 211 C). It then represents the boundary line between the conjunctiva of the tarsus and of the fornix. The tarsal conjunctiva is shrunk so as to cover but a small area, and the conjunctiva of the fornix has been drawn up in part upon the tarsus. It of course is not intimately adherent to the tarsus; and thus is explained the fact that in such cases the tarsus can readily be shelled out from the conjunctiva—a thing which is impossible when the conjunctiva is normal. This, in fact is an operation that is done in cases of old trachoma. (See § 929.)

The *tarsus* in recent cases is thickened by the deposition of numerous lymphocytes.

A B C

FIG. 211 A.—NORMAL UPPER LID, EVERTED. The cilia jut out from the anterior edge of the border of the lid in several rows. The posterior edge of the border of the lid is sharp; the shadow directly beneath it represents the shallow groove of the sulcus subtarsalis. The upper convex border of the tarsus now looks downward and is stretched into a straight line, while the straight lower border of the tarsus now has an arched course. Through the conjunctiva are indistinctly seen the Meibomian glands.

FIG. 211 B.—UPPER LID IN OLD TRACHOMA, EVERTED. Cicatrix in the sulcus subtarsalis. The tarsal portion of the lid is somewhat lower than in the normal lid. Near the posterior edge of the lid margin and parallel to it runs a longitudinal cicatrix from which minute bands pass upward. At the convex border of the tarsus a cicatrix formation of slighter degree is present.

FIG. 211 C.—DRAWING OF THE RETROTARSAL FOLD UP OVER THE TARSUS. As a result of shrinking, the lid is not only lower but has also become shorter horizontally, so that it looks as if it were derived from a smaller individual than the normal lid. The posterior edge of the lid margin, owing to the shrinking, has been flattened out from a right to an obtuse angle, and has ceased to be sharp. The tarsal surface of the lid is divided into two parts by the cicatricial line which runs parallel to the lid margin. The lower part adjoining the lid margin is covered by the shrunken tarsal conjunctiva, which shows minute vertically running vessels and folds of similar course. The part lying above the cicatricial line is covered by the retrotarsal fold which has been drawn up upon the tarsus. The convex border of the tarsus, which looks downward, shows in its centre a bulge such as frequently occurs in trachoma.

Later on it shrinks, its glands (both Meibomian and acinous glands) disappearing and nothing being left but a tough connective tissue, which contains very few vessels and which sometimes is converted to a large extent into fatty tissue.

469. Complications.—The disturbance of sight, of which many patients complain, is founded upon a complication affecting the cornea, and appearing under two different forms, pannus and ulceration, which very frequently occur together.

Pannus [9] *trachomatosus* consists in the deposition upon the surface of the cornea of a newly formed, gelatinous, vascular tissue, which pushes its way from the edge toward the centre of the cornea. At the spot where the pannus is located, the surface of the cornea is uneven and studded with fine projections, and there is a gray, translucent, superficially situated, cloudy mass, which is traversed by numerous vessels. The latter spring from the vessels of the conjunctiva, which pass over the limbus and out upon the cornea and, after arriving within the pannus, branch in an arborescent fashion (Fig. 231). Pannus ordinarily begins its development at the upper margin of the cornea, although it may also begin below or at one side, and

[9] *Pannus*, a cloth.

covers first the upper half of the latter (*p*, Fig. 207 A). Quite often it terminates below in a sharp, straight, horizontal border. Afterward pannus develops at other portions of the corneal margin, until at length the entire cornea is covered by it. When pannus is pretty fully developed, the iris likewise participates in the inflammation (iritis). Disturbance of vision sets in as soon as the pannus has advanced into the pupillary area of the cornea. If this region is entirely covered by pannus, vision is reduced until it is limited to the recognition of large objects, or even to the mere ability to distinguish between light and darkness. Pannus, provided the further changes, described below, have not occurred in it, is capable of complete retrogression, so that the cornea can reacquire its normal transparency.

FIG. 212.—CROSS SECTION THROUGH THE MARGIN OF A CORNEA AFFECTED WITH PANNUS. Magnified 125x1.

Beneath the epithelium, *E E*, is the limbus, *L*, greatly thickened by cellular infiltration; from it the pannus, *P*, in which are perceived the cross sections of several vessels, extends between the epithelium and Bowman's membrane, *B*, over the cornea, *C*. *S*, sclera.

Pannus is a layer oi new-formed tissue, which, starting from the limbus, spreads over the cornea (Fig. 212, *P*). It is a soft tissue extremely rich in cells, and greatly resembling the infiltrated trachomatous conjunctiva. This tissue abounds in vessels, and occurs in alternately thicker and thinner layers, for which reason the pannus looks uneven and nodulated. Pannus, when it is quite superficial, insinuates itself between Bowman's membrane (Fig. 212, *B*) and the epithelium (Fig. 212, *E*), the latter being thus lifted off from Bowman's membrane and made to cover the pannus. In such a case it is possible for the cornea to regain completely its normal structure and transparency after the resorption of the pannus, since then the epithelium is once more directly applied to Bowman's membrane. But when the pannus lasts for a long time it destroys Bowman's membrane, and this is still more true of the less frequent cases in which the pannus from the very outset penetrates beneath Bowman's membrane. (According to Ritter and Rählmann it regularly does this.) Later on, the most superficial layers of the cornea are infiltrated with a tissue abounding in cells. Then the complete restoration of the transparency of the cornea has become impossible.

A rare metamorphosis that has been observed in pannus is a process of *sclerosis* in which there develops from the pannus a dense white or yellowish tissue containing very few vessels. This tissue resembles a dense scar, e. g., such as occurs after deep ulcers of the cornea, but, unlike the latter, replaces only the superficial layers of the cornea—those extending, for example, from the upper border to the centre of the cornea, if the pannus itself had covered the upper half of the latter. Another change in old pannus consists in the development of small, intensely *white spots*, which frequently form a group in the pupillary region of the cornea. The appearance of the spots, which lie close to the minute blood-vessels of the pannus, reminds one of lead incrustation. The spots are superficial in seat, and may be removed by scraping (§ 611).

Pannus consists primarily in an infiltration of the *upper part of the limbus*. That

the morbid process extends from this point into the cornea and not peripherally is due to the circulatory and nutritive conditions which obtain in this region (§§10, 72) and which cause other inflammatory processes too to spread from the limbus into the cornea and not into the conjunctiva scleræ. The fact that the pannus begins in the limbus and usually, moreover, in its upper part is obviously due to contact infection, since it is just this part that even in the daytime is constantly in contact with the diseased conjunctiva of the upper lid. The roughness of the palpebral conjunctiva is a contributory factor, since it causes injury to the cornea in a mechanical way.

For *pseudo-pterygium* see § 504.

The *ulcers of the cornea* either develop at a spot which is otherwise normal, or they occur in connection with pannus. In the latter case they are found principally at the free border of the pannus, more rarely within the latter. For a more detailed account (§§ 537 et seq.). Ulcers heal, leaving behind them cloudy spots, the influence of which upon vision is dependent upon the degree of their opaqueness and also upon their situation with regard to the pupillary area of the cornea.

Trachoma is also frequently complicated with infection of the lacrimal sac. Such sacs are very friable and rupture easily (Butler).

470. Course.—The beginning of the disease shows a clinical picture which varies with the inflammatory symptoms accompanying the fundamental lesion. In the majority of cases, the disease sets in with moderate symptoms of irritation—photophobia, lacrimation, pain—which augment with the increase in the objective changes. Not infrequently, however, trachoma develops so *insidiously* that for a long time those whom it has attacked are not aware of it. In places where trachoma is endemic this seems to be the rule. Such persons sometimes do not have their attention called to their disease until the pannus as it covers the cornea begins to disturb their sight. These cases belong as a rule to the granular form of trachoma. When the inmates of barracks, schools, etc., that are infected with trachoma undergo medical examination, there are always found a number who do not complain of any troubles whatever and who regard themselves as perfectly healthy, while examination shows in the retrotarsal folds a very considerable development of trachoma granules. In contrast with cases running this insidious course are the caes of what is called *acute trachoma.* In these the disease begins with very violent inflammatory accessories; the œdema of the lids, the great swelling of the conjunctiva, the profuse purulent secretion would almost lead us to imagine the case to be an acute blennorrhœa. The correct diagnosis can be made as a rule by our finding the conjunctiva studded with numerous trachoma granules. But if these are absent during the first days of illness, or if, because of the great swelling of the conjunctiva, they are not apparent, the subsequent course of the disease may be the only thing that can clear up the nature of the latter; which it does, since the hypertrophy of the conjunctiva, that is characteristic of trachoma, soon develops. Such acute cases occur chiefly during the prevalence of an epidemic of trachoma, and they are particularly contagious because of the profuse secretion. They are dangerous to sight not so much on account of pannus as of corneal ulcers, that make their appearance during the acute stage.

These acute cases are rare. Trachoma is essentially a chronic disease which only exceptionally begins with great inflammation. The majority of the acute cases, in fact, are not simple trachoma, but trachoma to which is superadded a second infection with bacteria, which like the gonococcus or the germs causing catarrhal ophthalmia, can set up acute conjunctivitis (§§ 452 and 463).

The subsequent case of the disease likewise varies greatly. It depends on (*a*) how far the anatomical changes in the conjunctiva have advanced, and (*b*) to what extent they are combined with inflammatory accidents and complications of a similar character on the part of the cornea.

As regards the first point, in the slightest cases the hypertrophy of the conjunctiva amounts to but little, and the cicatricial contraction that is left by it is correspondingly insignificant; so much so, perhaps, that it may scarcely be possible any longer to make the diagnosis of trachoma, if some time has elapsed since it occurred. In other cases the disease gets well with comparatively slight scars, and this may even occur without any treatment, inasmuch as the hypertrophy of the conjunctiva attains only a moderate pitch and then spontaneously abates. It is not such a very rare occurrence to find in a case the characteristic scars of trachoma in the conjunctiva without the patient's remembering that he ever had any trouble with his eyes. But in other, severe, cases the hypertrophy of the conjunctiva constantly progresses and sometimes reaches an extraordinary pitch, and consequently, also, serious changes are left in the conjunctiva. Serious, too, must be called every case in which the cornea has become implicated. It must be remarked, however: (1) That the symptoms of irritation do not always by any means bear a fixed proportion in the objective changes; cases with very great hypertrophy of the conjunctiva and thick pannus often running their course without inflammatory accidents, and vice versa. (2) That similarly no fixed relation exists between the changes in the conjunctiva of the lids and those of the cornea. We see cases with very pronounced proliferation of the palpebral conjunctiva without pannus, and, on the other hand, cases with pannus and ulcers associated with a trifling affection of the conjunctiva. (3) In one and the same case the course is often very variable, in that sometimes intermissions or even spontaneous partial recoveries, sometimes relapses and exacerbations, occur. The latter are surely to be expected if, in a case that has been improved by treatment, treatment is too soon discontinued; but it is noticed that they also occur without any known cause under appropriate treatment properly carried out. Thus, a suddenly occurring supplemental attack of pannus can in a short time annihilate the results of months of treatment.

Owing to the ready transmission of infection from one eye to the other, trachoma is most always found in both eyes, although cases are occasionally seen in which it is confined to one.

471. Stage of Sequelæ.—It is only the lightest cases, or those that come under treatment early, that are completely cured. In other cases

there are left sequelæ, which are accompanied by a permanent impairment of the eye. These affect either the lids and conjunctiva or the cornea. They are as follows:

1. *Distortion of the lids* with faulty disposition of the cilia. The distortion is produced by the cicatricial contraction of the conjunctiva and the tarsus, as a result of which the tarsus bends in such a way as to be convex anteriorly. This distortion is recognizable even from an inspection of the lids while in situ, from the fact that they bulge more than usual. It appears still more clearly on everting the lids, especially in the upper lid, in which the distortion is always more pronounced. We find the conjunctival surface of this lid traversed by cicatrices, among which one that is particularly striking is a cicatricial band which runs in the form of a narrow white line some millimetres above the free edge of the lid and parallel with it (Fig. 211 B). Along this line there is a furrow-like depression produced by the drawing in of the conjunctiva and the tarsus. On everting the lids we feel that corresponding to this spot and lying, therefore, in the neighborhood of the free border of the lid, there is an angular bend of the tarsus (t_1, Fig. 207 B). From this bending of the tarsus the whole lid acquires a sort of boat-like or bowl-like shape.

The cause of the distortion of the tarsus lies partly in the cicatricial contraction of the conjunctiva; for, as the conjunctiva grows shorter upon the posterior surface of the tarsus, it tends to bulge the latter forward. But the distortion is mainly produced by changes in the tarsus itself. The latter is as much the seat of inflammatory infiltration in severe cases of trachoma as is the conjunctiva itself. It is hence increased in size and, when we evert the lid, we feel that it is thicker, wider, and at the same time less pliable, so that sometimes the eversion of the lids is rendered considerably more difficult. From such a state of things the experienced observer would infer that he has to fear a subsequent distortion of the tarsus with its consequences. The infiltration and thickening of the tarsus are greatest near its lower margin, along the line at which the blood-vessels passing to the conjunctiva from in front perforate the tarsus (see § 10 and Fig. 4, *rp*). There is no doubt but that it is chiefly along these vessels that the inflammatory infiltration makes its way from the conjunctiva to the tarsus. Hence, cicatricial contraction, which succeeds the infiltration and which makes the whole tarsus thinner and narrower, is greatest at this spot and produces there an angular bending of the tarsus, corresponding to which is the cicatricial line that is seen running horizontally upon the conjunctiva tarsi, and the position of which agrees in general with that of the sulcus subtarsalis present in the normal lid (Fig. 211 B).

The immediate consequence of the distortion of the lid is an alteration in the position of its free border and of the cilia springing from it. The free border of the lid no longer looks straight downward (or in the lower lid straight upward), but backward (inward). The internal margin of the lid, which in the healthy state is sharp, becomes rounded off and can no longer be distinctly recognized (Fig. 207 B, r_1), this being due partly to the way in which it is drawn by the contracting conjunctiva, partly to the pressure of the eyeball upon it. By the turning inward of the free border of the lid the direction of the cilia (c_1) is changed, so that they now no longer look forward but backward, and hence touch the surface of the cornea (*trichiasis*). Another factor besides the distortion of the tarsus that contributes to this false position of the cilia, is the tension which the contracting conjunctiva

exerts. This tends to draw the skin, and with it the cilia, over the free border of the lid and up upon the posterior surface.

If the distortion of the lid progresses, the entire border of the lid turns backward and *entropion* is produced (§ 438). In trichiasis and entropion alike there is a permanent condition of irritation, which is excited and maintained in the eye by the constant contact of the cilia with the cornea; if this condition lasts any length of time, diseases of the cornea make their appearance as a consequence of the mechanical injury produced by the cilia (see §§ 537 and 605).

The opposite kind of anomaly of position of the lid—that is, its turning outward, or *ectropion*—also occurs as a result of trachoma. The cause of this is that the conjunctiva, when it is thickened and has undergone great proliferation, crowds the lid away from the eyeball; the contraction of the muscular fibres of the orbicularis then suffices to complete the eversion of the lid. This kind of ectropion is usually found only in the lower lid (see § 439).

2. *Symblepharon Posterius.*—When the cicatricial contraction of the conjunctiva reaches a high degree, the folds of the region of transition flatten out completely; the conjunctiva passes directly from the lid to the eyeball (f_1, Fig. 207 B). If the lower lid is drawn down with the finger, the conjunctiva stretches tightly in the form of a vertical fold between the lid and the eyeball, and if the lid is drawn down still farther, the eyeball, being fastened tightly to it by the conjunctiva, must follow. This condition is characterized as symblepharon posterius (see § 505). In particularly severe cases the lower half of the conjunctival sac is reduced to a shallow groove between the lid and the eyeball.

3. *Xerosis Conjunctivæ.*—This condition develops when the conjunctiva, owing to excessive atrophy, loses its secretory functions. A steady diminution in the lacrimal secretion, which occurs at the same time, contributes to the production of the xerosis. Xerosis manifests itself by the following signs: The secretion, formerly copious, now becomes steadily scantier and assumes a tough, sticky, viscid character. In conjunction with this, a feeling of dryness develops in the eye. Subsequently there appear on the conjunctiva tarsi several dry-looking places, to which the lacrimal fluid cannot adhere any more than if they were smeared with grease. This condition tends to spread, until finally the conjunctiva may be affected by it throughout its whole extent. The cornea, which as a rule has become partly cicatricial from previous pannus and ulcers, likewise suffers from the deficient moistening; its epithelium becomes thicker, epidermoid, dry upon the surface, and hence opaque. Thus is produced that melancholy condition which is called *xerophthalmus* and which forms the worst termination of trachoma; the eye is rendered incurably blind, is disfiguring to its possessor, and in addition keeps torturing him with a constant, very tormenting sense of dryness (see § 507).

4. *Corneal Opacities.*—These are after-effects, both of ulcers of the cornea and of pannus. A recent pannus, it is true, can disappear completely by a process of absorption, so that the cornea reacquires its normal

transparency. But often further changes take place in the pannus, which render its complete disappearance impossible. Among them is to be reckoned in the first place (*a*) *the transformation into connective tissue*, which the pannus undergoes if it lasts for a long time. In this the same change takes place in the pannus as in the trachomatous conjunctiva, a portion of the cells of which the pannus is composed growing into spindle-cells and finally into connective-tissue fibres. As a consequence of this the pannus becomes thinner, its surface grows smooth, the vessels with a few exceptions disappear, and at length the pannus is transformed into a thin membrane of connective tissue which covers the surface of the cornea and hardly admits of further resorption. In cases in which the pannus is quite thick and succulent and covers the whole cornea, (*b*) *ectasia of the cornea* sometimes results. That is, as the tissue of the pannus, which is soft and abounding in cells, penetrates more deeply into the cornea, the tissue of the latter softens and gives way before the intra-ocular pressure (keratectasia e panno). Such a cornea never becomes perfectly clear again. The same thing is true, finally, of those cases in which (*c*) pannus is complicated with *ulcers;* the regions which are occupied by the latter likewise have permanent opacities left upon them

Trachoma, then, is a disease which is distinguished by its duration, extending over years, and which in many cases renders those who are attacked by it half or wholly blind. If we add to this the fact that because of its infectious nature it is exceedingly apt to spread, we shall understand how, for those regions in which it is endemic, it is a veritable scourge.

472. Etiology.—Trachoma originates exclusively in infection proceeding from another eye affected with trachoma. Infection takes place by transfer of the secretion; contagion by means of the atmosphere, the existence of which was formerly accepted, seems not to occur. In all probability the secretion owes its infectious character to a micro-organism, as to whose nature, however, the investigations have not as yet been concluded. Since it is the secretion alone that transmits the infection, the danger of infection, which any given case carries with it, is in direct proportion to the amount of the secretion; the more profuse the latter, the greater being the danger to those in the immediate neighborhood of the patient. Ordinary trachoma which is attended with very little secretion, is in all probability but slightly contagious. We often see families, some member of which suffers from trachoma and yet fails to infect the others with whom he is all the time brought into intimate contact—and that too, when the commonest hygienic precautions are entirely neglected. It seems probable that something must be added to chronic trachoma to make it actively contagious, and that in most cases this something is an intercurrent conjunctivitis which furnishes the secretion that acts to carry the contagium and which also at times serves to produce an acute exacerbation of the trachoma (§ 470). The transfer of the secretion from one eye to another generally takes place indirectly through the medium of the finger or very frequently through the medium of any article which, like sponges, towels, handkerchiefs, etc., is brought into contact with the eyes in making the toilet. A special opportunity for this

to occur is afforded when a pretty large number of people have their sleeping apartments in common, and so, too, make common use of the articles above mentioned. Hence, trachoma spreads most extensively in barracks, penal establishments, poorhouses, boarding schools, orphan asylums, day schools of all kinds, etc. Moreover, outside of such institutions the same factor asserts itself, trachoma mainly attacking poor people who live crowded close together and bestow little care upon cleanliness. When conditions of environment favor, however, it may occur among those living an isolated life (e. g., in the country districts in Australia). Irritative conditions, like smoke, dust, etc., are predisposing causes since they not only cause congestion of the lids and sponginess of the adenoid tissue, but also induce the patient to rub his eyes frequently. Trachoma, finally, varies in its geographical distribution. It is most frequent in Arabia and Palestine, and in Egypt, which is regarded as its proper home (ophthalmia Ægyptiaca, Egyptian ophthalmia). In these countries scarcely anyone is free from it. In Europe it is much more wide-spread in the east than in the west. It is extemely prevalent in central Asia, very common in China and Japan, and only moderately prevalent in the East Indies.

In this country it is common in our eastern cities, especially among the foreign population and most of all among the Russian and Polish Jews and immigrants from Eastern Europe generally and among the Irish and Italians. It occurs frequently, too, among the Chinese and Japanese. It is also common, however, and often in a severe form, among our native Americans, particularly in some parts of the Middle West (Illinois, Kentucky, West Virginia), but it is rare among the native whites of the Southern States. It is prevalent among the Indians, while negroes, in this country at least, and in Cuba, are almost exempt (Swan Burnett and others).

It seems probable that this exemption of the negroes, especially the full-blooded negroes, and of the gypsies and the Ceylonese is due to a real racial immunity, which, to be sure is not absolute. Attempts, to explain it as due to peculiarities of environment fail, when applied to the conditions obtaining among the negroes in the cities of our Southern States (Bruns, Calhoun), (cf. the relative immunity of the negro to folliculosis—§ 458). The Canadian Indians (as opposed to the Indians of the United States, who are racially different) and the Esquimoes are even more immune (Yarn). On the other hand, the Chinese, Japanese, Jews, Arabs, Egyptians, Poles, and Irish seem specially susceptible.

In Europe it is very frequently found in the low lands (Belgium, Holland, Hungary, and the whole region of the lower Danube), and it was formerly thought that elevated regions were almost entirely free from it. This is true of Switzerland and the Tyrol; but it is very prevalent in the Caucasus and in the mountainous parts of West Virginia and Kentucky.

It has been thought that trachoma is rare in very young children, but in the Near East it is common even in infants, although usually in benign forms, complications being infrequent (Butler, Friedenwald).

Trachoma has been more or less *endemic* in *Europe* since antiquity. It is mentioned in Aristophanes, in the Ebers papyrus, and in a pseudo-Hippocratic manuscript. Celsus gives a good description of the roughness of the lids and the purulent discharge that it

ccasions. For treatment the ancients employed scarification of the conjunctiva, which s still to-day made use of by some, and which was accomplished by means of various nstruments and also by friction with fig leaves.

But trachoma first obtained particularly *wide prevalence in Europe* in the beginning f the nineteenth century. The French and British armies in Egypt were taken down with it, so that a very large proportion of the soldiers were affected, and when returned to their homes caused a rapid spread of the disease which was hence called ophthalmia militaris. Added to this in the case of the Russian, Prussian and Austrian armies was the enlistment of infected recruits from the eastern countries of Europe, where the disease had always been prevalent (Boldt). So it happened that when in the Napoleonic wars the armies came repeatedly in contact with each other and with the civil population, the disease became more widely disseminated and occurred in epidemics. In some countries it became frightfully prevalent. In the English army, during the year 1818, there were more than 5,000 on the invalid list, who had been rendered blind as a consequence of trachoma. In the Prussian army, from 1813 to 1817, 20,000 to 30,000 men were attacked with it; in the Russian army, from 1816 to 1839, 76,811 men were subjects of the disease. In Belgium, in 1840, one out of every five soldiers was affected with trachoma. The French army, which was formerly supposed to form the starting-point of the disease, was just the one that, relatively speaking, was least attacked. The armies disseminated trachoma among the civil population through the discharge of soldiers affected with eye diseases, through the quartering of troops, etc. When they had so many trachomatous soldiers in the Belgian army that they did not know what to do, the Government applied to Jüngken, who had then great repute as an ophthalmologist in Berlin. He recommended them to dismiss the trachomatous soldiers to their homes. By this fatal measure trachoma soon became diffused in Belgium to an extent observed in no other European state.

Among the *civil population* trachoma finds a favorable soil for its dissemination in places where many men dwell together, hence, among the poorer classes, but particularly in large public asylums. If trachoma has made its way into such an establishment, and no measures are taken against its spreading, soon a great number or even all the inmates will be attacked by it. On board ship, where the crew live so closely crowded together trachoma can spread very quickly. Mackenzie tells the story of the epidemic which raged upon the French slave ship Rodeur in the year 1819. The disease broke out, during the voyage, and first among the negroes who, to the number of 160 were crowded together in the hold. When they took the unfortunate people up on deck, because the fresh air seemed to have a favorable influence on the ophthalmia, many threw themselves overboard, so that they had to desist from doing this. Soon one of the sailors also was attacked, and three days later the captain and almost the whole crew were taken down with the disease, so that it was only with the greatest difficulty that the ship could be brought to port.

According to the descriptions of that time, trachoma then ran a very acute course, and was attended with profuse secretion, circumstances which explain the rapidity with which the disease spread and the frequency with which it ended in blindness due to suppuration of the cornea. Now that epidemics have ceased, these acute cases are rare, and the disease has become comparatively benign. At present trachoma exists n many countries as an endemic disease but nearly always as a chronic form. At the same time its prevalence has diminished, especially in central and western Europe. In eastern Europe it is still very common and in Egypt and Palestine, it is scarcely possible to find a native who has a normal conjunctiva.

Trachoma, therefore, not only appears under a varying clinical aspect, sometimes acute and threatening, sometimes chronic and mild, but it also seems to have *changed its character in the course of time*. The explanation of the way in which this has occurred was afforded by the study of trachoma in its native land, Egypt, where ophthalmia Ægyptiaca still rages as in the olden time. Investigations have shown (Müller, Morax) that in Egypt almost every native suffers from trachoma, with which as a rule he has been infected already in childhood. This runs just as chronic a course as with us and very frequently produces pannus. But, in addition, many of the natives are attacked during the hot season by an acute inflammation of the eyes, in the secretion from which there is

found one of the germs causing acute conjunctivitis, and most often the Koch-Week bacillus. It is cases of the former kind that by causing suppuration of the cornea pro duce the blindness that is so prevalent in Egypt. Now all these different inflammation of the eyes were lumped together under the name of Egyptian ophthalmia and wer regarded as identical with trachoma, while as a matter of fact, only the chronic cases ar pure trachoma and most of the acute cases are mixed infections. A knowledge of thi fact leads us to suppose that in Europe, too, during the great trachoma epidemics, wit their cases that ran such an acute and dangerous course, it was generally a question o *mixed infections*, being so either from the outset or from the subsequent superaddi tion of a second infection (Morax). However, the existence of an acute inflammatio in pure trachoma cannot be altogether excluded. The transfer of a pure trachomatou secretion to man does sometimes set up trachoma with acute inflammatory symptom (Addario, Greef).

Halberstaedter and Prowazek regarded as the *exciting cause* of trachoma the cel inclusions (see § 477), which they found in the trachomatous conjunctiva and which the regarded as a parasitic organism. But secretion from conjunctivæ, whether trachoma tous or not, containing these structures, does not if inoculated in the higher monkey produce trachoma but a transient conjunctivitis. Moreover, these structures do no occur in all cases of trachoma and they do occur in a benign conjunctivitis (inclusio conjunctivitis), which clinically resembles acute trachoma (see § 477), but which clears u without leaving any scarring or other permanent changes in the conjunctiva. It seem certain therefore that these inclusions are not the cause of trachoma and, according t Noguchi and Cohen when found in trachoma they are but the evidence of a complicatin inclusion conjunctivitis.

473. Differential Diagnosis.—There are several conditions resemblin trachoma, particularly in the formation of granulations. Chief among thes are (1) *follicular catarrh* (see § 458).

The two diseases are very similar, in that lymph follicles occur as characteristi formations in both. In follicular catarrh they are smaller, are more sharply limited, an project farther above the surface of the conjunctiva; in trachoma they are larger, desti tute of sharp outlines, and less prominent. Follicles proper are often oblong-ova (cylindrical) and placed side by side, in a row like a string of pearls, while trachomatou granulations are round and more rarely present any such arrangement in rows. Bu these characteristics are sometimes so obscured that even experts cannot in many cases make the diagnosis with certainty, and the subsequent course of the disease alone afford the desired information. Even in the histological structure no thorough-going distinc tion can be found between follicles and trachomatous granulations. A further resem blance between follicular catarrh and trachoma consists in the fact that they bot chiefly occur among bodies of men who are confined in a small space. It is therefor easy to understand that these two diseases have repeatedly been confounded with eac other; and a number of authors, in fact, explain follicular catarrh as being a kind o trachoma distinguished by its mildness and freedom from danger. But follicular catarr in certain ways differs radically from trachoma. In the first place, the cell inclusions, s frequent in trachoma, do not occur in follicular catarrh. In the second place, the latter in some cases at all events—e. g., in the form produced by atropine (see § 126)—ma arise without any contagion whatever, while trachoma is always conveyed by contagion A final and more important distinction between the two diseases is the course. Folliculа catarrh is not associated, or is associated to only an inconsiderable degree, with papillary hypertrophy of the conjunctiva; it never leads to shrinking of the conjunctiva, to pannus, or to any of the other sequelæ; it is a disease perfectly devoid of danger, one which, even without any treatment, finally gets well and leaves no trace behind; so that on this account alone the differentiation of the two diseases is not only theoretically, but also practically, of great importance.

If a physician in any case is doubtful whether he is dealing with a trachoma or a follicular catarrh—a distinction which often cannot be made in the first examination,

specially in cases of acute onset—he must denote the case as suspected trachoma, must ake the steps necessary to avoid the spread of the disease, and must begin the treatment. f this treatment in a few days leads to rapid decline of the inflammation and diminution n size of the granulations, the case is one of follicular catarrh, since a trachoma would be nuch more obstinate. (It must be admitted, however, that in follicular conjunctivitis he follicles are also often very persistent.) The right diagnosis can also be made if it s possible to demonstrate the presence of cell inclusions, which would indicate trachoma ather than a follicular conjunctivitis.

Probably we must regard the formation of granulations composed of adenoid tissue 1ot as anything at all specific, but only as a particular form of reaction, which the con- junctiva of the eye, like other mucous membranes, exhibits toward many different sorts of irritants. Such a formation of granulations occurs in its most pronounced form in rachoma and in follicular catarrh. It may also occur in the following conditions:

(2) Certain cases of *tuberculosis* of the conjunctiva (§ 495), which begin with an abundant development of granulations, quite as in trachoma.

(3) Certain conjunctival affections, regarded as *syphilitic*, in which there is an abundant formation of granulations (Goldzieher and Sattler).

(4) Cases in which nodules resembling follicles have *formed round small foreign bodies*, as when plant hairs or caterpillar hairs have gotten into the eye and penetrated into the tissue of the conjunctiva.

(7) *Parinaud's conjunctivitis* (see § 477).

(6) *Sporotrichosis* and *blastomycosis* of the conjunctiva (see § 477).

(7) *Inclusion conjunctivitis* and *desquamative epitheliosis* (see § 477).

474. Therapy.—The treatment of the trachomatous *conjunctiva* has a twofold object in view; on the one hand, it seeks to do away with the inflammatory complications and the increase of secretion, which is associated with them; on the other hand, to further the disappearance of the conjunctival hypertrophy. In this way it is most likely that the process of shrinking in the conjunctiva will be restricted as much as possible, so as to avert the evil consequences resulting from its cicatricial contraction. We attain both objects by the proper employment of caustics, of which two are almost exclusively in use: nitrate of silver in 2-per-cent solution and sulphate of copper in the form of a stick. The silver has the feebler action, and is therefore borne better; copper, being applied in substance, has a correspondingly stronger cauterant action, but also causes more irritation (§ 465). These remedies are, as a rule, applied once a day, it being only in severe cases that they are used twice a day. The indications for the two remedies are as follows: Nitrate of silver is employed in all recent cases with violent inflammatory symptoms and great secretion. It can also be used when there are ulcers upon the cornea that are still in the progressive stage, if we take care that none of the solution comes into contact with the cornea itself. Copper sulphate is suitable for those cases in which the inflammatory symptoms are small, and its chief use is in removing the hypertrophy of the conjunctiva. For this purpose it acts much more energetically than the silver solution, and should therefore be used in preference to it in all cases in which its application is allowable at all. Great inflammatory irritation, and particularly the presence of ulcers of the cornea in a state of active progress, contraindicate its use.

From these indications it follows that, as a rule, we treat a recent case with the silver solution until the inflammatory symptoms have disappeared and the secretion has diminished. As soon as this has occurred—and

several weeks are generally sufficient for the purpose—we replace the silver solution by bluestone. At any rate, we must avoid using the silver solution for too long a time on account of the argyrosis which may develop as a result of it. The copper is now to be used, the application of it being made stronger or weaker according to the degree of hypertrophy, and is to be kept up for months and even years until every trace of hypertrophy has vanished and the conjunctiva has become free from congestion and smooth throughout. At first the application is made every day; but when only slight remains of the hypertrophy exist, it is sufficient to make the application every other day, and subsequently every third day; and at this stage, the milder acting alum pencil (a sliver of alum whittled down to a fine edge) may be substituted for the bluestone. Moreover, the application should be made less and less energetically all the time, until finally, when the cure of the trachoma is complete, the application is entirely suspended. In these later stages of the disease we can instruct the patient how to evert the lid himself and touch it with the bluestone, so that he need not come so often to the physician. Or, we may prescribe for him an ointment of copper sulphate (½ to 1 per cent) or of copper citrate (10 per cent; Arlt, Jr.) which he himself can rub into the conjunctival sac. Another effectual substitute for the copper stick is a 10-per-cent solution of copper sulphate in glycerin. This is diluted with from 15 to 60 parts of water, and the mixture is dropped into the eye three or four times a day. The aqueous mixture must be made up fresh daily, and its strength increased as fast as the patient can stand it (Prince). A 12-per-cent solution of glycerite of tannin may at times replace the copper treatment (Stuckey and Hughes). When there is great cicatricial contraction of the conjunctiva the bluestone is not applicable at all, and must be replaced by ointments. A 1-or 2-per-cent ointment of white or yellow mercurial precipitate (the latter acts more energetically) may be rubbed into the conjunctival sac. In relapses with great inflammatory irritation, such as often occur in the course of the treatment, the copper is always to be replaced for a short time by the silver solution. If, however, the symptoms of irritation are very violent, the silver solution itself cannot always be borne, and must then be replaced for some time by milder remedies, such as instillations or compresses made with weak solutions of corrosive sublimate or boric acid.

Nicola has obtained good results, especially in cases with marked granulations and pannus, by the daily application of a ferment obtained from the Nepeta citriodora combined with resin and glucosides derived from thyme and sage.

The *operative treatment* of trachoma is indicated in cases in which very numerous granulations are present in the retrotarsal folds. In recent cases with abundant, succulent granulations, we remove the latter by curetting, or the galvano-cautery or, better and more thoroughly, by expression or brossage (see § 912). Cauterization with carbon dioxide snow has also been used with success (Butler).

Keining's method of daily repeated friction of the conjunctiva with a 1:2,000 sublimate solution or the massage of the conjunctiva with a rounded

glass rod is also efficient, mainly owing to its mechanical effect—i. e., to its causing expression of the granules. A sublimate solution of 1:500 may be used, rubbed well into the retrotarsal folds. It causes no more irritation than the copper stick and by many is held to be at least equally effective.

None of the mechanical measures should be applied, if there is any evidence of considerable inflammatory irritation of the eye, such as marked redness and swelling of the conjunctiva and secretion in corresponding amount. If these are present, they are to be allayed by a sufficiently prolonged treatment with the silver solution before any operation is done.

Neither an immediate nor a radical cure of trachoma is effected by these mechanical means, since along with the larger granulations, small ones in process of development are always present, which cannot be removed, and which grow bigger afterward. Hence it is necessary after the reaction, produced by the operation, has subsided to apply caustics in the usual way. But it must be conceded that in suitable cases the duration of treatment is considerably shortened by expression and similar procedures.

Phototherapy and actinotherapy have been used for removing the granulations, but the good results thus obtained are said not to be permanent.

The treatment of trachoma must be kept up until the hypertrophy of the conjunctiva is completely done away with, as otherwise relapses are to be looked for sooner or later. The chief difficulty in the treatment lies in its great length, it often requiring many months for a complete cure. Those patients who have not the endurance nor the means necessary for such a course, give up treatment as soon as their subjective troubles have disappeared, without, however, being completely cured. Then we commonly see them returning after some time with a relapse, which is often more severe than the disease for which we originally treated them. This lack of completeness in the treatment is the reason why the disease with many men drags on through their whole life.

To avoid this protracted and incomplete treatment and particularly to prevent complications, many advocate the early performance of a radical operation (excision of the tarsus and infected conjunctiva—see §§ 918, 929). This seems indicated, at all events, whenever the disease shows no prospect of yielding to the milder measures already outlined or when pannus or other threatening complications are setting in. In children, however, the radical operation should not be done, unless absolutely necessary.

475. Treatment of Complications and Sequelæ.—The treatment of complications affecting the cornea is conducted on the principle that the affections of the cornea, caused by a conjunctival trouble, are best cured by the treatment of the conjunctival trouble itself. Hence, *ulcers of the cornea* when occurring in connection with trachoma are not combated directly, but have their cure brought about by means of applications made to the conjunctiva. The only limitation to this is that, where there are corneal ulcers in active progress, the silver solution is demanded and the bluestone, on the other hand, is contraindicated, and further, that contact of the caustic with the cornea should be avoided as far as possible. Magnesium sulphate eye baths (see § 134) are said to be specially usful in the

corneal complications of trachoma (Kilpatrick). For the iritis, which not rarely is associated with ulcers of the cornea, atropine in 1-per-cent solution is instilled. In other respects, ulcers of the cornea are to be treated according to the rules which are in general applicable to them (see §§ 541 et seq.). Only it must be noted that bandaging, which is generally indicated in the case of ulcers of the cornea, should be avoided as far as possible when trachoma is present, because by the closure of the eye the secretion is retained in the conjunctival sac, and thus both the conjunctival and the corneal troubles are aggravated.

Pannus, in recent cases, disappears of itself, simply from applications being made to the conjunctiva. If the pannus is unusually dense, it is allowable to make careful applications of the caustic to the pannus itself. Since pannus is often associated with slight iritis, atropine should be instilled from time to time, in order to keep the pupil dilated and prevent the formation of posterior synechiæ. In recent pannus we may try subconjunctival injections of 1:1000 mercury cyanide (§ 135) and in thick pannus, peritomy (§ 918).

In very old pannus, which already is partly made up of connective tissue and has lost all but a few of its vessels, we may use the *jequirity* treatment (De Wecker). We apply a 3- to 5-per-cent fresh infusion of jequirity to the conjunctiva of the everted lids very thoroughly two or three times a day. This produces an inflammation which reaches the desired height on the second or third day, when the lids are reddened and œdematous, the conjunctiva is strongly injected and covered with a croupous membrane, and slight chemosis is often present. Then the further application of the remedy is discontinued, as if we push it, we may cause necrotic disintegration of the conjunctiva and cornea. We now allow the inflammation to run its course, simply keeping the eye clean; when the inflammation has completely subsided, the cornea is found to have gained in transparency as compared with its former state, and sometimes to a very considerable extent. This very energetic treatment is adapted only to those old cases of trachoma in which symptoms of marked inflammation are wanting, the conjunctiva is in great part cicatricial, and the cornea is entirely covered by old pannus.

Jequirity (the seeds of the Abrus præcatorius) has for a long time been in use in Brazil, where trachoma is endemic, as a popular remedy for this disease. Its action depends on an unorganized ferment (abrin) which is excessively poisonous. Römer by immunizing animals with abrin, has obtained an *antitoxin* (§ 90) and by using increasing concentrations until the desired degree of inflammation is produced and by limiting an excessive action by means of the antitoxin, he has reduced the dangers of the original jequirity treatment to a minimum.

Of the *sequelæ* of trachoma, the distortion of the lids and its consequences, trichiasis, entropion, and ectropion, demand operative treatment (see §§ 925-929, 931). The symblepharon posterius, which is produced by the shrinking of the conjunctiva, is not amenable to treatment. Xerosis of the conjunctiva is also incurable, so that treatment must be limited to the amelioration of the patient's sufferings. To diminish the sense of dryness, frequent instillations of milk, glycerin, or mucilaginous substances (e. g., the mucilago seminum cydoniorum) may be made. For bad cases, Rudin, in order to preserve the eyeball from desiccation, has advised refreshing the edges of the lids and stitching them together, so as to unite them throughout except for a small space in their middle.

476. Hygienic or Prophylactic Treatment.—In addition to the foregoing measures for the treatment of trachoma, it is self-evident that care must be taken to keep the eye clean, for which purpose we may prescribe weak antiseptic solutions. The patient should have a nourishing diet; he ought not to be kept in his room, but, on the contrary, should be made to go out as much as possible in the open air and take exercise, and if necessary may be directed to engage in some light out-of-door work.

The disease being of such an infectious character, its dissemination should be checked by suitable *prophylaxis*. The physician must set a good example, and must cleanse his hands carefully after touching a trachomatous eye. He must call the attention of the patient affected with trachoma to the contagious nature of this disease. He must teach him how to protect from infection the other eye, which may be still healthy, and how to avoid spreading the disease among those in his immediate neighborhood, his family, his fellow-workmen, etc. For securing the latter object, the prime requisite is that the patient should have his own washing materials, linen, bed, etc., and should keep them exclusively for his own use.

Since trachoma is largely spread by an associated conjunctivitis, its dissemination can be prevented by cutting the latter short or preventing it. For this reason, when bodies of men are gathered together and there is possibility of trachomatous infection, the routine instillation of zinc sulphate has been used as a prophylactic (Stuckey and Hughes).

The prevention of epidemics of trachoma in public establishments, such as barracks and institutions and schools of every sort, constitutes an important duty of the officials in charge of such places. These officials should take care that the members of their community have separate washing materials, linen, etc., for their use. They should be kept apprised of the presence of any trachomatous patients by means of frequent medical inspection, and, as soon as such a patient is found, he should be immediately removed from the community; for, where no trachomatous patient is found no extension of the disease is possible.

In this country a great deal has been done in the way of prophylaxis by excluding trachomatous immigrants, by the systematic inspection and treatment of children in the public schools and in some places by making trachoma a reportable disease. It is important that the public should be educated with regard to the grave significance of the disease and the way in which it can be avoided.

477. Conditions Resembling Trachoma.—(1). Inclusion Conjunctivitis (Noguchi and Cohen) is a condition marked both by follicles in the retrotarsal folds and by a granular appearance of the tarsal conjunctiva and characterized by the presence of cell inclusions (Fig. 213). It runs a moderately severe course, is but little affected by treatment, and lasts usually from two to four months. When then it subsides it leaves the conjunctiva completely normal. It is thus distinguished from trachoma, which it externally resembles and with which, indeed, it is frequently associated (see below).

The cell inclusions were discovered in cases of trachoma by Halberstædter and Prowazek, who regarded the different forms as stages in the development of a protozoan organism (chlamydozoon), which they considered to be the cause of trachoma. These and other authors found similar inclusions in cases of ophthalmia neonatorum, gonor-

rhœal ophthalmia, and Koch-Weeks' conjunctivitis. Some regard them as varying forms of the gonococcus or of the Koch-Weeks bacillus, others as degenerative products. The most probable view seems to be that they are either pathogenic organisms (Noguchi) or at least, specific morbid structures associated with a peculiar tye of benign conjunctivitis (inclusion conjunctivitis) and that, when present in trachoma or other conditions, they denote simply a mixed infection, i. e., a combination of trachoma etc., with an inclusion conjunctivitis. This is inferred from the fact that inoculation of the higher monkeys with secretion derived from cases of either inclusion conjunctivitis or of trachoma

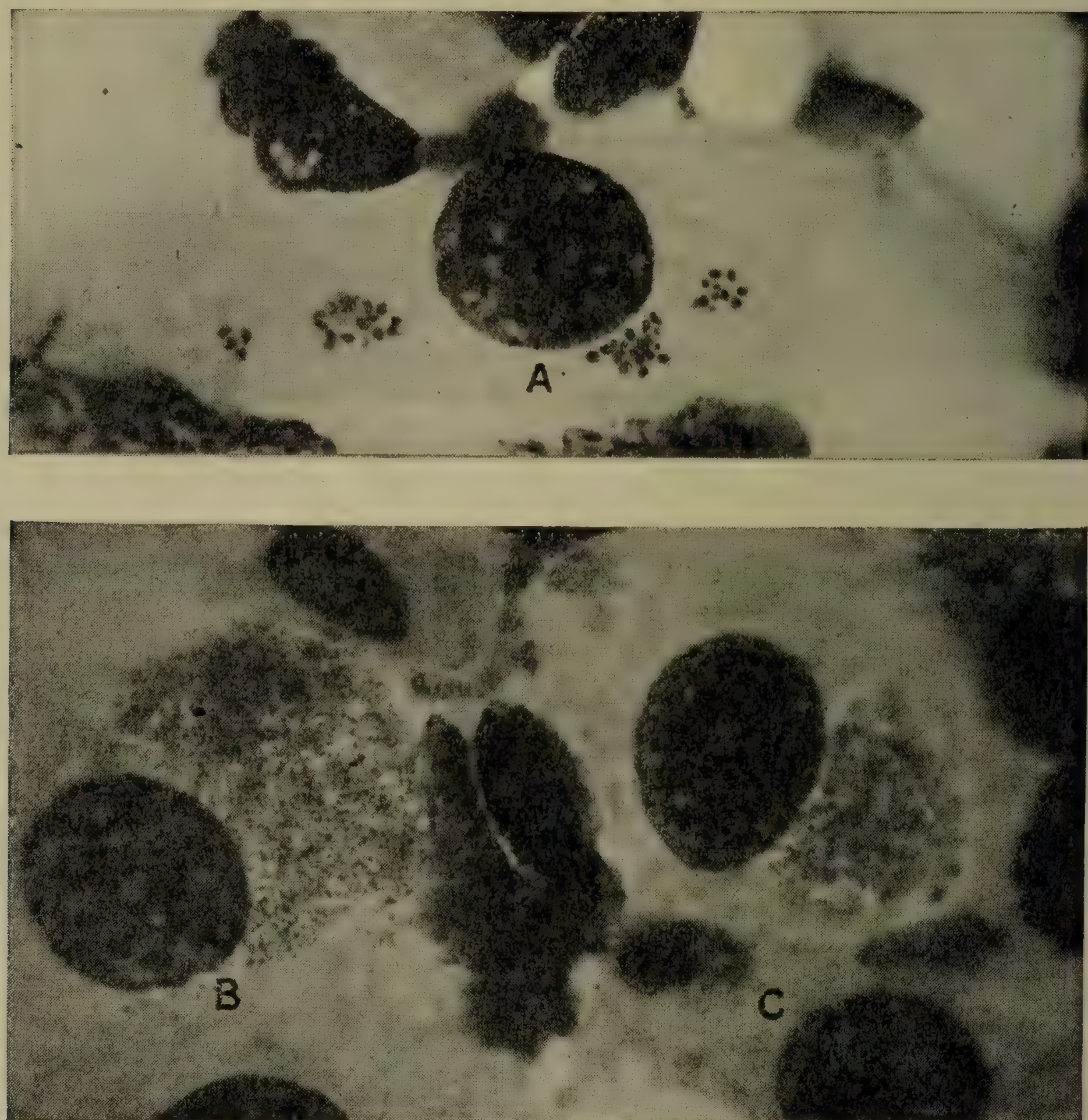

FIG. 213.—CELL INCLUSIONS (AFTER NOGUCHI AND COHEN).

In Giemsa-stained film preparations the cell inclusions (so-called trachoma bodies) appear either as coarse and often ovoid or polygonal bodies, staining blue, and of about the size of a gonococcus (A) or as minute granules, more or less regularly round and staining pink (B). The former, called initial bodies by Lindner, are few in number; the latter, known as elementary granules and regarded as later stages in the development of the organism, usually fill completely one side of the cytoplasm, presenting a peculir semilunar cap over the nucleus, to which latter the concave side of the inclusion conforms. There are in some cases intermediate forms (C), which may be interpreted as representing phases of development from the initial bodies to the elementary granules.

with inclusions, produces simply a benign transient conjunctivitis with inclusions, and never trachoma.

(2) DESQUAMATIVE EPITHELIOSIS.—This is a disease also marked by cell inclusions and clinically closely resembling trachoma. It is caused by the cytozoon atrophicans, and may occur in conjunction with Samoan conjunctivitis (see § 451), or independently, and produces an acute conjunctivitis with œdema of the lids and infiltration of the cornea. The conjunctiva contains follicles, in which are large epithelioid cells containing reddish cell inclusions. Epithelial atrophy and vacuolation of the cell-walls are marked (Leber).

(3) PARINAUD'S CONJUNCTIVITIS.—In this disease, which develops acutely with fever and other evidences of constitutional disturbance, reddish or yellowish granulations form in the highly inflamed conjunctiva, and these not only in the retrotarsal folds but also in the conjunctiva of the lids and even in the conjunctiva of the eyeball. The granulations sometimes grow so as to form quite large swellings; in many cases also there are very small superficial gray-coated ulcers in the conjunctiva. A characteristic sign of the disease is the swelling which affects the pre-auricular lymph gland and indeed the whole parotid region, sometimes even down to the neck. The swollen glands not infrequently suppurate. The disease ends in recovery within from a few weeks to a few months without producing any further bad results. Usually but one eye is affected. Parinaud ascribed the disease to infection derived from animals, but this cannot always be traced, nor is tuberculosis, which is present in some cases, uniformly present. Verhoeff found a leptothrix forming filamentous masses walled off by an area of cell necrosis, and Bayer and Herrenschwand a bacillus (bacillus pseudotuberculosis rodentium), which is found in rodents (rabbits, etc.).

(4) SQUIRREL-PLAGUE CONJUNCTIVITIS (*Conjunctivitis tularensis*).—This is a very severe conjunctivitis transmitted from squirrels and attended with fever, glandular swelling, and the formation of necrotic ulcers in the palpebral conjunctiva. It has been regarded as due to the bacillus tularensis, but Bayer and Herrenschwand consider it and the closely allied conjunctivitis necroticans infectiosa of Pascheff as only varieties of Parinaud's conjunctivitis and caused by the same organism.

(5) SPOROTRICHOSIS CONJUNCTIVÆ.—This rare disease is caused by various species of sporotrichon and produces small, light-yellow, soft nodules in the conjunctiva with points of ulceration and with purulent discharge. There is swelling of the neighboring lymph glands. Nodules also occur in the lid-margin. The canaliculi may contain concretions enclosing the sporotrichon. The disease resembles Parinaud's conjunctivitis, from which it can be differentiated by the discovery of the sporotrichon (Wilder, Bedell). A cure usually results from the internal use of potassium iodide.

(6) BLASTOMYCOSIS CONJUNCTIVÆ.—Polypoid nodules in the tarsal or retrotarsal conjunctiva attended with slight catarrhal conjunctivitis are occasionally produced by an organism apparently a blastomyces (Carpenter, Posey); remedy, excision. (Cf. § 421.)

VI. CONJUNCTIVITIS DIPHTHERICA

478. Symptoms.—Conjunctivitis diphtherica,[10] like gonorrhœal conjunctivitis and trachoma, is a purulent inflammation of the conjunctiva which spreads by contagion, and the secretion of which is infectious. Its cause is the diphtheria bacillus of Löffler. The inflammation produced by this is generally violent, and in the severe cases is among the most intense of any that are observed in the conjunctiva. The lids are much swollen and reddened, hot, and painful to the touch. In particularly severe cases the lids are actually of board-like hardness, so that it is impossible to evert them, and scarcely possible even to open the palpebral fissure. The lymphatic glands in front of the ear or in the neck are swollen. The appearance of the conjunctiva is characteristic of the property that the diphtheria bacillus possesses of producing a profuse exudation which has a great tendency to coagulate. This coagulation either affects the exudate that is poured out upon the surface of the conjunctiva, and which consequently clots to form membranes, or it takes place within the tissue of the conjunctiva. Consequently we may distinguish two forms of diphtheria of the conjunctiva, the clinical aspect of which is depicted below. This, however, does not suffice for a sure diagnosis, since the same picture may be

[10]From διφθέρα, a membrane.

produced by other bacteria beside the Löffler bacillus (see § 481). Hence, a bacteriological examination is also necessary, and the more so because upon it will depend in part the treatment (e. g., as to employment of antitoxin and regulations to prevent the transmission of the disease). The two forms are:—

(*a*) The *superficial* or *croupous* form. This is characterized by the presence of a grayish-white membrane, which adheres pretty closely to the surface of the conjunctiva, but still can be usually removed from it with a forceps. When this is done we find the conjunctiva beneath to be greatly reddened and swollen and in some places bleeding, but we notice no great losses of substance in it. The membrane which we have detached consists of a minute fibrous meshwork of clotted fibrin, in which pus corpuscles and a few epithelial cells from the conjunctiva are imbedded. The croupous membrane covers the tarsal conjunctiva; more rarely, the retrotarsal fold and even the conjunctiva of the eyeball. In most cases after from one to two weeks the membrane gradually disappears. The eye now merely presents the condition of an intense catarrhal inflammation which gets well without leaving any permanent changes in the conjunctiva. In severe cases the disease is complicated with corneal ulcers, which, however, but rarely lead to extensive destruction of the cornea.

(*b*) The *deep* form (*diphtheria* of the conjunctiva in the narrower sense). This runs a much more serious course than does the superficial form, as in order for it to occur, the exudate must coagulate while still within the tissues of the conjunctiva, so that the vessels are compressed by it, and the mucous membrane consequently is rendered rigid and bloodless and falls a prey to necrosis. Hence, on everting the lids we find, beside the marked swelling and redness of the conjunctiva, spots in which the conjunctiva is somewhat depressed, smooth, and of a grayish-yellow color, and often contains a few dirty-red speckled markings (ecchymoses). In the severest cases, either a very large portion of the conjunctiva, or even its entire surface, acquires this character and is uniformly gray and hard, just as it is after being intensely cauterized, e. g., by the action of lime. The lymph glands in front of the ear and often also those in the neck are swollen and painful.

The condition just described, which develops rapidly after a short period of incubation, is called the first stage of the disease, or stage of *infiltration*. It keeps up for from five to ten days, according to the extent of the diphtheritic process upon the conjunctiva. Then the spots of diphtheritic infiltration begin gradually to disappear. Where the infiltration is not so very dense, resorption of the exudate occurs, but in those spots from which the circulation has been altogether cut off by the infiltration and the tissue has consequently mortified the necrotic portions slough away. Thus are produced in the conjunctiva losses of substance, which soon become covered with granulations such as cover a raw surface. Meanwhile the secretion has become more abundant and more purulent, for which reason this second stage is characterized as the stage of *blennorrhœa*. The third stage is that of *cicatrization*, in which the granulating surfaces, that are produced by the sloughing off of the gangrenous portions of the conjunctiva, gradually grow

smaller and are covered over with a new epithelial lining. Since the latter change is effected by a drawing in of the neighboring conjunctiva, the conjunctival sac as a whole is contracted; frequently, too, in single spots adhesions are produced between the conjunctiva of the lid and that of the eyeball (symblepharon). The more extended the diphtherial process the more striking is the subsequent cicatricial contraction of the conjunctiva. The result may be trichiasis, cicatricial entropion, or even xerophthalmus.

The deep form of diphtheria is much more severe than the croupous, not only in regard to its effect upon the conjunctiva, but also in other respects. Hence the cornea is much more frequently and much more seriously affected. The greater the extension of the diphtherial process upon the conjunctiva the more certain is corneal suppuration to occur. If the entire area of the conjunctiva is infiltrated and rigid, the cornea is probably always irretrievably lost.

The general condition of the little patients is very much disturbed. They have high fever and are greatly prostrated. Weakly children not infrequently succumb to the severity of the general disease. The prognosis, therefore, in the severe cases is very serious, not only as regards the eye, but also with respect to life itself.

We owe the first exact description of conjunctival diphtheria to von Graefe, who, in Berlin, had an opportunity of seeing many cases of this disease. His description relates to the deep variety, of which he distinguishes two groups of cases. In the first group—that of diphtheria en plaques—constituted by the lighter cases, the diphtherial spots are found under the form of large or small islands, which occur especially on the conjunctiva of the lids and between which lie areas of tissue that is not so much diseased. In the severer cases, on the other hand, the diphtherial foci rapidly coalesce, so that the entire conjunctiva becomes rigid and bloodless (confluent diphtheria).

479. Etiology.—That the two forms just described, which differ so much in their appearance and course, are, nevertheless, the same disease, namely diphtheria, is proved from the fact that Löffler's bacilli are found in the conjunctival secretion in both. Often, too, the patients present other important and undoubted diphtherial affections. Small-sized diphtherial patches are frequently found at the edges or angles of the lids, the nostrils, or the angles of the mouth; sometimes even there is a fully developed nasal or pharyngeal diphtheria.

Diphtheria of the conjunctiva is mainly observed in those countries where diphtheria of all sorts is a frequent occurrence, and occurs especially at times when an epidemic of pharyngeal diphtheria is prevailing. It is rare in America. It can often be proved that children affected with diphtheria of the conjunctiva had previously been thrown with others, who soon afterward developed pharyngeal diphtheria; and such children may themselves in their turn spread the disease to others still. The predisposition to diphtheria diminishes with the age. Consequently, diphtheria of the conjunctiva usually attacks children, and most frequently those between the second and eighth year of life. Adults are only exceptionally attacked and then generally by one of the lighter forms.

After Löffler had discovered in the membranes of pharyngeal diphtheria the bacillus that bears his name it was soon after shown to be present in diphtheria of the conjunctiva also (Babes, Kolisko and Paltauf, and others). On the other hand, no one supposed that the cases in which a membrane forms upon the conjunctiva must also be regarded as diphtheria until the presence of the Löffler bacillus was demonstrated in them too (first by Gallemaerts). The same thing occurred in this case as in that of pharyngeal diphtheria and laryngeal croup, whose etiological identity was long unrecognized. It is supposed that membranous inflammation of a mucous membrane implies a feebler action of the diphtheria bacilli than does diphtheritic inflammation proper, the comparatively sight effect of the bacilli in the former case being due either to their having lost their virulence or to the patient's being more refractory to the influence. But apart from this, the severity of the inflammation is influenced by the fact that beside the Löffler bacillus other germs occur on the inflamed conjunctiva, such as the staphylococcus and streptococcus, which aggravate the symptoms. In fact, the streptococcus by itself is competent to produce a disease of the clinical aspect of conjunctival diphtheria (§ 481).

480. Treatment.—In the severe cases of diphtheria of the conjunctiva the injection of antitoxin is indicated. The injection is best made beneath the skin of the lids. In addition the serum is often instilled into the conjunctival sac. In the lighter (croupous) cases we may content ourselves with employing local treatment alone. In the first stage of the disease this is chiefly limited to careful cleansing of the eye, for which purpose the best thing for us to employ is a weak antiseptic liquid (beside solutions of corrosive sublimate and potassium permanganate, a solution of quinine is particularly recommended). Cold compresses, which would seem to be indicated by the great swelling and redness of the lids, must be applied only when the conjunctival circulation is not too seriously embarrassed by the diphtherial infiltration. Otherwise, it is better to employ warm compresses which by dilating the blood-vessels increase the circulation. As regards the conjunctiva itself, Fieuzal has recommended painting it with lemon juice; and painting it with strong sublimate solution (1:1,000), either directly or after the removal of the membrane, if present, is highly spoken of. Except for this purpose there is no object in removing the membranes in the croupous variety, since these at once re-form. When, after separation of the membranes or the slough, the conjunctiva has become strongly congested, soft, and succulent, and the secretion begins to be abundant, we may commence the application of a nitrate-of-silver solution, by means of which we bring the swollen conjunctiva more rapidly back to its normal state. In so doing we must first proceed with great caution, use a pretty weak solution (one per cent) and discontinue the application at once if membranes or deep infiltrations once more develop.

We keep on making the applications to the conjunctiva as long as the latter is red and swollen and discharges a copious secretion. If in the deep form, after the subsidence of the disease, partial necrosis and sloughing of the conjunctiva have set in, we try during the subsequent period of cicatrization to oppose, as far as possible, the formation of adhesions between the lids and the eyeball (doing this by frequently drawing the lid away from the eyeball or by laying a pledget soaked in oil between the two), for adhesions once formed can be removed only by an operation. Complications affect-

ing the cornea are to be treated according to the rules that will be given further on for purulent inflammation of the cornea in general.

All operative procedures, whether upon the cornea or upon the lids, should be avoided in the first stage, as the wounds thus produced generally become diphtherial too.

In consideration of the very infectious character of diphtheria, our special aim must be *prophylaxis.* Accordingly, we remove from the vicinity of the patient all persons who are not indispensably necessary for purposes of nursing; but, most of all, we insist upon the removal of children, as these are particularly susceptible to infection. If diphtheria has attacked only one of the patient's eyes, the other must be protected against infection by a carefully applied occluding bandage, just as in gonorrhœal conjunctivitis.

The persons who have charge of the patient must be particularly enjoined to cleanse the hands carefully after touching the diseased eye, to destroy at once the materials employed in cleansing, etc.

VII. Non-Diphtherial Membranous Conjunctivitis

481.—The terms croupous and diphtheritic are primarily anatomical terms denoting definite forms of inflammation. Croupous inflammation is characterized by the deposition of an exudate upon the surface of a tissue, where, by coagulation, it hardens into a membrane. The essence of the diphtheritic inflammation, on the contrary, consists in the exudation of a great mass of material within the tissue itself, with consecutive necrosis of the latter. Diphtheritic inflammation may be regarded as a croupous inflammation carried to a higher pitch, in so far as the same injurious cause may, when acting to a slight extent, produce a croupous, when acting to a greater extent, a diphtheritic inflammation of the mucous membrane. In the conjunctiva either form of inflammation, having the characters described in § 478, may be due to other causes than the diphtheria bacillus.

One of the most serious kinds is *membranous streptococcus conjunctivitis.* It is found particularly in children as a result of the exanthemata (measles, small-pox, scarlet fever) and in the severe form leads to death or blindness. Croupous or diphtheritic membranes are also found in isolated portions of the conjunctiva in cases of *gonorrheal* conjunctivitis, and a severe membranous conjunctivitis is caused by the *thrush fungus* (Pichler). Less violent membranous inflammations are caused by the pneumococcus, meningococcus, and Koch-Week's bacillus.

These acute, spontaneously developing forms of membranous conjunctivitis were formerly regarded as a substantive disease called conjunctivitis cruposa.

To the cases of membrane formation upon the conjunctiva that pursue a more *chronic* course belong the very rare instances of *herpes iris* of the conjunctiva. These latter can readily be diagnosticated, provided the characteristic exanthem of herpes iris (a central reddened or pigmented area of skin surrounded by a wall of vesicles) is also to be found upon the skin. This, however, is not always present. Sometimes, too, a formation of membrane like that upon the conjunctiva occurs upon the mucous membrane of the mouth. In some cases the disease recurs frequently.

Furthermore, there have been described cases of membrane formation upon the conjunctiva which were peculiarly chronic cases—lasting for months, or even years—the nature of which is still doubtful (Arlt, Hulme, Morton, and others).

External irritants of a *chemical nature* may set up a croupous or a diphtheritic conjunctivitis according to the intensity of their action. Such substances include bodies, both inorganic and organic, like ammonia, nitrate-of-silver solution, and jequirity infusion. We see their effect if in our practice we apply nitrate of silver too often or too intensely to an inflamed eye. The first effect is to produce a membranous, and, if we keep on, a diphtheritic inflammation.

Losses of substance on the conjunctiva (and the same thing is seen in other mucous membranes) very soon become covered with a membrane of coagulated fibrin, under which the healing of the wounds proceeds. This process is observed after operations (e.g., tenotomy), injuries, and also spontaneously developing raw spots—as, for example those occurring after rupture of pemphigus vesicles (see § 490).

Use of Terms Diphtherial and Diphtheritic.—Since other agencies than diphtheria may cause membranous inflammation and since diphtheria itself at times causes a non-membranous conjunctivitis, it is well to make the term *diphtheritic* denote the anatomical type of inflammation characterized by massive exudation, whatever the cause of this exudation may be; and to make the term *diphtherial* denote a condition of whatever anatomical character produced by the Löffler bacillus (Roser). The word *croupous* should be used as a purely anatomical term.

VIII. Phlyctenular Conjunctivitis [11]

482. Characteristics.—In its simplest, typical form, phlyctenular conjunctivitis presents the following picture: A little red eminence, of about the size of a millet seed, develops at some point upon the limbus of the conjunctiva. This is the *efflorescence* or *phlyctenule* (Fig. 214). In the beginning it is conical, its apex being covered by the epithelium of the conjunctiva. In a short time the epithelium at the summit of the phlyctenule separates, and the tissue that lay beneath it breaks down, so that the apex of the cone, so to speak, melts away and the cone itself bears on its top a minute gray ulcer, which thus lies above the level of the neighboring, healthy conjunctiva. By a continuation of the breaking-down process the cone at length disappears entirely, the ulcer sinks to the level of the conjunctiva, and speedily becomes clean and then covered with epithelium. Thus the ulcer heals, without a visible mark being left upon the conjunctiva.

As the phlyctenule springs up, the adjacent part of the conjunctiva becomes hyperæmic, the injected vessels being directed from all sides toward the little nodule. Hence, the reddened portion of the conjunctiva shows the form of a triangular sector, the apex of which lies in the limbus and corresponds to the nodule. The remainder of the conjunctiva is perfectly free from congestion.

The simplest type of phlyctenular conjunctivitis, therefore, consists in the formation of a sharply circumscribed, nodular exudate, to which there corresponds an injected district of the conjunctiva. Phlyctenular conjunctivitis is hence a *focal* affection of the conjunctiva of the eyeball, and is thus distinguished from all the varieties of conjunctival inflammation

[11] The term phlyctenular conjunctivitis is so firmly fixed in usage that it is used here in place of Fuchs' title conjunctivitis eczematosa. The name phlyctenular conjunctivitis, like the synonymous terms conjunctivitis pustulosa and herpes conjunctivæ (Stellwag), is based on the erroneous idea that the efflorescence on the conjunctiva or cornea is a hollow vesicle filled with fluid (φλύκταινα, bladder; pustula; herpes vesicle). But the efflorescence is in reality never a vesicle, but a solid, though soft projection, which is formed chiefly by an accumulation of leucocytes (Figs. 214 and 230). The softening and liquefaction of this cellular mass do not begin in the interior of the projection, but at its apex, so that no cavity is formed (vesicle or pustule), but a loss of substance (ulcer) occurs, lying upon the free surface at the apex. The name herpes corneæ, moreover, can give rise to a confusion with true herpes corneæ (herpes febrilis and herpes zoster, see §§ 565 and 566. Other synonyms are conjunctivitis lymphatica (or scrofulosa or exanthematica) and phlyctænulosis.

hitherto described, which are diffuse inflammations, in that they extend in a uniform fashion over large sections of the conjunctiva.

483. Varieties.—The clinical pictures which phlyctenular conjunctivitis actually exhibits present modifications of the simplest type above described which differ most widely from each other. These modifications concern—

(*a*) The *number* of the phlyctenules. It is rare that we find but one of these; generally there are several, and not infrequently a good many, present at the same time. The fewer they are, the larger they generally grow; in rare cases they attain almost the size of a lentil. Where there are many nodules present they are small; often we find the entire limbus, or even the cornea itself, covered with very minute eminences, so that the surface of the

FIG. 214.—EFFLORESCENCE (PHLYCTENULE) IN THE LIMBUS. Magnified 62×1.

The sclera, *S*, is distinguished by its more delicate fibrillation and its blood-vessels from the more homogeneous, non-vascular cornea, *H*. The nodule is situated at a point corresponding to the boundary between the two membranes but more over the sclera than over the cornea. It consists of densely packed round cells, between which the blood-vessels are recognizable under the form of lighter-colored striæ. In the vicinity of the nodule the vessels of the conjunctiva (*c*) and episclera (*e*) are bordered by extravasated leucocytes. The epithelium (*E*) of the conjunctiva is bulged forward by the nodule and at the apex of the latter is thinned, and, owing to the penetration of the round cells into the epithelial layer itself, has lost the sharp border ordinarily existing between it and the connective tissue.

eyeball looks as if fine sand had been strewn over it. Such very small nodules commonly disappear in a few days by resorption, without any preliminary disintegration. When multiple phlyctenules are present, the injected portions of the bulbar conjunctiva belonging to the separate nodules become confluent, and the conjunctiva then appears reddened all over, so that the focal character of the diseases is obscured, and declares itself only by the presence of separate nodular exudates. So, too, when the inflammation is great, the palpebral conjunctiva also participates in the injection, so that, in that case, phlyctenular conjunctivitis is no longer an affection limited to the bulbar conjunctiva.

(*b*) The *site* of the phlyctenules may be not only in the limbus itself but also exterior to the latter, in the anterior segment of the bulbar conjunctiva, and likewise interior to the limbus, in the cornea itself. In the latter the small gray nodules are situated in the most superficial layers of the cornea (Fig. 230). By the breaking down of the nodule there is pro-

duced in the cornea a very shallow loss of substance, which leaves scarcely any opacity. Often, however, the affection assumes a more serious form from the fact that the exudations have a tendency to spread farther in the cornea, extending either into the depth of the latter or along its surface. If the infiltration extends through Bowman's membrane into the parenchyma proper of the cornea, an ulcer is produced by its disintegration which penetrates more deeply and can even perforate the cornea. In that case, after the ulcer heals, a permanent opacity remains.

(*c*) The corneal ulcers which result from the phlyctenules may assume a serpiginous character—that is, they may spread by a sort of creeping process along the surface of the cornea. In this way is produced the *vascular fasciculus* (Fischer), which is also called keratitis fascicularis. This affection begins by the development of a small ulcer out of a phlyctenule at the rim of the cornea. After some days this ulcer becomes clean in its peripheral half—that is, in the part turned toward the corneal rim. At the same time, in accordance with the ordinary behavior of regressive corneal ulcers, blood-vessels develop, which run from the limbus to that edge of the ulcer which is healing. These serve to keep up the process of cicatrization. But in the meantime, that margin of the ulcer which is toward the centre has remained infiltrated and gray. Inasmuch as at this place the infiltration and the subsequent purulent disintegration keep on extending, the ulcer constantly advances toward the centre of the cornea, while on its peripheral side it heals equally fast, and trails the blood-vessels after it. The vascular fasciculus accordingly appears under the guise of a narrow red band formed of blood-vessels (hence the name), and extending from the corneal margin some distance into the cornea. At its apex it bears a small gray crescent, the infiltrated, advancing margin of the ulcer. The arrest and recession of the process first occur when the ulcer is completely healed. Until this has taken place the vascular fasciculus can advance far into the cornea, to its centre or even beyond it. The small ulcer, however, always remains superficial, and a perforation of the cornea due to it is never observed. When the vascular fasciculus has at length come to a standstill, the vessels gradually disappear from it, and there only remains a superficial opacity of the cornea which corresponds in shape to the long-drawn-out form of the vascular fasciculus. This opacity never clears up again completely, and hence, when found at any time during the whole subsequent life of the patient, enables us to diagnosticate the previous existence of a vascular fasciculus.

(*d*) The severest cases of phlyctenular conjunctivitis are those in which the exudation from the start makes its appearance in the deep layers of the cornea as a widely diffused *deep-lying* infiltration. We then find the cornea occupied to a considerable extent by an opacity of a uniform gray or yellowish color, with hazy outline and seated in the deeper layers of the cornea; the surface of the cornea over it is stippled. In the bad cases the infiltrate, originally gray, becomes more and more yellow, and finally breaks down into pus, so that an extensive loss of substance is produced in the cornea. In the benign cases, on the contrary, the infiltrate gradually

disappears again by resorption, and the cornea regains its transparency either wholly or in part. It is astonishing to what extent even extensive infiltrates can undergo resolution.

(*e*) Instead of appearing as separate circumscribed foci, the exudate may occur under the form of a continuous new formation of tissue upon the surface of the cornea—that is, under the form of *pannus*. This is called phlyctenular (or eczematous) pannus, to distinguish it from trachomatous pannus. It does not, like the latter, show a predilection for the upper part of the cornea, but develops from any spot whatever upon the corneal margin. It is ordinarily thin and not very vascular, and is quite apt to undergo complete resolution.

The authors separate the phlyctenular affections of the conjunctiva from those of the cornea; they speak of conjunctivitis and of keratitis phlyctænulosa according as the efflorescence is located upon the conjunctiva or the cornea. In this way, for mere love of system, a picture of disease that, clinically speaking, is a perfect unit, is torn in two. In fact, we have here really but one single disease, which is localized sometimes in one place, sometimes in another. Frequently enough we find in the same eye, at the same time, one phlyctenule in the conjunctiva of the eyeball, a second in the limbus, half in the conjunctiva and half in the cornea, and a third upon the cornea itself (very exceptionally phlyctenules occur even on the conjunctiva of the lids). Hence, in the foregoing description of the disease, the expression phlyctenular conjunctivitis is employed for the disease in general, no matter upon what part of the surface of the eyeball it is localized. This can be done without doing violence to anatomy, inasmuch as the outermost layer of the cornea must be looked upon as the continuation of the conjunctiva over the cornea. Accordingly, we can regard the involvement of the cornea in phlyctenular conjunctivitis as an involvement of the "conjunctival layer" of the cornea. For the same reason we regard pannus trachomatosus as one of the symptoms of conjunctivitis trachomatosa, and not as an independent affection of the cornea.

Phlyctenules of the cornea begin with an accumulation of leucocytes beneath Bowman's membrane (Fig. 230). This is so adherent that it cannot be lifted up by the accumulated cells like the delicate conjunctiva scleræ, and for this reason phlyctenules on the cornea, in contrast with those on the limbus, are not at all or but little prominent. When the infiltration of the uppermost lamellæ of the cornea has reached a certain point, the epithelium over it and then also ultimately Bowman's membrane break down. When this occurs the true loss of substance begins, and the phlyctenule is converted into an ulcer. Pannus also usually begins beneath Bowman's membrane.

484. Diagnosis.—The differential diagnosis between phlyctenular conjunctivitis and the other affections of the conjunctiva and the cornea is, as a rule, easily made. The characteristic mark of the former lies in the focal character of the affection as well as in its localization upon and immediately about the cornea.

Only one other variety of conjunctivitis, the conjunctivitis ex acne, shares this peculiarity with phlyctenular conjunctivitis; but that disease is readily distinguished from the latter by the accompanying acne rosacea upon the face (see § 490). In spring catarrh also (§ 491) little nodules occur upon the limbus, but never break down into ulcers. Of the diffuse inflammations of the conjunctiva, catarrh might be confounded with conjunctivitis. For in intense and obstinate cases of phlyctenular conjunctivitis, the affection spreads to the palpebral conjunctiva, which may become very much reddened, swollen, or even velvety; in that case, too, a mucous or muco-purulent secretion forms upon the conjunctiva. It is often difficult to distinguish between such cases and catarrh, especially if just at the moment of examination there are no characteristic phlyctenules present upon the conjunctiva of the eyeball. Fortunately, a mistake in

diagnosis does no harm, since, with such a condition of the conjunctiva existing, gentle cauterization with the nitrate-of-silver solution is always indicated, no matter what the origin of the disease. The pustular form of acute conjunctival catarrh forms a sort of intermediate stage between catarrhal and phlyctenular conjunctivitis (see § 451).

Phlyctenular pannus is to be chiefly distinguished from pannus trachomatosus by the fact that an exact examination of the conjunctiva of the lids and of the retrotarsal fold either discloses the changes of trachoma or establishes the fact of their absence.

Corneal ulcers which have been preceded by phlyctenules sometimes are located so very near the margin of the cornea that they extend into the limbus conjunctivæ; and, since corneal ulcers as peripherally situated as this occur only in connection with phlyctenular conjunctivitis, the diagnosis of an antecedent phlyctenular conjunctivitis can be made even years after it has elapsed from scars having such a situation (Fig. 239).

The *vascular fasciculus* can readily be confounded with an ordinary corneal ulcer, to which, in the course of healing, vessels have made their way from the limbus so as to form a red bridge between the limbus and the ulcer. In such a case there is no fear of the ulcer's extending into the pupillary area of the cornea, and, after the ulcer heals, only a small, rounded macula is left, and not a long, opaque stria, as in the case of the vascular fasciculus. The distinction between these two affections can be made as follows: In the vascular fasciculus, the advancing margin of the ulcer, infiltrated with gray, is readily visible; the blood-vessels as they run up to it lie in the furrow which the ulcer has channeled in the course of its progress—lie, therefore, at or below the level of the corneal surface. In the case of a simple ulcer with development of vessels, this furrow is wanting, and the vessels have a quite superficial situation.

485. Symptoms.—Phlyctenular conjunctivitis is generally accompanied by profuse lacrimation. Mucous or muco-purulent secretion, on the contrary, such as occurs in catarrh, is not present as a rule; hence the lids do not ordinarily stick together in the mornings. The only exception to this is formed by those old cases in which the inflammatory process has passed over to the palpebral conjunctiva, and has, we may say, thrown it into a state of concomitant catarrhal inflammation.

The *subjective symptoms* consist of photophobia combined with spasm of the lids (blepharospasm). Slight in some cases, in others they reach an extraordinary pitch; children creep into a dark corner of the room, bury their faces in their hands, and struggle so violently against any attempt at opening their eyes that the examination on the part of the physician is made with great difficulty. The intensity of these symptoms bears no definite relation to the severity of the disease; in fact, it is precisely in that form of corneal affection which spreads more extensively and penetrates more deeply that the evidences of irritation are often rather slight. The annoyance suffered, contrary to what takes place in conjunctival catarrh, is, generally speaking, greater in the morning than in the afternoon and evening.

The photophobia in many cases persists obstinately for months. The parents then bring the children to the physician, with the statement that they have been "blind" for such or such a number of weeks. The children offer the greatest resistance to the forcible opening of the eyes, especially when there are excoriations at the external commissure, which give pain and bleed readily when the lids are separated. Hence, in such cases the palpebral fissure is to be opened cautiously and not too wide, so as to avoid making the struggles of the children still greater. The lids, and especially the upper lid, are rendered œdematous by the constant blepharospasm, because the veins of the lids, which pass between the fibres of the orbicularis, are compressed by persistent contraction of this muscle. Furthermore, a state of inversion of the lids (entropion spasticum) may be induced by this forcible squeezing together of the eyelids. Finally, cases have been

described in which children who have suffered for a long time from blepharospasm were perfectly blind after the disappearance of this symptom (von Graefe, Schirmer, Leber, and others). Such blindness is transient. As in the most cases no objective changes were demonstrable as the cause of the blindness, the latter is probably to be regarded as a central, purely functional affair, like hysterical amaurosis.

486. Course and Prognosis.—A single typical phlyctenule upon the limbus passes through all its phases up to complete subsidence in eight to fourteen days. If several phlyctenules are present, the process of cure requires a proportionately longer time. Nevertheless, the disease would not last so very long if it limited itself to a single attack. This, however, is but rarely the case. Usually, after a period of quiescence, or even before the first attack of inflammation has quite run its course, the eye becomes red again, and new nodules shoot up in or near the limbus. Thus the disease may, with longer or shorter intermissions, last on for months or years. Its beginning occurs in childhood; the separate attacks, however, are often protracted until the time of puberty, or sometimes even later; the affection being situated now in one eye, now in the other, then in both at the same time. Finally, the attacks become less and less frequent, and at length cease altogether.

The constant wetting of the lids by the tears frequently leads to blepharitis, to eczema of the skin covering the lids, and, as a consequence, to ectropion of the lower lid. Excoriations are frequently present at the external angles of the lids, and blepharophimosis often develops later on.

The *prognosis* of any single attack of phlyctenular conjunctivitis is favorable, in so far as the eye is but rarely rendered entirely blind by it. Superficial phlyctenules disappear without leaving a trace behind; ulcers which penetrate into the parenchyma proper of the cornea leave permanent opacities, which, however, are in most cases thin and superficial (maculæ of the cornea). On the other hand, the prognosis of the disease as a whole is rendered unfavorable by the frequent recurrences. In persons who have gone through many recurrences of phlyctenular conjunctivitis the corneæ often bear quite a number of maculæ as signs of past attacks. Thus the sight is impaired, squint or myopia often develops, and the persons thus affected often become incapable of doing fine work. In addition to this, children, in consequence of the frequently repeated inflammation of the eyes, fall behind in their physical and mental development. Finally a not infrequent outcome is secondary blindness, often occurring many years after the inflammation has passed, and representing the after-results of the corneal scars with inclusion of the iris which remain after the corneal ulcers.

487. Etiology.—Phlyctenular conjunctivitis is one of the most frequent of eye diseases, and has its origin in the scrofulous diathesis. Like the latter, it is a disease of childhood and youth. In very young children—those under the age of one year—it occurs but seldom, and it generally ceases at the time of puberty. Adults are attacked by it only in case they have carried the disease along with them from their childhood. The enormous majority of children affected with phlyctenular conjunctivitis belong to the poorer classes. Such children receive insufficient and unsuitable

nourishment, live in damp, poorly ventilated dwellings, and are kept constantly dirty. It has been found frequently as a post-war condition in the under-nourished French peasant children (Rutherford). Other children affected are those who, though once healthy, have become run down as a result of other diseases (scarlet fever, measles, typhoid fever, whooping cough, etc.). Such children as these look either pale and thin or bloated and spongy-looking. The glands at the lower jaw, in the neck, and in front of the ear, are swollen. Partly as a result of the suppuration of these glands, and partly as a result of the breaking down of the scrofulous infiltrations in the skin, ulcers and fistulous passages are produced, which require months and years for their cure, and leave characteristic and disfiguring scars behind. Patches of moist eczema occur at various spots upon the body, most frequently upon the face; and the constant coryza from which many of these children suffer is to be attributed to an eczematous affection of the nasal mucous membrane. Adenoid vegetations, too, are not rare. In the lids we find blepharitis. The nose and the upper lip are rendered thick by frequently recurring attacks of inflammation. More profound affections that occur are caries of bones (caries of the petrous bones appearing under the form of an otorrhœa being frequent), tuberculosis, and in girls, delayed and irregular menstruation.

Some of the above-mentioned symptoms of scrofula, and often several of them at once, accompany most cases of phlyctenular conjunctivitis. Sometimes, though rarely in comparison with other cases, the disease is observed in an individual who otherwise is quite healthy, just in the same way that other indications of scrofula occur at times as altogether isolated phenomena.

In seeking the true nature of the efflorescences of phlyctenular conjunctivitis we must keep two facts in mind.

(1) Phlyctenules when quite recent are sterile, containing neither the ordinary pyogenic germs nor tubercle bacilli. Inoculation from them into a test animal does not lead to tuberculosis. Hence it can not be assumed that, like other inflammations of the conjunctiva, they are referable to ectogenous infection, and as little are they to be regarded in the light of true tuberculous nodules.

(2) In a great number of patients with phlyctenular conjunctivitis there are changes which are certainly tuberculous, most frequently appearing under the form of scrofulous lesions of the glands and bones, and not infrequently also of pulmonary tuberculosis. But even in those patients who otherwise show no clinically demonstrable evidence of scrofula or tuberculosis, the examination with tuberculin (by subcutaneous injection or cutaneous inoculation) proves with rare exceptions the presence of latent tuberculosis. If then the phlyctenules are not actual tuberculosis nodules and yet, on the other hand, they occur with such preponderating frequency in tuberculous subjects, we should not be far out in explaining them as being due to the action of toxic substances, in the same way as, for example, nodules develop in tuberculous individuals after the rubbing of a tuberculin ointment into the skin (Moro). In harmony with such a connection is the observation that after the inoculation of tuberculin in children whose eyes were hitherto healthy phlyctenules sometimes appear on the conjunctiva after an incubation period of about 14 days. It has been suggested that the development of phlyctenules in tuberculous subjects is an example of anaphylaxis (Verhoeff). In any event it seems clear that the relationship of this disease to tuberculosis is at most an indirect one. As the results of treatment indicate there are probably other contributory causes, (disturbance of metabolism due to gastro-intestinal toxæmia, pharyngeal and tonsillar infection, etc.).

488. Therapy.—In the lighter cases, the *local treatment* consists in the application of irritants, of which calomel and the yellow-precipitate (or Pagenstecher's) ointment are most in use. The calomel in a finely powdered state is sprinkled in a thin layer upon the conjunctiva of the lower lid (or directly on the cornea) by means of a camel's-hair brush; the precipitate ointment (0.05 to 0.15 of yellow oxide of mercury to 5 of fat), is introduced into the conjunctival sac by means of a glass rod, and is then rubbed about with the lids, so as to be distributed over the whole conjunctiva.

Both remedies are irritant in their action; the precipitate ointment more so than the calomel. Hence, in the beginning of the disease, where the eyes are in a marked state of irritation, it is best to employ calomel; and subsequently, when the inflammatory symptoms are diminishing, to replace this by the yellow ointment. The remedies mentioned are to be applied only once a day, but it is advisable to keep on with them for quite a long time, in order to prevent relapses. Both find a contraindication in the presence of recent infiltrates or progressive ulcers in the cornea. In such cases, before having recourse to irritant remedies, we must wait, meanwhile employing atropine, until the process of infiltration has subsided or the ulcer has become clean. Pannus and vascular fasciculi do not contraindicate calomel or the yellow ointment. Neither should be put into the eye late at night (§ 125).

If we dust *calomel* into the eyes of patients to whom at the same time iodine is being administered internally, we not infrequently observe a strong corrosive action from the calomel; for the latter forms with the iodine excreted in the tears the very corrosive mercuric iodide (Schläfke). These two remedies, therefore, are incompatible.

In the cases of ulcers of larger size covered with exudation, as well as in the case of deep infiltration of the cornea, hot moist compresses applied several times a day for one or two hours at a time over the closed eyes, prove most efficient. With regard to the treatment of deep ulcers and of the cicatrices that follow ulcers, the general rules set forth under the head of affections of the cornea are applicable. A bandage should be applied only in case of imperative necessity (e.g., when there are ulcers penetrating deeply into the cornea); otherwise its use had better be avoided. It hinders the ready escape of the tears which are so copiously secreted, and, as it very soon becomes wet through with the secretion, it is likely to set up eczema of the skin of the lids.

In phlyctenular conjunctivitis, *general treatment*, conducted with due regard to the etiology, is of special importance. The child's nourishment should be strengthening and administered at regular hours. Sweets, especially candy, cakes, pies and pastry of all kinds should be forbidden, and only very plain, but substantial food allowed. It would seem that regulation of the diet alone may do a great deal toward effecting a cure in these cases. Care must be taken that the dwelling place be dry and well ventilated, and the child should be sedulously kept out in the open air, irrespective of any photophobia that may exist. Indeed, in any case, we

must not yield too much to this symptom of photophobia, and it would be quite a mistake to keep children in a dark room just because they shun the light.

In most cases the *blepharospasm* soon yields if the conjunctival trouble, which forms the basis of it, has been ameliorated by appropriate treatment. In case the spasm of the lid is particularly obstinate, we often obtain rapid improvement if we succeed in getting the child to open the eyes for some time at least once a day. We may secure this if we drop in cocaine a number of times in quick succession until the surface of the eyeball has become insensitive. Other effective measures are to douche the child all over with cool water or to dust dionin powder into the conjunctival sac. These procedures must be repeated daily until the spasm of the lids has yielded. The œdematous thickening of the lids produced by their spasm is combated by massaging them.

As invigorating measures, spongings with cold water are of service; also a sojourn in the country, especially at the mountains or the seashore. After the acute inflammation has run its course, the use of brine baths or of sea baths is of great service in preventing relapses. Unfortunately, the carrying out of all these regulations is only too often frustrated by the force of circumstances and by the poverty of the patients.

The medicinal treatment of scrofula consists in the administration of cod-liver oil and of the preparations of iodine, iron, arsenic and quinine. Calomel in broken doses (0.003–0.006 gm. three or four times a day) is also used with good results. It may be given in these doses for weeks at a time with advantage.

The sort of remedy employed and its dose must be adapted to each individual case. In case of a clinically demonstrable tuberculosis methodically continued tuberculin injections may be tried. These are quite helpful, especially in preventing relapses (Derby, Wilder, Norman). Furthermore, the cure of the eye disease is favorably influenced by treating any coexisting scrofulous affections, especially blepharitis, and also eczema of the face and of the nasal mucous membrane, diseased tonsils and adenoid vegetations. For blepharitis and eczema the application of white-precipitate ointment (1 to 2 per cent) or of ichthyol ointment (5 per cent) does good service. In blepharitis this ointment is smeared at night over the closed palpebral fissure. Eczematous spots upon the face are covered with a pledget of linen smeared with the ointment, and, to relieve eczematous coryza, the ointment is introduced from the anterior nares as far into the nose as possible and rubbed in. For relieving moist eczema of the face, we can also employ with great advantage a 5- to 10-per-cent nitrate-of-silver solution applied with the brush, after removal of the crusts, to the raw cutaneous surface, which thus becomes covered with a thin eschar, under which the raw spots heal rapidly. This application must be repeated at first daily, afterwards at intervals of several days, and be kept up as long as crusts continue to form.

Many of the patients, who generally belong to the poorest classes, suffer from head lice, the removal of which often has a strikingly favorable effect on the eye trouble.

489. Acute Exanthemata.—Besides eczema, the following exanthemata, both acute and chronic, are associated with diseases of the conjunctiva. *Measles* is regularly associated with a conjunctivitis. This appears under the form of an acute conjunctival catarrh, develops early (before the eruption of the exanthem upon the skin), and generally disappears of itself after two or three weeks without leaving any bad consequences behind. Only in exceptional instances does the conjunctivitis of measles take on a blennorrhœal or even a diphtheritic aspect (without actually turning into true blennorrhœa or diphtheria). In such cases the cornea is endangered (cf. § 481). During convalescence in measles, when the conjunctival inflammation had already become slight, sometimes numerous hordeola (§ 433) form in the Meibomian glands of both upper and lower lids (Fuchs). The purulent contents then discharge partly through the orifices of the glands, partly upon the inner surface of the lid after breaking through the tarsus and the conjunctiva.

In *variola,* smallpox pustules not infrequently develop upon the conjunctiva, generally upon the tarsal conjunctiva near the free border of the lid. Smallpox pustules which develop upon the conjunctiva of the eyeball near the limbus are dangerous from their setting up a purulent keratitis in the adjacent part of the cornea—a condition which should not be confounded with the ulcus serpens that develops in smallpox (see § 550). (For vaccine pustules, see § 496).

In smallpox and *scarlet fever* as well as in measles (see supra) we sometimes meet with a severe streptococcus infection leading to destructive diphtheritic conjunctivitis see (see § 481).

In the initial stages of *typhus* there is a hyperæmia of the conjunctiva

490. Chronic Exanthemata.—(1) Acne Rosacea Conjunctivæ.—This disease of the conjunctiva, described by Arlt, begins as follows: A minute nodule forms, with moderate symptoms of irritation, upon the limbus or at some distance from it in the cornea (Verhoeff). This efflorescence breaks down after some days, and the ulcer thus produced heals without leaving any visible cicatrix behind. This affection bears the greatest resemblance to the simple typical picture of phlyctenular conjunctivitis, and shares with the latter its peculiar tendency to frequent recurrence. On this account it is very tormenting to the patient. It is possible to make the differential diagnosis chiefly from the fact that conjunctivitis ex acne attacks only adults and those who are at the same time affected with acne rosacea. It is important to make the correct diagnosis, since otherwise we might labor in vain to prevent the recurrences—a thing which can be done only by a suitable and long-continued treatment of the acne rosacea. The conjunctivitis itself is most speedily cured by dusting in calomel and the corneal lesions may be cured by peritomy (Verhoeff).

(2) Pemphigus Conjunctivæ.—In this rare disease the conjunctiva, although reddened as a whole, displays one or two spots that are deprived of their epithelium and covered with a gray coating. While these spots are slowly undergoing cicatrization—a process attended with shrinking of the subjacent conjunctiva—spots of the same nature appear in other places. Thus there is produced a constantly increasing cicatricial contraction of the conjunctiva, whose progress, it is true, is very slow (extending over months and years), but is irresistible. The conjunctiva becomes whitish, cloudy, and tense. First, the retrotarsal folds vanish, then folds make their appearance, stretching from the lids across to the eyeball, and finally the lids are drawn in so that trichiasis results. The conjunctiva at the same time grows continually drier, and the lacrimal secretion dries up, owing to the fact that the excretory ducts of the lacrimal gland become occluded by the shrinking of the conjunctiva. Ultimately—although it may not be until years after the disease has begun—ulcers form upon the cornea, which later gets to be clouded all over, and likewise dry upon its surface. In the bad cases the lids at length become completely adherent to the eyeball, so that the cornea is permanently covered by the lids and the eye is incurably blind (symblepharon totale). Hence, the prognosis of pemphigus is very unfavorable—the more so as both eyes are always attacked. In some cases, however, the disease may remain stationary for a number of years (Buck). One disagreeable symptom is the overpowering fetor that often accompanies pemphigus (Stieren).

In pemphigus of the conjunctiva, contrary to what happens in pemphigus of the skin, bullæ are only exceptionally found, their place being taken by denuded areas in the conjunctiva. This is explainable from the anatomical character of the latter. Its epithelium is so soft and delicate that it cannot, like the epidermis, be lifted up in broad layers by serous exudation, but ruptures and is thrown off in the form of shreds, so that only as an exception do we chance to see vesicles, and these very small. The raw spot on the conjunctiva produced by the rupture of the vesicles soon become covered with a gray coating, as so often occurs in wounds of mucous membranes.

A further distinction between pemphigus of the conjunctiva and, indeed, of the mucous membranes generally, on the one hand, and pemphigus of the skin, on the other, consists in the fact that the process in the mucous membrane, in correspondence with the more delicate structure of the tissue, goes deeper in and hence leads to scar formation, while the pemphigus vesicles of the skin heal without leaving scars behind.

Pemphigus of the conjunctiva is rarely found in conjunction with eruptions of pemphigus upon the skin. More frequently there exists with the pemphigus of the conjunctiva an analogous affection of the mucous membrane of the mouth, throat, or nose. In these localities the pemphigus runs a course like that in the conjunctiva and may, particularly in the buccal cavity, lead to shrinking of the mucous membrane, and thus to stenosis of the mouth. But it may also happen that a lesion of the kind just described exists in the conjunctiva without pemphigus being present elsewhere in the body. That such cases as these, which were first described by von Graefe as *essential phthisis of the conjunctiva*, are also to be ascribed to pemphigus, is not certain, though probable.

Treatment has usually no power to restrain the process. Arsenic is administered internally for the pemphigus; and to make the patient easier, mucilaginous remedies are instilled into the eyes as in xerophthalmus (see § 475). A number of these cases are associated with syphilis, and in these antiluetic treatment may be given, although, as a rule, with little prospect of success. Transplantation of pieces from another mucous membrane into the conjunctival sac may be tried in order to replace the conjunctiva that has been destroyed.

(3) Lupus Conjunctivæ.—Lupus of the skin sometimes is continued over the edges of the lids upon the conjunctiva. In this situation it appears as an ulcer, the bottom of which is covered with granulations in which tubercle bacilli can be made out. Lupus of the conjunctiva must therefore be regarded as a form of tuberculosis of the conjunctiva (see § 495).

In the case of *other exanthemata*, too, for instance in macular and papular syphilides, in pityriasis, psoriasis, ichthyosis, herpes iris, dermatitis herpetiformis, lepra, etc., the conjunctiva is sometimes characteristically implicated. In *lepra*, yellowish, translucent, non-vascular nodules generally develop near the margin of the cornea. These lie both in the conjunctiva and in the subjacent sclera and sometimes grow out upon the cornea. When upon the latter they not infrequently have the appearance of a new growth. There is an associated iritis sometimes combined with the formation of lepra nodules in the iris; also cyclitis. The nodules in the different parts of the eye finally break down, and the eye is lost.

IX. Conjunctivitis Vernalis[12] (Spring Catarrh)

491. Objective Appearances.—Spring catarrh (Saemisch) is a chronic disease, persisting for years and setting up very characteristic changes in the conjunctiva either of the tarsus and at the limbus or of both parts simultaneously. The outlying portion of the bulbar conjunctiva and the retrotarsal fold show no particular changes. In marked cases the conjunctiva of the tarsus is covered with papillæ, which are broad and flattened so as to make the conjunctiva appear hard like a pavement of cobble-stones (Fig. 215). Over the whole lies a delicate, bluish-white film, as if a thin layer of milk had been poured over the conjunctiva, so that the injection is

[12] Synonym: Conjunctivitis æstivalis.

not very apparent. In the slight cases the tarsus shows only a bluish-white film without hypertrophy, or there is a peculiar, persistent fawn-colored congestion of the bulbar conjunctiva, along with a ciliary injection.

The changes at the limbus (Fig. 216), which most frequently appear on its nasal or temporal aspect in the interpalpebral area, consist of pale, grayish-red, uneven nodules of gelatinous aspect, in which there are sometimes found small white calcareous-looking dots (Tranta's dots). The nodules extend a little way into the cornea, from which they are sharply demarcated by an abrupt border; in the conjunctiva they extend further and are more sloping. The conjunctival vessels, running toward them are dilated.

FIG. 215.

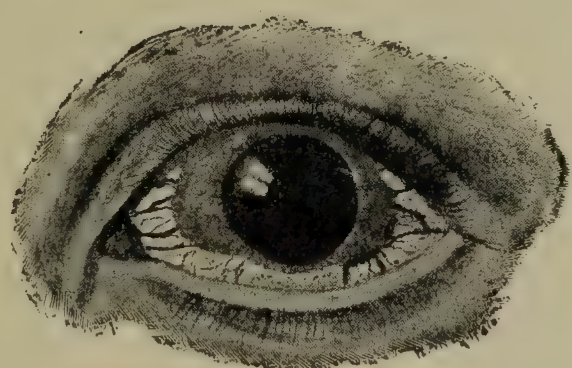

FIG. 216.

FIG. 215.—SPRING CATARRH. OUTGROWTH ON THE CONJUNCTIVA OF THE LIDS. Taken from a man eighteen years of age who had suffered from the disease for two years. The everted lid shows the pavement-like papillæ which are largest at the upper margin of the tarsus; from this point downward they become smaller and disappear completely at a short distance from the free border of the lid. The limbus was normal.

FIG. 216.—SPRING CATARRH. OUTGROWTH AT THE LIMBUS. Taken from a boy thirteen years of age who had had the disease for two years. The outgrowth is of unusual extent, being particularly broad and going all the way around the cornea. From it dilated veins run backward in the conjunctiva. In the conjunctiva of the upper lid nothing wrong could be made out except that it had a slightly satiny texture.

The cornea beyond its rim of nodules remains clear, except that sometimes we see in it, parallel with the margin of the nodule, a narrow gray stria like the arcus senilis, separated from the margin of the nodule by a narrow strip of transparent cornea. This opacity usually remains permanently even after the disappearance of the proliferations. Very rarely the proliferations advance a greater distance into the cornea under the guise of a tissue which is like a pannus but which contains very few vessels and hence is pale and gelatinous-looking. This may actually cover the whole cornea.

The papillæ on the tarsal conjunctiva are hard—sometimes as hard as cartilage. They are produced by overgrowth of the subconjunctival connective tissue which later undergoes hyaline degeneration. The presence of this hyaline layer is probably the reason for the bluish-white coloration of the conjunctiva. In the deeper layers there is present a somewhat more marked cell infiltration consisting mainly of plasma cells. The epithelium itself is diseased primarily as a result of the changes in the underlying tissue; it proliferates and sends offshoots into the deeper parts. The proliferations in the limbus show anatomical changes like those which the papillæ on the lid present. The secretion, which is usually scanty, is marked by the presence of numerous eosinophile cells (Herbert, Pusey), and their demonstration can be utilized for diagnosis in doubtful cases.

Spring catarrh is not a catarrh, as the not altogether suitable name chosen for it would indicate, but is a disease sui generis. It was first described by Arlt (1846), who regarded it as a peculiar variety of phlyctenular conjunctivitis. Subsequently Desmarres described it as "hypertrophie périkératique," von Graefe as gelatinous thickening of the limbus, Hirschberg as phlyctæna pallida. Saemisch was the first to lay stress on the characteristic exacerbation of the disease during the warm season, and therefore called it

spring catarrh, by which name it is at present commonly designated. Horner discovered the peculiar character of the tarsal conjunctiva, and thus completed the picture of the disease.

492. Symptoms and Course.—Quite as characteristic as the changes, objectively perceptible, are the statements given by the patients. They say that during the winter they experience little or no annoyance from their eyes, but as soon as the first warm days come in spring the eyes begin to grow red and to water; the patients are greatly tormented by photophobia, and especially by a constant itching in the eyes. The warmer the weather, the greater the intensity of the subjective troubles; conversely, the patients feel easier if, for instance, there occur in summer a series of cool, rainy days. In autumn the troubles once more abate, and during the cold season they disappear completely, only to begin anew in the following spring. The difference in the objective condition at different seasons of the year is considerably less than one would suppose from the great change in the patient's subjective state, and consists principally in the eyes' being free from discoloration in winter and injected in the summer, while the growths upon the conjunctiva appear but slightly smaller in winter than in summer.

Spring catarrh is a rare disease, chiefly affecting the male sex and the ages of boyhood and youth. Many patients seem to be in other respects quite healthy, while others, without being scrofulous, display, besides pallor of the complexion, multiple swellings of the lymphatic glands, especially on the neck and lower jaw. Almost always both eyes are attacked. The disease generally keeps on making its return annually for three or four years, and often longer still, for ten or even twenty years, until finally it becomes extinct, without leaving any marked trace of its presence behind. The prognosis is therefore good as regards the ultimate outcome, but bad as regards the duration, as up to the present time we know of no remedy for curing the disease or for preventing its annual return.

Differential Diagnosis—The papillary growths in spring catarrh are the reason for this disease's being commonly regarded as trachoma, and being treated with nitrate of silver or with bluestone. The result of this treatment is that the growths do not recede and the irritation of the eye is heightened. The papillæ in vernal catarrh are much larger (broader), harder, and also paler than in trachoma, and above all are distinguished by the bluish-white lining which is absent in papillary trachoma. Spring catarrh and trachoma sometimes occur simultaneously, especially in Palestine, where spring catarrh is fairly frequent (Butler). Particularly important in the differential diagnosis is the history. The extremely characteristic statements in regard to the return of the disease every year in the spring often establish the correct diagnosis before we have even looked at the eye. In hay fever, to be sure, there is also generally a return of the conjunctivitis every year in the spring (see § 452). But this recurrence is an acute one, and runs its course within a few weeks, while, on the contrary, the symptoms of spring catarrh last during the whole of the warm season. Moreover in the latter the respiratory symptoms of hay fever (coryza etc.), are absent.

Etiology.—Kreiblich and Dimmer conjecture that spring catarrh may be produced by sunlight, comparing it with the dermatoses thus caused. It is true that the limbic proliferations do recede under a bandage, but they also do so under a dressing that admits the light but keeps out the air. Climate may have an influence since change of residence has a favorable effect.

493. Treatment.—Inasmuch as we are unable to cure the disease, the *treatment* must be limited to the amelioration of the subjective symptoms. We combat the inflammation by instilling mild astringents, e.g., solutions of zinc (½ per cent), boric acid (3 per cent), ichthyol (1 to 2 per cent). For the itching the frequent instillation of a weak solution of acetic acid (5 drops of acidum aceticum dilutum to 10 gm. of water) does good service. Dionin is also said to afford much relief (Swift), and anæsthesin has helped in some cases. In many cases the dusting in of xeroform or the instillation of adrenaline has a good effect. For the proliferations in the limbus massage with precipitate ointment may be used. To shield them from the light we direct the patients to wear protective glasses (of grayish-smoke, euphos or Crookes glass). If we have patients of pale aspect and with swollen glands we administer arsenic and iron internally Furthermore, in summer the advice must be given to keep cool, use cold baths and showers, or reside in the mountains.

If the growths are of a considerable size they may be removed either by ablation or by means of electrolysis or carbon dioxide snow. Diathermy (§ 118) has also been employed and actinotherapy especially with radium has been used with success.

494. Amyloid Degeneration of the Conjunctiva.—This rare disease has been observed mainly in Russia and the countries adjacent, and was first described by Oettingen in Dorpat. It consists in a peculiar degeneration of the conjunctiva, by reason of which the latter becomes yellowish, translucent like wax, non-vascular, and very friable With this there is associated a considerable thickening of the membrane, so that it forms large swellings which look like new growths. The affection begins in the retrotarsal fold, and from this passes over to the conjunctiva of the eyeball and of the lids; in the lids the tarsus also is subsequently implicated in the degeneration. In a case that has lasted a long time the following clinical picture is found: The patient cannot open the eye because the two lids, transformed into large, misshapen swellings, cover it up. If the lids are drawn as far apart as possible, the wax-like conjunctiva is seen rising up under the form of a rigid prominence all about the cornea, which latter is either clear or is covered by pannus. Thick swellings, seated in the retrotarsal fold, protrude between the lids and the eyeball; the plica semilunaris also is enlarged until it forms a misshapen mass. These various swellings are so friable that they often tear when an attempt is made simply to separate the lids for examination, although when they tear they bleed very little. The disease runs a very chronic course, dragging on for years without any real inflammatory symptoms, until at length the patient is deprived of the use of his eyes by inability to open the misshapen lids.

The microscope shows that the degeneration of the conjunctiva originates from the subconjunctival cellular tissue. This at first is very abundantly infiltrated with cells (adenoid proliferation). Later there form in the tissue dully lustrous, homogeneous bodies, which are called by the name either of amyloid or of hyalin, according to the reaction that they give with stains. Hyalin may occur as a preliminary stage in the development of amyloid; but generally it represents a variety of tissue degeneration (*hyaline degeneration of the conjunctiva*) distinct from the amyloid, and showing no tendency to pass over into the latter. Hyaline and amyloid degenerations present almost precisely the same clinical picture, so that a positive distinction between the two can be made only by examining excised pieces of conjunctiva. Finally, calcification or ossification may take place in the degenerated mucous membrane.

The disease attacks people in middle life, and ordinarily both eyes are affected. Very frequently amyloid degeneration is preceded by trachoma of the conjunctiva, which, however, should not be regarded as the cause of the affection, inasmuch as the latter can

develop in eyes that previously were healthy. The actual cause of the disease is not known. In every instance it is a purely local process, for the individuals attacked by it are sound as far as the rest of the body is concerned, and do not suffer from amyloid degeneration of the internal organs, with which, therefore, amyloid degeneration of the conjunctiva has nothing at all to do.

Medical treatment is powerless against this disease. We must confine ourselves to removing the growths upon the conjunctiva to such an extent that the lids can be opened and vision thus rendered possible. It is by no means necessary—indeed it is not at all advisable—to remove by a radical operation all the diseased parts, since the portion of the growth that is left behind generally atrophies of itself afterward.

Conjunctivitis Petrificans.—Leber describes under this name a disease in which white spots appear in the conjunctiva which are produced by the deposition of lime and from which ulcers develop whose floor is rendered white and hard by lime deposits. This disease drags on for years with frequent remissions, and produces adhesion of the lids to the eyeball.

X. Tuberculosis of the Conjunctiva

495. In the conjunctiva tuberculosis ordinarily appears under the guise of ulcers located in the tarsal conjunctiva. The diseased lid even on external inspection looks thickened. The ulcer is either covered by grayish-red granulations (Fig. 217), or has a yellowish-red, lardaceous-looking base. In its vicinity small gray nodules (tubercule nodules) or outgrowths resembling a cock's comb are often found in the conjunctiva. The ulcer shows no disposition to heal; on the contrary, it spreads, although it does so very slowly. It may pass over to the conjunctiva of the eyeball; and even the cornea is sometimes covered over by a sort of pannus. In especially severe cases the ulcer does not remain confined to the conjunctiva, but eats through the entire thickness of the lid, so that even on external examination a deficiency of tissue is observable in the lid. Quite early in the disease the lymphatic gland in front of the ear becomes swollen; afterward the lymph glands about the lower jaw and in the neck also become enlarged The clinical picture afforded by the disease is sufficiently characteristic to make the diagnosis from it alone; but to be quite sure upon this point, we should remove bits of tissue from the ulcer and either demonstrate the presence of tubercle bacilli in them by the ordinary methods, or else, by inoculating them into rabbits' eyes, set up tuberculosis in the latter.

Tuberculosis of the conjunctiva generally attacks only one eye. The patient does not suffer pain; it is only by the swelling of the lid, the purulent secretion, and subsequently by the diminution of visual power, that he is annoyed and made aware of the existence of his trouble. The disease occurs, almost without exception, in young people, and runs an uncommonly chronic course, being often protracted over many years. Even after an apparently radical cure it shows a great tendency to recur, and it can, by infecting the rest of the organism, finally lead to death from tuberculosis.

Tuberculosis and *lupus* of the conjunctiva are to be regarded as essentially identical affections, in that both represent ulcerative processes produced and maintained by the presence of tubercle bacilli. In fact, the first cases of tuberculous conjunctival ulcers were described as primary lupus of the conjunctiva (i.e., lupus without coincident lupus of the skin) (Arlt). The two processes are distinguished only by external differences relating to their aspect and course. Thus, as a rule, lupous are distinguished from tuber-

culous ulcers of the conjunctiva by the fact that they have migrated from skin to conjunctiva, and that, like lupus of the skin, they show spontaneous cicatrization on one side while on the other the ulcer keeps advancing (see also § 490).

Tuberculosis of the conjunctiva may originate either in ectogenous or in endogenous infection. In individual cases, to be sure, one often can not determine which sort of infection is present. We should, e.g., think the infection *ectogenous* if a quite circumscribed tuberculous ulcer were situated on the conjunctiva of a man in whom clinically demonstrable evidences of tuberculosis were not present elsewhere. For example, a particle of dust carrying bacilli might get into the conjunctival sac and by its sharp angles produce a small superficial lesion of the conjunctiva, which is thus infected (tubercle bacilli, according to the researches of Valude, not penetrating into the conjunctiva when the epithelium is intact). In favor of an infection of this sort is the fact that we so often see tuberculous ulcers begin in the region of the sulcus subtarsalis, where small foreign bodies are so readily retained. In such cases, the conjunctival tuberculosis may represent the only focus of the disease in the body—*primary* tuberculosis of the conjunctiva. This may remain for a long time confined to the conjunctiva; indeed, in exceptional cases, it may even heal spontaneously. The rule, however, is for tuberculosis to spread from here to other parts of the organism. This extension may be by way of the lymphatic circulation, the neighboring lymph glands being first attacked by the tuberculosis. Or the disease may extend by continuity, the tear passages first and then the nasal mucous membrane being infected by the tears containing the bacilli. Those cases of conjunctival tuberculosis are regarded as *secondary* in which there is at the same time tuberculosis of the internal organs (especially the lungs). The eye may then be infected by the patient's getting some of his own tuberculous sputum into it. Or the tuberculosis extends by continuity, a tuberculous or lupous affection of the nasal mucous membrane passing to the conjunctiva through the tear passages. (Not infrequently therefore, we find conjunctiva, lacrimal sac, and nasal mucous membrane attacked at the same time by tuberculosis, and careful study of the history of the case and exact examination generally render it possible to ascertain whether the affection has passed from conjunctiva to nose or vice versa.)

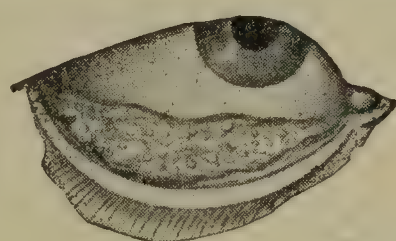

FIG. 217.—TUBERCULOSIS OF THE CONJUNCTIVA.

Taken from a girl eighteen years of age who looked healthy but had a clearly demonstrable affection of the apices of both lungs. The conjunctiva of the lower lid is to a large extent occupied by a grayish-red outgrowth demarcated from the rest of the conjunctiva by a border which rises abruptly and in places is actually overhanging. On its surface the outgrowth bears numerous grayish-coated ulcerated areas. On the conjunctiva of the upper lid there is merely a little papillary hypertrophy. The gland in front of the ear appears on palpation to be the size of a pea.

An *endogenous* (hæmatogenous) infection is present when the tubercle bacilli have been carried into the conjunctiva through the circulation. This sort of infection may be assumed to exist especially in those cases in which the disease occurs under the form of discrete miliary foci in the conjunctiva (see § 473). A like origin obtains for the *tuberculides* of the conjunctiva described by Bayer, Guzmann, and Friede. These are grayish or whitish-yellow nodules, scarcely as large as the head of a pin, situated in the bulbar conjunctiva. They occur simultaneously with tuberculides on the skin, are unattended by symptoms of irritation, do not break down, and disappear after lasting a short time. They show the microscopic structure of miliary tubercles (Salzmann).

Treatment consists in radically excising or curetting the ulcer and then cauterizing the raw surface; using here the galvano-cautery loop or lactic acid (pure or 50 per cent), and in the latter case taking care that none gets on the cornea. If the tuberculous foci are so extensive that complete removal would be followed by great deformity of the lids or marked symblepharon, we may try to dissipate the tuberculous tissue by tuberculin injections or phototherapy. For after treatment it is best to apply iodoform for

a long time to the conjunctival sac in powder or 10- to 20-per-cent ointmen —iodoform being specially efficacious in tuberculous processes.

496. Ulcers of the Conjunctiva.—Ulcers of the conjunctiva, besides occurring as result of tuberculosis, are also observed in the following conjunctival affections:

(*a*) As one of the symptoms of a conjunctivitis, an example being the minute ulce originating in the efflorescences of phlyctenular conjunctivitis or those which hav given its name to the pustular form of catarrh (see § 451.)

(*b*) In squirrel-plague conjunctivitis (see § 477).

(*c*) In sporotrichosis of the conjunctiva, and Parinaud's conjunctivitis (see § 477

(*d*) After the separation of necrotic portions of the conjunctiva (in diphtheria, c after injury with heat, caustics, or too strong medicinal applications).

(*e*) As a result of exanthemata; ulcers, for instance, which are derived from a vario lous pustule, aphthae, or from the rupture of a bulla of pemphigus upon the conjunctiva also vaccine ulcers due to transfer of the vaccine poison from a vaccine pustu (Purtscher). These are heavily coated ulcers and are attended with considerable swel ing of the pre-auricular lymph gland (cf. §§ 420 and 557). They may be transmitte from vaccine ulcers of the lids (see § 420) and lead to perforation of the cornea or com plete symblepharon (Bedell).

(*f*) Upon the tarsal conjunctiva there is quite often a small raw spot, from whic rises a little mass of granulations. Here we have to do with a chalazion which has broke through on the inner side of the lid. As a rule, a slender sound can be introduced throug the granulations into the cavity of the chalazion.

(*g*) Ulcers in glanders.

(*h*) Ulcers due to the breaking down of an epithelioma of the conjunctiva.

(*i*) Syphilitic ulcers. Generally we have here to do with those losses of substanc which have arisen from the breaking down of an initial sclerosis. These, as a rule, ar situated near the free border of the lids, but are also observed in the retrotarsal fold an even in the conjunctiva of the eyeball. The transmission of syphilis appears to tak place most frequently by kissing, and in small children also by the practice which man nurses have of moistening the agglutinated edges of the lids with saliva in order to ope them. In some countries it is the custom to remove foreign bodies from the conjunctiva sac by licking them out with the tongue. Occasionally, also, syphilitic ulcers have bee observed, which were produced by the breaking down of gummata of the conjunctiva (Hirschberg). Syphilitic ulcers of the conjunctiva are among the greatest of rarities Still rarer is soft chancre of the conjunctiva.

XI. Injuries of the Conjunctiva

497. Foreign Bodies in the Conjunctiva.—Small-sized foreign bodies, like grains of dust, particles of coal or of ashes, which so often get into the eye during a railroad journey, the wing cases of small beetles, etc., fall first upon the surface of the eyeball, are brushed away from this spot by the movement of the upper lid, and then generally stick to the inner surface of the latter at a spot not far from its free border, where a shallow furrow, the sulcus subtarsalis, that runs parallel to the edge of the lid, catches the foreign body. The pain which such a foreign body causes, and which is often quite considerable, does not originate in the conjunctiva itself, which has very little sensitiveness, but in the cornea, inasmuch as with every movement of the lid the foreign body is carried over the cornea and scrapes it. Hence the pain is absent as long as the eye is kept quietly closed. It is easy to remove the foreign body after the lid is everted. Sometimes the

foreign body, especially if of scale-like form adheres to the eyeball, instead of the lid, lodging particularly on the limbus where it may be mistaken for a phlyctenule.

In other cases, small, sharp-pointed foreign bodies penetrate into the conjunctiva, and may remain there a long time. This is particularly true of powder grains and little particles of lead or stone thrown into the eye by an explosion. Foreign bodies of this sort remain fixed in the conjunctiva of the eyeball without giving rise to any further irritation, and may therefore be left in situ. They can be removed if necessary by cutting them out along with the conjunctiva in which they are imbedded. Larger-sized foreign bodies are retained in the conjunctival sac only when they get into the upper retrotarsal fold. In this spot they stay, remaining still even during the act of winking, cause no irritation of the cornea, and therefore produce at first but little trouble. After some time has elapsed they begin to excite the symptoms of chronic conjunctival catarrh and ultimately may produce a circumscribed purulent inflammation with the formation of ulcers and cockscomb-like proliferations. If the lid is everted this hypertrophied part of the fornix protrudes and with it the foreign body, which previously had lain hidden from view, but now can readily be removed with a forceps.

Many foreign bodies act directly as caustics (see §§ 499, 500).

By the entrance of caterpillar hairs (see § 730, Ophthalmia Nodosa) or of plant hairs into the conjunctival sac, acute inflammations of the conjunctiva develop often with the formation of small nodules in the conjunctiva inclosing the hairs.

Sometimes foreign bodies are introduced into the eye purposely. Chief among these are what are called crab's eyes—lapides cancrorum. These are flat, calcareous concretions derived from the stomach of the crab, which are in great favor among the laity for removing foreign bodies from the eye. The crab's eye is introduced between lid and eyeball, and then pushed across the cornea, carrying with it, it may be, mechanically any foreign body there present. (In this country, eye stones—the flat opercula of certain molluscs—are used for the same purpose). Sometimes in performing this manipulation the crab's eye slips into the superior fornix and remains there unnoticed We may then find it there months or even years afterwards, entirely imbedded in the outgrowths of the conjunctiva, which has undergone chronic inflammation. Foreign bodies, like sand, ashes, scrapings from a wall, etc., or irritant substances like ipecac are also intentionally introduced into the eye in order to simulate eye disease, and so avoid military service. The trick is often easy to recognize from the fact that the conjunctivitis thus set up is confined to the lower lid, upon which the foreign body was placed.

498. Wounds of the Conjunctiva.—These are quite common and are often associated with marked ecchymosis (see § 509). One should never neglect in such cases to examine carefully to make sure whether the deeper parts are injured or not. Conjunctival wounds heal readily. If they gape much, they can be brought together with fine stitches, the neighboring conjunctiva being undermined if necessary to draw the lips of the wound together without tension.

499. Burns and Corrosive Injuries of the Conjunctiva.—Burns are the result of hot water or steam, corrosive gases (chlorine, mustard gas, etc.), hot ashes (especially cigar ashes), exploding powder, flames striking against the eye, molten metals, etc. Of corrosive injuries, which may be produced both by acids and by alkalies, those that arise from the action of lime are

the most frequent, the lime getting into the eye usually under the form mortar, or sometimes of chlorinated lime (Denig; cf. also § 500). As rule the injury is greater in the lower part of the conjunctival sac than in th upper, because the eyeball turns up the moment the injury is inflicted, s that its lower part is exposed in the palpebral fissure, and also because th hot or corrosive liquid flows down at once into the lower part of the con junctival sac.

If the point of an indelible pencil gets into the eye, the coloring matter is dissolve staining the eye a violet blue. This coloration disappears if the foreign body is remov at once, although some remains for several days at the immediate site of the latte But if the bit of pencil remains in the eye for some days it acts as a caustic, causi obstinate ulceration of the conjunctiva, injury of the cornea (Salzmann), and ev cataract and panophthalmitis (Stephenson). The baneful effects of the aniline prepar tions can be averted if a 5- or 10-per-cent solution of tannin is at once instilled (Vogt).

The action of burns is the same as that of caustics; the conjunctiva the affected spots is destroyed and converted into an eschar. These spo stand out as gray or white patches in the midst of the reddened and swolle portions of the conjunctiva that are not escharotic. The eschars separat in consequence of a delimiting suppuration, and the resultant granulatin losses of substance heal by a drawing in over them of the neighborin healthy conjunctiva. The final result is therefore always the formation of cicatrix. This may cause a diminution in size of the conjunctival sac, o if of great extent, an adhesion of the lids to the eyeball (symblepharon).

Corrosive injuries, especially those affecting the limbus, quite fre quently cause a rise of tension, which develops several days after the injur and may last a week or more (Kuemell).

That the effect of burns, e.g., from particles of molten metal flying into the eye is often quite superficial is due partly to the cooling action of the tears, partly to th fact that the vaporized moisture forms a layer between the hot body and the eye (Eales

The *prognosis* of an injury by burns or caustics with regard to th preservation of sight depends primarily upon the condition of the cornea which, indeed, is always implicated in any extensive lesion of the conjunc tiva. Next, the losses of substance in the conjunctiva itself must be con sidered since the adhesions that develop from them may more or less harm the function of the eye. The amount of injury done often cannot be deter mined in the first two or three days, so that the prognosis must be guarded

The prime *therapeutic* requisite, when we get an eye under treatmen soon after injury by caustics, is the complete removal of any corrosive sub stance still present. We remove solid particles with a linen pledget or a for ceps, and then wash the conjunctival sac out thoroughly with a gentl stream of water. In the further course of an injury by burns or caustics, w must restrain subsequent inflammation by cold compresses, atropine, bandage, etc. If the tension rises we may have to use miotics. After sepa ration of the eschars, our aim must be to confine the resulting adhesion within the smallest limits. For this purpose we repeatedly draw the lid away from the eyeball to prevent adhesion of the two opposed raw surfaces

If the defect extends so far as to implicate the retrotarsal fold, we try by transplanting a flap of skin or mucous membrane to prevent the formation of an adhesion between the lid and eyeball starting from the fornix (symblepharon posterius). If, nevertheless, this does develop, it must be removed, as far as can be, by operation later. Denig, believing that the secondary infiltration of the cornea is due to chemical decomposition taking place in the burned portions of the conjunctiva, recommends that in severe cases these burned portions be removed at once and replaced by grafts taken from the mucous membrane of the mouth.

500. Traumatic Conjunctivitis.—After the action of irritants on the eye an acute traumatic conjunctivitis is produced evidenced by intense reddening of the conjunctiva with great photophobia, lacrimation, and pain, with which is associated in violent cases œdematous swelling of the lids. Such irritants are acrid vapors, liquids,or dust-like particles which get into the eye either by accident or as part of the day's work in certain industries. Examples of this occupational conjunctivitis are seen in cigar-makers, men whose work exposes them to metal, stone or emery dust, workers on certain kinds of wood, those engaged in preparing aniline dyes and other chemicals, workers in pitch or those employed on tarred roads, hop-pickers, oyster-shuckers, etc. A very violent, but transient conjunctivitis is produced by eel's blood spurting into the eyes. Some of the irritants mentioned, e.g., the aniline dyes, also cause discoloration of the conjunctiva (§ 499). Discoloration without marked irritation occurs from the impact of silverdust in silver polishers (argyrosis). For the physician it is important to know that chrysarobin, which is used as a remedy (especially for psoriasis), may cause acute conjunctivitis, and must then be discontinued. Under the same guise of an acute traumatic conjunctivitis occurs the conjunctival inflammation which follows the action of intense light, e.g., after dazzling by the reflection from snow (snow blindness) or by the electric arc light (ophthalmia electrica). (See § 87.)

XII. Pterygium

501. Symptoms and Course.—A pterygium is a triangular fold of mucous membrane which extends from the conjunctiva of the eyeball to the cornea, either at the inner or the outer side of the latter (Fig. 218). The blunt apex of the triangle lies in the transparent portion of the cornea, and is solidly and immovable united to it. The base of the triangle spreads out in the conjunctiva of the eyeball, and passes into it without there being any sharp line of division between the two. The apex is called the head, the base the body, of the pterygium. The part that lies between the two and corresponds to the margin of the cornea is the neck of the pterygium. Here the limits of the fold of conjunctiva are the most sharply defined, its borders being inverted in such a way that a small sound (*S*, Fig. 218) can be pushed beneath them for a short distance. A pterygium of recent origin is succulent and abounds in vessels which run converging from the base to the apex and impart to the pterygium its red color. In fact, from its similarity in form and vascularity (venation) to the wings of many insects (Hymenoptera), the name of pterygium is derived.[13] The fold of conjunctiva forming the pterygium is tightly stretched, so that there are produced a number of radially disposed furrows or flutings; furthermore, in pterygia which are situated at the inner side of the eye the plica semilunaris is often

[13] From πτερύγιον, the diminutive of πτέρυξ a wing.

quite obliterated and is included in the body of the pterygium (Fig. 218).

In the course pursued by a pterygium two stages must be distinguished. In the first the pterygium keeps on gradually growing, for years it may be, toward the centre of the cornea, which it may finally reach or even pass (*progressive pterygium*). Finally, the pterygium comes to a standstill so as to remain permenently attached to the same point upon the cornea (*stationary pterygium*). Whether in any given case we are dealing with a progressive or a stationary pterygium is determined mainly by the character of the apex of the growth. In the progressive stage this appears surrounded by a gray non-vascular zone which looks thick and gelatinous. In a stationary pterygium this marginal zone is found to be thin and cicatricial looking, and the whole pterygium is thin, pale, nearly destitute of vessels, and tendinous.

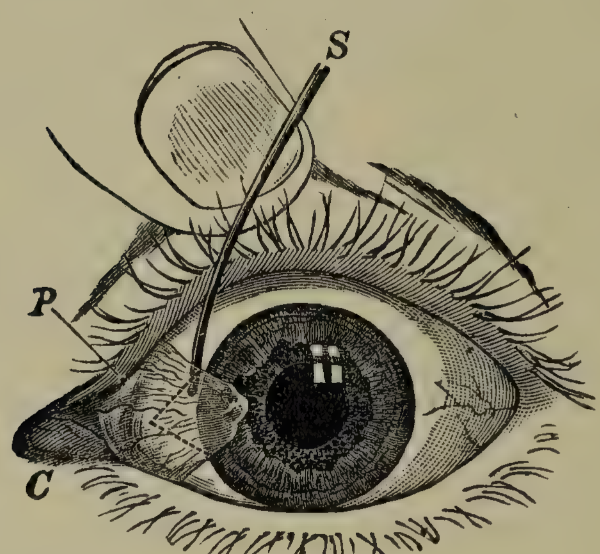

Fig. 218.—Pterygium.

A sound, *S*, is carried beneath the edge of the pterygium, *P*. The dotted line shows the way in which the section is made in removing the pterygium. *C*, caruncle. The adjacent plica semilunaris has been flattened out by the tensive force of the pterygium, and is hence invisible. *P*, upper punctum lacrimale.

Pterygium occurs only in that part of the cornea corresponding to the interpalpebral fissure. It is found most frequently on the inner side of the cornea; when there is already one in this spot another may form on the outer side also. The two pterygia may even meet in the centre of the cornea. A genuine pterygium is practically never observed at the upper or the lower margin of the cornea; on the other hand, it is not infrequently the case that both eyes are attacked simultaneously by pterygium, so that we sometimes see patients who have four pterygia, one on the outer and one on the inner side of either cornea.

A pterygium when small causes no trouble. When large it is disfiguring especially if, as happens often, it is inflamed so as to be reddened and swollen, and in this case it is associated also with moderate symptoms of irritation. When a pterygium has grown so far that it enters the pupillary area, an impairment of sight is produced, which increases as the pterygium advances. A large pterygium by the traction it exerts may impair the mobility of the eye and cause diplopia.

502. Etiology.—Pterygium is found especially, but no means exclusively, in elderly people and especially in those who are much exposed to dry heat, bright sunlight, wind, or dust, as in the case of farmers, coachmen, masons, stonecutters, cooks, seamen, etc., while among the well-to-do classes pterygium is comparatively rare. It seems referable to long continued irritation causing a chronic and progressive shrinking and degeneration in the bulbar conjunctiva and the conjunctiva corneæ, and leading to the formation of a tense fold of conjunctiva which is drawn up gradually over the cornea.

Pterygium and pinguecula have a like situation and probably a like causation. External irritation certainly plays a part in the formation of both. The formation of a pterygium is preceded by a shrinking of the corresponding area of the conjunctiva, for if we see a pterygium at the very earliest stage of its development we can note the tension of the conjunctiva and the displacement of the semilunar fold. The fact that the conjunctiva is stretched tightly over the cornea accounts for the triangular form of the fold, its incurved margin, and its great tension in a horizontal direction.

A pterygium consists mainly of fibrillar connective tissue, which is covered with the epithelium of the conjunctiva. The apex of the pterygium, however, is often covered by the corneal epithelium, so that the pterygium may be said to force its way underneath the latter (Fig. 219, at *g*). In the tissue of the pterygium are found new-formed tubular

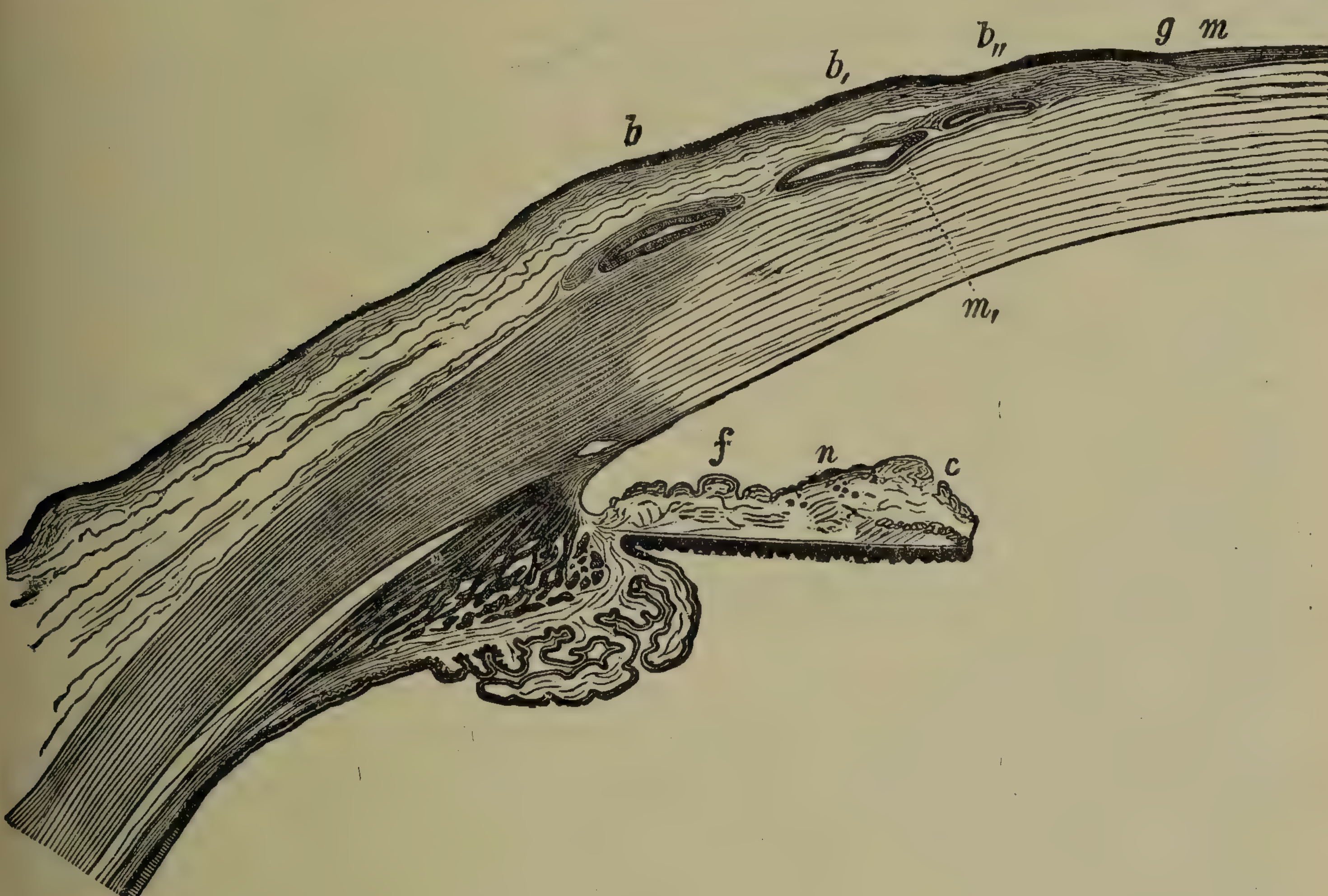

FIG. 219.—LONGITUDINAL SECTION THROUGH A PTERYGIUM. MAGNIFIED 12 × 1.

The apex of the pterygium is marked by the termination of Bowman's membrane at *m*. At *g* is the boundary between the epithelium of the conjunctiva and of the cornea. At b, b_1, b_{11}, lie cavities lined with epithelium. These are the cross sections of spaces which jut in from the lateral margin of the pterygium, between the latter and the surface of the cornea, and which are divided by the section close to their blind extremity. Beneath them at m_1, are to be seen remains of Bowman's membrane. The eye had a wide pupil, consequently the iris in the section looks short and thick; its pupillary portion rises abruptly from the anterior capsule of the lens and indeed actually is overhanging so as to conceal the retinal pigment layer of the iris (compare *p*, Fig. 20). Conspicuously visible on the anterior surface of the iris are the very protuberant contraction folds, *f*, also the orifice of a crypt, *c*, and lastly an accumulation of pigmented cells, *n*, in the superficial layers, which appears as a brown spot in the otherwise blue iris (nævus iridis).

glands and also larger spaces lined with epithelium (b, b_1, b_{11}); from both of these small cysts may develop. Bowman's membrane, where it lies beneath the pterygium, is destroyed; indeed, the uppermost lamellæ of the cornea itself are replaced here and there by the tissue of the pterygium. This explains why, even after the ablation of the pterygium, the cornea does not regain its normal transparency.

503. Treatment.—This consists in an operation (see § 913); which is indicated in every case of progressive pterygium. Even though the pterygium is still small, we cannot be sure that it will not grow into the pupillary area of the cornea. Hence we prefer by performing operation to guard in

time against injury to the eyesight. If the pterygium has approached sufficiently near to the centre of the cornea to cause disturbance of vision, this disturbance is indeed rendered less by the operation of ablation, but is not completely done away with, since those spots of the cornea, which were occupied by the pterygium, never again become perfectly transparent. In any case, the associated symptoms of irritation, the restriction of mobility, and the disfigurement are removed by the ablation. A stationary pterygium does not necessarily require operation; in this case we shall be guided mainly by the wishes of the patient with reference to the removal of the disfigurement, etc.

504. **Pseudo-Pterygium (Cicatricial Pterygium).**—Sometimes we observe, as a result of inflammatory processes, fixation of a fold of conjunctiva upon the cornea, giving a picture similar to that of true pterygium. For example, there may be a gonorrhœal conjunctivitis, with great chemosis and a pretty large marginal ulcer of the cornea. The chemotic protuberance of the conjunctiva becomes applied to the surface of the ulcer and adheres to it. After the inflammation has abated, the swelling of the conjunctiva goes down, and the chemotic protuberance disappears; but, at the spot where union with the cornea has taken place, the conjunctiva remains permanently fixed to the latter. We then see a triangular fold formed of conjunctiva extending over the limbus and upon the cornea, and attaching itself there. Ordinarily, at that part of the fold corresponding to the limbus a small sound can be passed all the way beneath the fold, a sign that the latter is adherent to its bed at its apex only and not throughout its whole extent. This is the most important distinguishing mark between a true and a false pterygium; another consists in the fact that a pseudo-pterygium does not keep on growing over the cornea, as a true pterygium does, but remains fixed forever to the spot at which it first became adherent. In its origin and its behavior a pseudo-pterygium is more like a symblepharon than a genuine pterygium.

Pseudo-pterygia are observed not only after gonorrhœal conjunctivitis, but also after diphtheria, burning by heat and by caustics, prolapses of the iris, the removal of new growths, etc. It is evident that they may develop not only at the outer and inner sides, but at any side whatever of the cornea. The pseudo-pterygia remaining after gonorrhœal conjunctivitis are usually found above, those produced by burns, etc., at the lower part of the cornea (in the area corresponding to the palpebral fissure).

Another kind of pseudo-pterygium is that which develops after a chronic superficial ulceration of the marginal portions of the cornea (*keratitis marginalis superficialis*, Fig. 241). Owing to the cicatrization which follows the ulcerative process the conjunctiva is drawn up steadily over the cornea. These pseudo-pterygia are very similar to true pterygia, for like the latter they keep on growing slowly over the cornea, and unlike other pseudo-pterygia are not completely perforate at the limbus. The differential diagnosis between these and the true pterygia can be made only in case we find the ulcerative process or its sequelæ (a superficial corneal opacity) upon the marginal portions of the cornea not implicated in the pterygium.

Small pseudo-pterygia may without disadvantage be left undisturbed; larger ones we generally remove in the same way as genuine pterygia, and unite with stitches the wounds left in the conjunctiva. In those cases in which the pseudo-pterygium is not adherent to the surface of the eyeball at a spot corresponding to the limbus, ablation and the use of the suture may be dispensed with; it is sufficient simply to free the point of the pseudo-pterygium from the cornea, upon doing which the former retracts of itself and disappears by a process of atrophy.

It sometimes happens that an *old pannus* which has already been transformed into connective tissue is united to the subjacent cornea only by loose cellular tissue, and thus acquires a certain freedom of movement, so that it can be shifted this way and that upon its bed along with the conjunctiva of the eyeball. So in this way, too, a picture similar to that of a pterygium may be produced.

XIII. Symblepharon

505. Characteristics.—By symblepharon,[14] we understand a cicatricial adhesion between the conjunctiva of the lids and the conjunctiva of the eyeball. In that case, when we attempt to draw the lid away from the eyeball we observe that in one or more places bands extend from the inner surface of the lid to the surface of the eyeball, and that these become tightly stretched and prevent us from drawing the lid away completely. These bands generally look tendinous, more rarely fleshy, and may be attached not only to the conjunctiva scleræ, but also to the surface of the cornea itself. If the adhesion between the two conjunctival surfaces extends so far

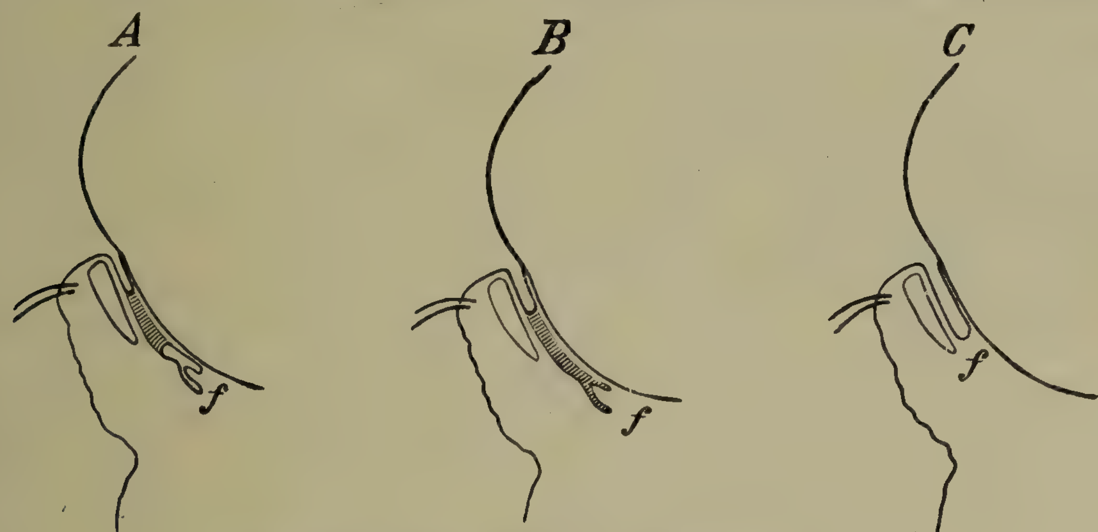

Fig. 220.—Symblepharon (Schematic).
A, symblepharon anterius; *B*, symblepharon posterius by adhesion; *C*, symblepharon posterius by cicatricial shrinking; *f*, fornix.

peripherally as to reach into the fornix, we call this *symblepharon posterius* (Fig. 220 B). If the adhesion does not extend as far as this, so that the cicatricial bands stretch like a bridge between the lid and the eyeball, and we can introduce a sound beneath them and carry it along the fornix between the lid and the globe, it is then called *symblepharon anterius* (Fig. 220 A). This distinction has been made for practical reasons, inasmuch as symblepharon anterius can be readily cured by an operation, while symblepharon posterius can be cured with difficulty or not at all. *Symblepharon totale* is a total adhesion between the lids and the eyeball—a condition which occurs but rarely.

506. Etiology.—Symblepharon develops whenever two opposed spots of the conjunctiva of the lid and of the eyeball have raw surfaces which come into contact with each other, and in consequence become adherent. Such a process of adhesion results of necessity when the two raw surfaces extend up into the fornix and there become continuous, since two raw surfaces which meet at an acute angle always begin to unite at this angle of junction. Causes which may give rise to the formation of raw surfaces upon the conjunctiva are burns by the action of the heat, burns from caustic substances, diphtheria, operations, ulcers of all kinds, etc.

The expression symblepharon is also employed in a somewhat different sense to denote the contraction of the conjunctiva which occurs as a result

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[14] From σύν, together, and βλέφαρον, eyelid.

of its gradual *shrinking*. In this case it is not a question of an adhesion between two raw conjunctival surfaces, but of a gradual diminution in size of the conjunctival sac. The folds of the retrotarsal region are first smoothed out; the conjunctiva of the lid passes to the eyeball directly (Fig. 220 C) and whenever the lid is drawn away is pulled out into tense perpendicular folds. In advanced cases the conjunctival sac is reduced to a shallow groove lying between the eyeball and the lid. Since contraction of the conjunctival surface due to shrinking always shows itself first in disappearance of the retrotarsal fold, all these cases belong to symblepharon posterius. This kind of symblepharon is observed principally after trachoma and in the rare cases of pemphigus of the conjunctiva.

Symptoms and Treatment.—Very slight cases of symblepharon produced no bad *results* worth mentioning. When there are more pronounced adhesions, the excursions of the eye are hindered, and thus possibly diplopia may be produced just as in the case of pterygium. Inasmuch as with the movements of the eye traction is made at the site of the adhesions, the eye gets into an irritated condition. If the adhesions extend into the region of the palpebral fissure they become disfiguring, and if they extend as far as the cornea they may injuriously affect the sight. Sometimes the lids are so fastened down by extensive adhesions that their complete closure is impossible, and lagophthalmus is produced with its consequences that are so baneful to the cornea. Total symblepharon is obviously associated with complete blindness (or at most with perception of light).

Treatment is by operation (see §§ 916 and 917).

For *epitarsus*, see page 453.

XIV. Xerosis

507. Symptoms.—Under the name of xerosis[15] conjunctivæ we designate an alteration of the conjunctiva, generally occurring in patches and consisting in a dryness of the membrane. The surface of the conjunctiva at the xerotic spots glistens like fat and is of a whitish color, and looks either like epidermis or as if it were covered with dried foam. If the change affects the scleral conjunctiva, the latter is thicker, less pliable, and lies in stiff folds. The tears flow over the diseased spots without moistening them, and in marked cases the secretion of tears is absent. An analogous change is also observed in the cornea, the surface of which looks dull, lustreless, and dry, while the parenchyma of the cornea at the same time is found to have lost its transparency (xerosis corneæ).

Etiology.—Xerosis occurs in two groups of cases. *In one it results from a local affection of the eye.* This occurs—

(*a*) In *cicatricial degeneration* of the conjunctiva. It is observed most frequently as the final outcome of trachoma, and also, but more rarely, after diphtheria, pemphigus, burns, etc. It begins in isolated spots, but may ultimately spread over the entire conjunctiva, and over the cornea also. In the latter case, since the xerotic cornea grows opaque, the eye becomes blind. This form of xerosis is incurable.

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[15] From ξηρός, dry.

(*b*) *Deficient sheltering* of the conjunctiva, so that the latter is constantly in contact with the air. This occurs in ectropion and in lagophthalmus (incomplete closure of the lids). In the former case the exposed portion of the tarsal conjunctiva, in the second case that part of the scleral conjunctiva and of the cornea lying in the course of the palpebral fissure, becomes covered with a thickened, dry, epidermoid epithelium, by means of which these parts protect, so to speak, their deeper layers against desiccation. Such cases can be helped only when some operation can be done that will provide the exposed conjunctiva or cornea with its normal shelter again.

In the second group of cases xerosis appears as a symptom of a *general disease* of a peculiar charcter, the real nature of which is as yet unknown. It occurs pre-eminently in persons who are depressed in nutrition, occurring, for example, very frequently as the result of the malnutrition produced by the war (Rutherford). The xerosis in these cases begins at the outer and inner parts of the conjunctiva of the eyeball under the form of small triangular areas which are covered by what looks like a fine dried foam and are not wet by the tears (Bitot). As a rule there is present at the same time a peculiar disturbance of sight, namely *hemeralopia* or night blindness (see § 261).

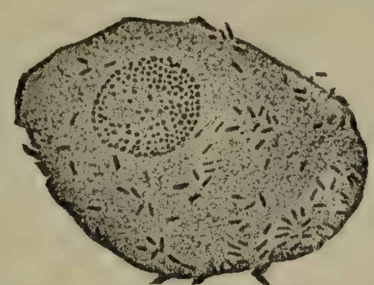

Fig. 221.—Xerosis Conjunctivæ. Magnified 820×1.

Epithelial cell scraped from the xerotic conjunctiva and stained by Gram's method. The nucleus of the cell is marked by the fact that numerous granules of keratohyalin lie immediately about it, although still in the protoplasm of the cell. Upon the cell are seen xerosis bacilli.

If it is adults that are affected, the xerosis and hemeralopia pass off within a few weeks. In small children, on the other hand, the disease often takes a malignant course. The xerosis spreads from the area occupied by the palpebral fissure over the whole conjunctiva of the eyeball and then over the cornea too. The latter becomes cloudy and suppurates under the guise of *keratomalacia* (see § 559), and the little patients die with the symptoms of a severe general disease. For the treament see §§ 475, 558, 559.

The separation, first made by Cohn, of xerosis into a variety dependent upon local causes and one caused by a general disease corresponds nearly to the ordinary division into xerosis parenchymatosa and xerosis epithelialis. In local xerosis the mucous membrane is diseased in its deeper as well as in the superficial lyers (xerosis parenchymatosa); while in xerosis produced by a general disturbance of nutrition the change affects the epithelium only (xerosis epithelialis). Many also make a distinction between xerosis partialis (sive glabra) and xerosis totalis (sive squamosa).

The anatomical changes which underlie xerosis mainly affect the epithelium. The uppermost layers of epithelial cells undergo keratosis, the protoplasm of the succeeding layers containing abundant granules of keratohyalin (Fig. 221). In consequence the epithelium appears thickened, whitish, cloudy, and epidermoid. Moreover, it is covered with the sebum-like secretion of the Meibomian glands, and hence assumes a fatty character, in consequence of which the tears do not adhere to it. To this circumstance the peculiar dry appearance is chiefly owing, for if the diseased spots are freed from their fat by pencilling with soap they can be wetted by the tears (Leber).

Reymond and Colomiatti and shortly afterward Kuschbert and Neisser described as occurring in xerosis a micro-organism, the bacillus of xerosis, which is extremely like the diphtheria bacillus. This is found under the form of short rods adhering in great

quantities to the surface of the epithelial cells (Fig. 221). It is, however, neither the caus of xerosis, nor indeed characteristic of the latter. It occurs frequently in the conjunctiva sac anyhow, and simply seems to find in the dying epithelium of the xerotic spots specially favorable soil for growth, so that it develops there in great quantities. Th real cause of idiopathic xerosis is probably not an infection, but a disturbance of nutri tion (see § 261).

The *cause of the dryness* of the conjunctiva in xerosis is not, as has been believed the deficiency of tears. In the beginning of the disease, as long as only small isolatec spots of the conjunctiva are xerotic, we often find actually an increase in the lacrima secretion. Per contra, xerosis of the conjunctiva has never been known to occur after extirpation of the lacrimal gland. The real cause of the dryness of the conjunctiva is rather the fact that the tears do not adhere to it. Nevertheless it is true that in advanced xerosis the lacrimal secretion diminishes, and may even disappear altogether, for, owing to the marked shrinking of the conjunctiva, the secretory ducts of the lacrimal gland, which empty along the upper retrotarsal fold, become closed, and as a result of this, atrophy of the gland itself ensues. Arlt found in a case of xerosis obliteration of the efferent ducts of the lacrimal gland produced by the great shrinking of the conjunctiva, the gland itself being diminished to one third of its normal volume and transformed into a tissue resembling fat. In that form of xerosis also which occurs in connection with kera-tomalacia there is a striking deficiency in the lacrimal secretion; but here probably we have to do with a nervous disturbance, namely, with an absence of the reflex lacrimal secretion, due to depression of the general nutrition and particularly of the nervous functions. In a case of this sort Cirincione found on autopsy an inflammation of the ciliary and gasserian ganglia.

XV. Extravasation of Serum and Blood beneath the Conjunctiva

508. Œdema of the Conjunctiva.—Œdema and also extravasation of blood, if of any extent, are observed only in the conjunctiva of the eyeball and in the retrotarsal fold, as these, on account of the laxity with which they are fixed to the subjacent parts, can be readily lifted up over quite large areas by fluid. The same thing does not occur in the conjunctiva tarsi, because it is too intimately adherent to the underlying tissue.

Since œdema, as a rule, is of importance merely as a symptom of some other affection, treatment will have to be directed to the latter. Should it be desirable to take any special measures against the œdema itself, the most suitable means for this purpose would be a pressure bandage, or, in the case of a tense œdema, scarification of the conjunctiva.

In the conjunctiva of the eyeball we meet with both inflammatory œdema (œdema calidum), occurring in inflammations of the eyeball or the organs near it, and with non-inflammatory œdema (œdema frigidum), resulting from simple transudation of fluid.

Inflammatory œdema is associated with the most various inflammatory affections, such as inflammations of the lids (erysipelas, hordeolum), of the margin of the orbit (periostitis), of the lacrimal sac (dacryocystitis), of the conjunctiva (particularly gonor-rhœal conjunctivitis), of the eyeball itself (purulent keratitis, irido-cyclitis, and panoph-thalmitis), and, furthermore, of structures posterior to the eyeball (tenonitis, phlegmon of the orbit, cerebrospinal meningitis). A marked inflammatory œdema is produced by dionin (§ 133). Inflammatory œdema occurs especially in old people, whose conjunctiva is particularly extensible and so loosely attached that we observe œdema in this situation sometimes with trifling conjunctival catarrhs. We commonly find the œdema most

pronounced in the region of the palpebral fissure, because here the counter-pressure exerted by the lids is wanting; and not infrequently an œdematous fold of conjunctiva is found in the palpebral fissure, pinched off, so to speak, from the rest by the lids.

Non-inflammatory œdema is caused by hydræmia or by stasis. In the former case it occurs as a symptom of albuminuria, and sometimes then recurs repeatedly but each time disappears again rapidly (œdema fugax). A *filtration œdema* of the conjunctiva of the eyeball is observed after operations or injuries which have produced a perforation in the most anterior section of the sclera, and arises from the fact that the aqueous from the anterior chamber oozes between the incompletely closed edges of the wound and lodges under the conjunctiva. Accordingly, when cicatrization occurs, the œdema commonly disappears. But if a minute opening remains in the sclera, the œdema, too, persists—a condition which is designated as cystoid cicatrization (see Fig. 279). This occurs after cataract operations in which incarceration of the iris prevents closure of the wound, and after trephining. The œdema appears as a bleb-like, sharply defined protrusion of the conjunctiva which looks pale itself but is surrounded by a slightly reddened areola. The cavity of the bleb is divided into loculi by fine cicatricial septa. The blebs can readily rupture and may also become infected (see § 609).

Not infrequently we see in the scleral conjunctiva small bright vesicles, arranged in a row like a string of pearls, or even joined so as to form longer sausage-like swellings. Here we have to do with dilated lymph-vessels that are filled with a clear liquid (*lymph-angiectasiæ*). These occur both in inflammation of the conjunctiva and also when the conjunctiva is perfectly healthy.

509. Subconjunctival Hæmorrhage.—The exudation of blood beneath the conjunctiva of the eyeball is known as *ecchymoma subconjunctivale.* We then see a spot of varying extent and of a vivid-red or dark-red color; sometimes the whole conjunctiva scleræ is suffused with blood. Ecchymosis is easily distinguished from an inflammatory redness of the conjunctiva by its uniformly red coloration, in which no network of vessels can be recognized, and also by its sharp limitation from the unsuffused portions of the conjunctiva, which are ordinarily quite normal and uncolored.

Ecchymoses of the conjunctiva develop after injuries and operations upon the conjunctiva (particularly after squint operations), and, furthermore, in violent inflammations, especially in catarrhal ophthalmia. Spontaneous suffusions in an otherwise healthy conjunctiva are frequent in old people, whose blood-vessels have brittle walls, the rupture of the vessels being often occasioned by great bodily exertion, or by coughing, sneezing, vomiting, straining, etc. In children also spontaneous ecchymoses of the conjunctiva are observed, chiefly after whooping cough. A special symptomatic significance belongs to those ecchymoses which develop, to all appearance spontaneously, after an injury to the skull. Here we have to do with cases in which a fracture of the base of the skull has occurred, and the blood, as it escapes, gradually oozes forward through the orbit until it lodges beneath the conjunctiva (see § 444).

Subconjunctival ecchymoses become absorbed in from a few days to a few weeks without producing any ulterior serious consequences, and in themselves require no treatment whatever. We generally prescribe compresses of lead water or hot water, more for the purpose of appeasing the patient than of obtaining a more speedy resorption.

Ecchymoses of the conjunctiva, free from danger as they are, terrify the patient by their conspicuous look, especially if, as is frequently the case, they spread still further

in the next few days after they first develop. In these cases the pinguecula stands out with peculiar distinctness, as a light, white or yellowish spot upon the red substratum. From the transparent cornea the ecchymosis is delimited by a narrow gray border. This is the inner margin of the limbus, which is too closely adherent to the cornea to be lifted up by the blood. In eyes with blue iris, the latter often has its color apparently changed to green in the spot corresponding to the ecchymosis. This is caused by the extension of the blood in a very thin layer (in which it appears green) between the lamellæ of the cornea, so as to make the iris which lies behind it seem green.

During the absorption of subconjunctival ecchymoses, the lymph vessels may become distended with blood (Elschnig). There is then produced an appearance like that of lymphangiectases except that dark red blood replaces the limpid contents. Occasionally, the same thing occurs without a subconjunctival hæmorrhage.

510. Emphysema of the Conjunctiva.—The entrance of air beneath the conjunctiva is sometimes observed simultaneously with the escape of air beneath the skin of the lids or into the orbital tissue (see §§ 443 and 876).

Hæmorrhage from the Conjunctiva.—Hæmorrhage from the conjunctiva ("bloody weeping") is due usually to traumatism (incised or contused wounds, rough or too frequent applications to the conjunctiva—especially too vigorous application of silver nitrate in ophthalmia neonatorum—,etc.). In other cases, ulceration or abrasion of polypi or other vascular tumors is the cause (see § 515). Very rarely hæmorrhages occur spontaneously in typhoid fever, in connection with menstruation, and conjointly with retinal hæmorrhages (Wallis and others). The bleeding may be profuse enough, at least in infants, to endanger life.

Pigmentation of the Conjunctiva.—Pigment is often present in the normal conjunctiva and most of all at the limbus, where, especially in dark-skinned persons, we not infrequently observe discrete dark brown spots or a more uniform brown coloration. Another instance of what may be called normal pigmentation is the pinguecula (§ 9). Pathological types of pigmentation are afforded by the pigment moles and melanosarcomata (see §§ 513 and 516), and the staining produced in argyrosis (page 438) and by various chemicals (§ 500). Circumscribed, often extensive, sharply defined black spots (*melanosis of the conjunctiva*) occasionally occur in chromidrosis, or as the result of an unabsorbed hæmorrhage, or from other causes (Blanchard, Wirtz, Randolph). These are not in themselves malignant, yet in most cases melanosarcomata develop from them later.

XVI. Tumors of the Conjunctiva.

511. Benign Tumors.—The most important benign tumors of the conjunctiva are dermoids, lipomata, nævi, and cysts.

Dermoid is a flat growth of solid consistence which, so to speak, straddles the margin of the cornea, being situated partly in the conjunctiva and partly in the cornea, with which latter it is immovably connected. It most frequently occurs on the temporal side of the cornea (Fig. 222). Its color is white or reddish, its surface epidermoid and often dry. It is sometimes covered with fine down, or even with longer hair. The growth has the composition of the external skin; it consists of a stroma of connective tissue, covered with epidermis, and contains hair follicles and various glands. It is, so to speak, an island of skin upon the surface of the eyeball.

Dermoid tumors are always congenital, and are frequently found along with other congenital anomalies, like harelip or wartlike appendages of skin in front of the ears. Sometimes they grow still larger after birth.

The chief harm that dermoids do is to produce a considerable disfigurement. If they are large, and especially if they are covered with hair, they cause mechanically an irritation of the eye, and also interfere with

vision, in so far as they encroach upon the pupillary area of the cornea. They are removed by a simple process of ablation, which consists in detaching the tumor as accurately as possible from the subjacent cornea and sclera. The resulting wound in the conjunctiva should, as far as possible, be covered by drawing the adjacent conjunctiva over it (see § 914). That portion of the cornea upon which the tumor was formerly situated remains clouded permanently. If remnants of the tumor have been left, the latter may in part form again.

Dermoid tumors should not be confounded with dermoid cysts (§ 879), of which, in fact, they are the direct opposite (Salzmann). In dermoid cysts the cutis is outside, the epidermis inside; in the dermoid tumor the layers have their normal arrangement, the epidermis being outside and the cutis with the subcutaneous fat lying within. The cutis resembles that of ordinary skin and, like the latter, contains, besides hair follicles, sebaceous glands and often also sweat glands. On the other hand, acinous glands also occur such as are present in mucous membranes (like Krause's glands of the conjunctiva). In rare cases dermoids contain hyaline cartilage or bone (the latter cases being described as *osteomata*), so that complicated structures develop which may be called teratomata.

Dermoids are supposed by Van Duyse to be remnants of amniotic bands, but since the latter do not have the structure of skin, it is more likely that they represent the remains of adhesions between the lid and eyeball, due to enforced contact maintained by the pressure of the amnion or of amniotic bands.

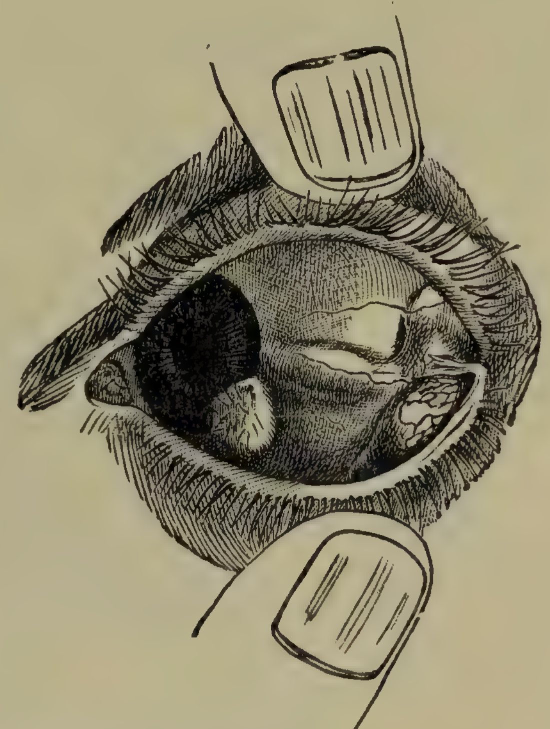

FIG. 222.—DERMOID TUMOR OF THE CORNEA AND SUBCONJUNCTIVAL LIPOMA IN A THIRTEEN-YEAR-OLD GIRL.

The hairy dermoid tumor is situated on the outer and lower margin of the cornea, lying to a small extent in the cornea, and more largely in the sclera. The eye has to be turned strongly inward in order to bring the lipoma well into view. The lipoma has a form differing from the ordinary in that it consists of two lobules, one, more bulging, lying beneath the lower lid, the other, flatter and lying under the upper lid. In addition it sends out a process which runs in the horizontal meridian of the eyeball as far as the outer margin of the cornea. It is everywhere covered by compact conjunctiva which resembles skin, but which allows the yellow color of the underlying fat to show through.

512. Lipoma.—Lipoma subconjunctivale forms a yellowish tumor, situated on the eyeball, far to the temporal side. It is of triangular form the sharply defined base of the triangle looking toward the cornea, while the two sides of the triangle, which are directed outward pass gradually into the orbital fat.

If the tumor is small it ordinarily remains concealed beneath the outer canthus, and can be brought into view only by turning the eye strongly inward (Fig. 222). Larger lipomata are visible in the palpebral fissure even when the gaze is directed straight forward, and hence cause disfigurement; but they do no other harm. Microscopical examination shows that the tumor consists of fat lobules. The conjunctiva that coats it is thickened and of a character resembling skin, and in this regard a lipoma is akin to the dermoids (lipo-dermoid). Like the latter it is congenital, but sometimes grows to a considerable size at the time of puberty. If it is desired to remove the tumor on account of the disfigurement it produces, we split the conjunctiva and remove as much of the mass of fat as is visible in the palpebral fissure, sparing, however, all normal conjunctiva. A radical removal of all the adipose tissue is unnecessary.

513. Nævus.—The pigmented mole (nævus pigmentosus) occurs especially in the bulbar conjunctiva close to the cornea, in the spot where the pinguecula develops. It

is a small, smooth, flat tumor, movable with the conjunctiva, and varying in color fron reddish to dark brown (Fig. 223). Their structure is altogether like that of nævi of th skin. In the delicate connective tissue of the conjunctiva which contains abundan colorless cells and a few chromatophores, lie nests of more or less pigmented nævus cell (Fig. 224, *z*). The origin of these cells is contested, since some derive them from the endo thelial cells of the lymph vessels (Recklinghausen), others from the surface epitheliun (Unna) or from the chromatophores (Ribbert). (Unna's view seems more probable. Apart from this, there is also a proliferation of the epithelium, the latter growing dowr into the deeper parts in the form of gland-like invaginations which are sometimes solid sometimes hollow (Fig. 224, *p* and *h*).

Nævi are congenital growths. They are benign, but sometimes malignant tumors develop from them (see § 516). Hence it is proper to remove them early, by excising the portion of conjunctiva involved.

514. Cysts.—Cysts in the conjunctiva are commonly represented by small vesicles

Fig. 223.

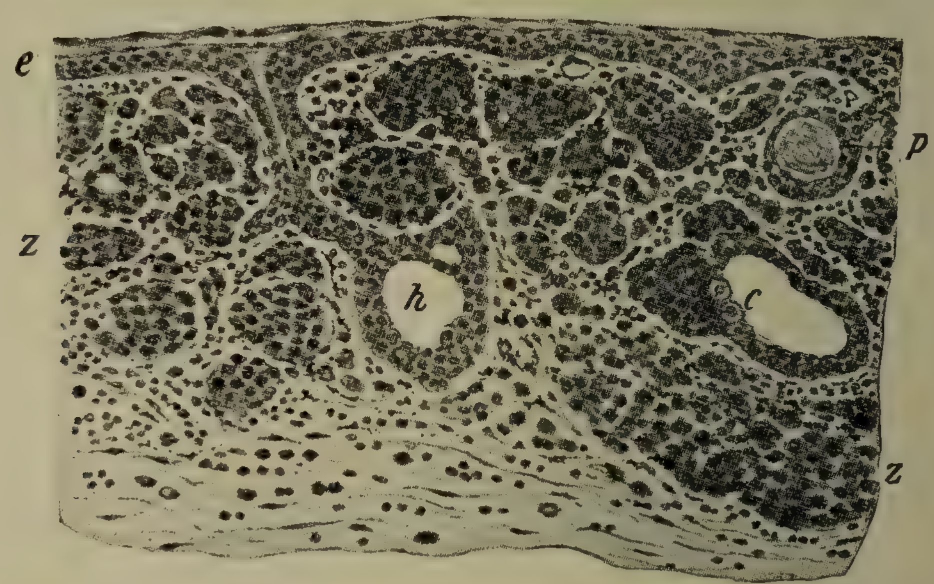

Fig. 224.

Fig. 223.—Nævus Pigmentosus Limbi. Natural Size

Fig. 224.—A Portion of the Same Nævus. Magnified 113×1. The tumor is formed of cells which, being packed closely together, are arranged in spherical agglomerations, *z*. These are separated by slender bands of connective tissue which carry pigment cells. The epithelium, *e*, of the conjunctiva in many places penetrates into the depth of the growth between the agglomerations of cells. There are thus formed either solid plugs composed of cells, many of these containing pearls of laminated epithelium, *p*, or hollow gland-like invaginations, whose lumen in its deeper portion is ordinarily dilated to form rather large cavities, *h*. In many of these spaces, e.g., at *c*, there are found within the epithelial cells coccida, which settle by preference in protected portions of the conjunctiva and act as cellular parasites.

filled with a limpid liquid. Most of these vesicles, and particularly those situated upon the conjunctiva of the eyeball, originate from dilated lymph vessels (see § 508). In the retrotarsal fold oblong, translucent cysts occur which take their starting point from Krause's glands or from new-formed glands in the conjunctiva. These cysts are covered with smooth epithelium and are unconnected with the eyeball, which itself is normal. In this regard they differ from the scleral cysts (§ 635) and the cysts of the lower lid which are associated with microphthalmus (§ 769). In both of these conditions the eyeball shows great pathological abnormalities. Cysts may also be produced in the conjunctiva by traumatism.

Larger cysts situated beneath the conjunctiva are formed by the cysticercus cellulosæ. The latter is observed for the most part in children or in adolescents. In such a case the conjunctiva at one particular spot is found traversed by dilated vessels, and forms a nodular protuberance (Fig. 225). Underneath may be felt the cyst, which, as a rule, can be readily pushed about upon the subjacent parts; in individual cases, however it is intimately adherent to the subjacent sclera or to one of the ocular muscles. If the cyst has very thin walls, the head of the worm can be recognized as a whitish spot in its interior. It is easy to remove the cysticercus by splitting up the conjunctiva and dissecting out the cyst, which consists of the cysticercus vesicle inclosed in a capsule of connective tissue that has been formed about the animal.

515. Polypi and other Tumors.—Under the name of *polypi* of the conjunctiva we understand soft, rarely rather hard, outgrowths which are attached to the conjunctiva by a pedicle, and whose smooth surface is covered with mucous membrane. These originate most frequently in the retrotarsal fold or in the conjunctiva of the lids. As a rule, they are so small as not to be disclosed until the lids are everted; sometimes, however, they are large enough to protrude between the lids. Larger polypi are frequently ulcerated as a result of the mechanical injuries to which they are necessarily subjected. In their nature polypi are small fibromata, which push out the conjunctiva in a sac-like protrusion before them. Treatment consists in ablation and a subsequent cauterization of the base with the silver-nitrate stick.

Papillomata of the conjunctiva are frequently confounded with polypi, but are distinguished from them by their surface being not smooth, but papillary—that is, nodulated, like a raspberry or cauliflower. They are either provided with a pedicle or are sessile, having a broad base and extending in a flat layer over quite a large section of the conjunctiva. They start most frequently from the region of the caruncle, but may also take their origin from other portions of the conjunctiva, and sometimes several papillomata at once are present in different portions of the conjunctiva. They must be removed very thoroughly, since they are extremely apt to recur and moreover, show a tendency to develop into carcinomata.

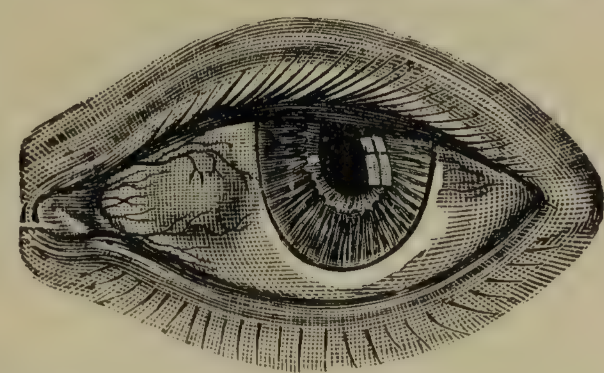

FIG. 225.—CYSTICERCUS SUBCONJUNCTIVALIS. Magnified $1\frac{1}{2}\times 1$.

In a girl ten years of age a small blood-red spot was observed on the nasal side of the cornea two months before she came under treatment. This spot became constantly more elevated and at the same time paler until it assumed its present form, that of a freely movable cyst, lying beneath the conjunctiva.

A third form of tumor, having an external resemblance to polypi are the *granulation tumors*. These, like polypi, form small, mushroom-like, pedicled tumors. Unlike polypi, however, they are not covered by conjunctiva, but consist of naked granulation tissue. They develop in places where a loss of substance exists in the conjunctiva, whether as a result of ulcers (or even large phlyctenules) or as a result of injuries or of operations (most frequently after tenotomies, at the site of the conjunctival wound, and, after enucleation, at the bottom of the conjunctival sac). In the case of chalazia, also, which have broken through the conjunctiva of the lids, we quite often see a granulation mass growing out of the opening. After existing for some time granulation tumors become more and more constricted at their base by the cicatricial contraction of the surrounding conjunctiva, so that they ultimately fall off of themselves if they have not been previously removed.

The three varieties of tumors above mentioned often contain an abundance of dilated vessels; in fact, some cases of very vascular polypi have been described as pediculated angiomata of the conjunctiva. It is hence easy to conceive how these tumors can readily give rise to repeated hæmorrhages, especially if they are ulcerated in spots and are mechanically injured, as, for example, by the rubbing of the lids upon them (see § 510).

Resembling the tumors, just described, are the firm, sessile or pedunculated masses produced by *hyperplastic subconjunctivitis* a condition of rapidly developing hyperplasia of the subconjunctival tissue of the lid and eyeball without involvement of the deeper parts (Berry, Morton). The apparent tumors, which cause no pain, may be treated by removal or incision.

Hœmangiomata of the conjunctiva are, as a rule, those which were originally situated in the lids and have gradually passed over upon the conjunctiva. Primary angiomata are of rare occurrence in the conjunctiva, and are generally found at the inner angle of the eye. They are for the most part congenital, and increase in size after birth. For treatment, see § 445.

Other benign tumors occurring, though very rarely, in the conjunctiva are fibromata, myxomata, and lymphangiomata. Furthermore, there are small connective-tissue

growths which consist of plasma cells and are hence called *plasmomata*. It is not yet determined whether these are to be classed among chronic inflammatory growths or among neoplasms in the narrower sense of the word.

516. Malignant Tumors.—The malignant growths in the conjunctiva are often secondary, being derived from the lids, in which such growths occur much more frequently as a primary affection than in the conjunctiva. Primary conjunctival tumors of a malignant type, comprising carcinoma and sarcoma, may develop at various points, but usually at the limbus.

Carcinomata (*epitheliomata*) often originate from papillomata (§ 515). From these they are distinguished by their smoother surface and the irregular fashion in which the vessels branch. Often they show whitish flakes of cornified epithelium. In other cases as shown in Fig. 226, they resemble a

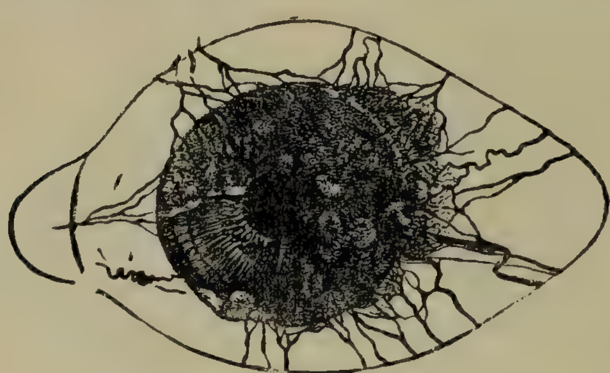

Fig. 226

Fig. 227.

Fig. 226.—Epithelioma of the Limbus and Cornea. The patient, a man fifty-six years' of age, had noticed, as much as thirteen years before, that a pellicle was beginning to grow on the outer border of the cornea in the left eye. With the exception of its lower inner quadrant the cornea is entirely covered by a gray deposit, the surface of whch is coarsely nodular and is traversed by numerous vessels which arise from the limbus. In the deposit can be seen with the aid of a magnifying glass scattered bright dots (epithelial pearls). Owing to these the deposit in spots acquires a greasy aspect. The new growth passes from the cornea on into the limbus and at the temporal side a little way into the conjunctiva of the eyeball, which at this spot has also taken on a nodular and in places gelatinous character.

Fig. 227.—Section through the Anterior Segment of the Same Eye. The section starts from the upper and outer part of the eye and runs downward and inward.

dense pannus, from which they are distinguished by the way, suggestive of a neoplasm, in which they invade the conjunctiva, by their more nodular surface, and by the greater cloudiness of the epithelium, which is veiled or in places entirely covered by the excessive development of vessels. The marginal portions of the epithelioma often are quite free, simply lying on the subjacent tissues, but at the starting point of the tumor, the epithelial growth often penetrates deeply, especially at the spots where the blood-vessels perforate the sclera. In this way the epithelioma may get into the interior of the eye, before any defect in the wall of the eyeball has been produced. Later the tumor ulcerates and then spreads along the surface of the exposed uveal tract.

Sarcomata are generally pigmented (melano-sarcomata) and are often derived from nævi (§ 513). Frequently they are attached to the substratum by only a slender base. They hence form brown or black, very prominent mushroom-like growths, which often overlie a great part of the cornea although, if we lift them up, we find the cornea beneath for the most part normal (Figs. 228 and 229). They show no great tendency to penetrate into the depth of the eye, but have a great tendency to recur.

Both epitheliomata and sarcomata develop in advanced life, and, if

they are not removed, spread steadily; sarcomata, especially, grow until at length they form gigantic tumors. At last the patients succumbs from exhaustion or from metastases which form in the internal organs. The tumors must therefore be removed as early and as radically as possible. This is true even of the antecedent, apparently benign growths (nævi and papillomata). As long as the tumors are still quite small and superficial, radical removal can be performed with preservation of the eyeball (see § 914). But these tumors, especially sarcomata, tend to recur even after very thorough removal, and Verhoeff and Loring believe that, in the case of sarcomata at least, enucleation should be performed at once. In cases in

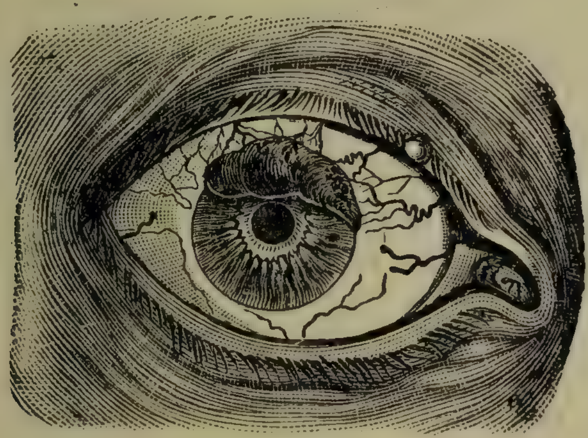

Fig. 228.

Fig. 229.

Fig. 228.—Melano-sarcoma of the Limbus Encroaching upon the Cornea. The dark brown, sausage-shaped tumor starts from the limbus and overlies the upper third of the cornea. On its surface can be seen dilated veins. In the sclera the anterior ciliary veins are conspicuously visible, emerging at some distance from the margin of the cornea. The border of the upper lid presents near the inner angle of the lids, a small limpid cyst. This is situated right in the row of lashes and is derived from a gland of Moll.

Fig. 229.—A Perpendicular Section of the Same Eye. The tumor simply lies upon the cornea without penetrating into it.

which because of the area occupied by the growths or because of other reasons, excision is considered inadvisable. Radium, applied in sufficiently large dosage, offers good chance of success, especially in epitheliomata (Treacher Collins, Benedict, Janeway).

If complete removal of the growth is no longer possible in any of these ways, the eye must be enucleated even though it still possesses visual power.

This is particularly the case if the tumor is so extensive that in its removal a large part of the conjunctiva would have to be sacrificed; for then the operation would result in such a considerable formation of cicatricial tissue with consequent displacement and traction on the eyeball that the latter becomes useless and might better have been extirpated in the beginning. So, also, the eyeball must be sacrificed in those cases in which the tumor grows down into the depth of the tissues at any point as is especially apt to happen along the anterior ciliary vessels. Such an occurrence is often not discovered until after the removal of the superficial growth, or may even not be discovered at all. In the latter case a recurrence in the same locality occurs soon after the apparently radical removal.

The *predilection for the limbus* shown by epitheliomata is to be considered as an analogue of the fact, that, in other parts of the body also, epitheliomata occupy by preference those spots where one kind of epithelium passes into another, as for instance, the boundary line between skin and mucous membrane (anus, lips, margins of the lids, etc.). An additional factor giving rise to the development of epitheliomata at the limbus is probably furnished by the anatomical character of the latter. The limbus is the only place in the ocular conjunctiva in which small papillæ are normally present. Between

these papillæ we sometimes find, even in the healthy eye, a proliferation of epitheliu the latter growing in the form of conical processes into the depth of the tissue.

The dissemination of epitheliomata takes place mainly by the lymph rather th the blood channels (Treacher Collins).

Melanotic *sarcomata* develop in those localities in which pigment is already n mally present. They hence occur upon the conjunctiva of the lids, and above all up the limbus, two divisions of the conjunctiva which even under physiological conditio contain pigment. If we accept Unna's view that nævi are of epithelial origin (§ 51 we must consider the pigmented tumors that are derived from them as carcinoma (melano-carcinomata) rather than as sarcomata (Salzmann).

Cylindroma also occurs, although very rarely in the conjunctiva.

The *plica semilunaris* and the *caruncle* which is situated upon it participate in t inflammations of the conjunctiva, so that it is unnecessary to treat of the disease of t parts separately. Sometimes the little hairs which the caruncle always has upon it a so long as to irritate the eye; in that case the hairs must be epilated. The new form tions of the caruncle are designated by the old name encanthis[16]. Benign new form tions, such as simple polypoid or papillary outgrowths of the caruncle, are call encanthis benigna; malignant new formations are called encanthis maligna.

[16] From ἐν, in, and κανθός, angle of the eye.

CHAPTER XXI

DISEASES OF THE LACRIMAL ORGANS

I. Dacryocystitis Chronica

517. Symptoms.—Chronic dacryocystitis is an inflammation of the lacrimal sac, due to impeded outflow and consequent stagnation of the tears. In well-marked cases the skin in the region of the lacrimal sac protrudes so as to appear fuller than on the other side. This swelling—tumor lacrimalis—is caused by the sac's being distended by an accumulation of its contents. In mild cases, at the outset and sometimes for a long time, there may be epiphora and more or less constant obstruction, without evident distention of the sac. When pressure is made on the tumor the contents are discharged externally through the puncta (more rarely through the nose) and appear, sometimes as a purulent, sometimes as a mucous or even limpid liquid. Associated with these symptoms, there is usually a stricture of the nasal duct. As evidence of this the escape of the tears to the nose is impeded or altogether prevented, so that the tears collect in the lacrimal and even in the conjunctival sac. As soon as they do so in any amount, they overflow upon the cheek (epiphora)[1].

The objective proof of the presence of a stricture is produced if with an Anel's syringe (Fig. 398, *h*) we inject water through the lower canaliculus, and make the patient bend his head down so that the water which gets into the nose shall run forward. If stenosis of the nasal duct is present, the water does not flow freely into the nose, but only drop by drop or not at all, and regurgitates from the lacrimal sac through the upper canaliculus. If in persons with no obstruction of the lacrimal passages we instill a mixture of adrenaline and cocaine into the eye so as to shrink up the mucous membrane of the tear passages and then instil a 30-per-cent solution of argyrol, the latter will gradually make its way down into the nose and, if the patient blows his nose, will stain the handkerchief. In a patient with lacrimal obstruction the argyrol goes down with difficulty or not at all. If in this case we inject the adrenaline-cocaine solution into the canaliculus with the syringe the argyrol that we have instilled will then usually go through to the nose, provided the obstruction is due simply to turgidity of the membrane, and not to a bony or fibrous stenosis. In the latter case in order to determine the site and extent of the stenosis we first make the passages insensitive by injecting a few drops of a cocaine solution and then explore the duct with a Bowman's sound, in doing which we come upon a spot where the duct is contracted so that only the finest sound passes or even altogether obliterated so that no sound passes at all.

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[1] From ἐπιφέρεσθαι to rush upon. We also say *illacrimatio* or *stillicidium* (from *stilla*, drop and *cadere* to fall).

518. Etiology.—The immediate cause of a chronic dacryocystitis is stricture of the nasal duct and consequent retention of the tears in the lacrimal sac. How this acts to cause decomposition of the secretion and inflammation of the sac (dacryocystitis) has already been described (§ 95).

The constriction of the nasal duct develops, as a rule, in consequence of affections in the nasal cavity. Such affections are:

1. *Inflammation of the nasal mucous membrane.* Under this head belongs coryza in its various forms, whether acute or chronic, and whether a simple catarrhal trouble, or one of scrofulous or syphilitic origin. The inflammation of the mucous membrane of the nose extends by continuity to the mucous membrane of the nasal duct, being particularly due to the engorgement of the numerous veins lying beneath the latter, an engorgement which of itself suffices to close the lumen of the duct. The inflammation of the mucous membrane then leads to cicatricial contraction of the latter and consequently to strictures in the nasal duct, so that the impermeability of the duct persists even after the inflammatory swelling subsides. In the ordinary form of ozæna (rhinitis atrophicans) there is no swelling even at the beginning, but a cicatricial contraction of the nasal mucous membrane, which may be continued from the inferior orifice of the nasal duct into the latter and thus lead to its constriction. 2. *Ulcers,* such as are often associated with the scrofulous and syphilitic inflammations of the nasal mucous membrane, and also lupous ulcers. As the ulcers heal, cicatricial constriction or even obliteration of the nasal duct takes place. This is the more to be apprehended if the subjacent bones are also implicated. 3. *Tumors.* These may block the lower orifice of the nasal duct, and thus obstruct the flow of tears. The most frequent tumors of this kind are polypi.

Thompson believes that dacryocystitis is caused not by extension of disease from the nose, but by the lodgement of foreign bodies passing down through the duct.

Chronic dacryocystitis affects the female more often than the male sex, perhaps on account of the more frequent use which the former makes of the lacrimal apparatus. Persons having the bridge of the nose flattened (flat noses and "saddle-noses," particularly when due to hereditary syphilis) also are predisposed to this affection.

Sometimes a chronic dacryocystitis is observed even in *new-born children* and this may actually be associated with perforation and the formation of fistulæ. The cause of the disease is a belated perforation of the thin diaphragm of mucous membrane which in the fetus closes the lower orifice of the nasal duct. The result is decomposition of the gelatinous contents of the nasal passages and hence inflammation of the latter (Rochon-Duvigneaud). Usually repeated expression of the lacrimal sac suffices for a cure; if not we must make the duct pervious by the use of sounds.

Chronic dacryocystitis may be due to *tuberculosis,* transmitted from the conjunctiva (§ 495) or the nose. The sac then feels very much thickened, and on opening it we find its inner surface lined with discolored granulations. Dacryocystitis is also common in *trachoma,* but it is hard to tell whether the inflammation here is trachomatous since even in ordinary dacryocystitis the sac contains follicles like those of trachoma. Trachoma of the sac is said to be frequent in the Near East where trachoma is endemic. Sacs thus diseased are very friable and rupture easily, so that lavage is dangerous (Butler).

Mycotic inflammation of the sac, including *blastomycosis* (cf. § 477) and *actinomycosis,* has also been observed (Bromlie, Nagel).

519. Course.—Chronic dacryocystitis is an eminently chronic disease, whose course is counted by years. A spontaneous cure may indeed occur, if,

as the swelling of the mucous membrane of the nasal duct abates, the lumen of the duct becomes free again, and at the same time the catarrhal inflammation of the sac subsides. This, however, occurs but rarely, because strictures of the duct have formed in the meantime. Hence the rule is that without artificial aid no cure takes place, but the following course of events ensues: The secretion, at first purulent, becomes after a time mucous and viscid; at length, owing to atrophy of the mucous membrane, secretion ceases altogether. Then the distended sac contains simply a clear liquid—namely, the tears which have accumulated in it. But still the epiphora keeps on, because the constriction of the nasal duct prevents the conduction of the tears into the nose.

As a result of the constant distention of the lacrimal sac by fluid, its walls at length lose their elasticity. When this condition, called *atonia sacci lacrimalis*, has set in, the tears are no longer carried down into the nose, even if the nasal duct again becomes perfectly pervious. The sac may keep distending more and more until it forms a fluctuating tumor of the size of a walnut and over. This either projects far forward, or it often extends deep into the orbit, so that the eyeball is displaced by it (exophthalmus). It is filled with a clear fluid; hence the name *hydrops sacci lacrimalis*.

Chronic dacryocystitis causes trouble chiefly through the epiphora, which compels the patient to dry his eye frequently. The epiphora increases in cold weather, and in wind, smoke, etc. If it lasts a long time it leads to chronic catarrh of the conjunctiva and to blepharitis ulcerosa. If these two conditions are found in one eye alone, they must always excite the suspicion of there being a lesion of the lacrimal sac. As the disease goes on, the moistening of the lower lid by the tears leads to eczema of the lid, contraction of the skin covering it, and ultimately to ectropion. By these conditions, again, the epiphora itself is increased. In the presence of chronic dacryocystitis ulcers of the cornea may develop from erosions of the latter, and operation wounds readily become infected.

520. Treatment.—If nasal disease is present, we must use the proper means for its relief. To prevent accumulation of secretion and the distention of the sac, the patient is told to empty the latter very frequently by pressing the finger on the inner canthus. The sac can also be cleaned by *syringing*, (see § 903), and many obstructions of the milder type, especially in the earlier stages, can be cured in this way—often by a few treatments. Ethylhydrocupreine (optochin) dropped into the eye may help in subacute cases (Posey).

In simple eversion of the punctum, without ectropion proper the epiphora can be relieved by *slitting the canaliculus* (§ 904). The tube is thus converted into an open groove, which looks backward and hence dips into the lacus lacrimalis and takes up the tears.

The usual form of treatment consists in gradual dilatation of the duct with sounds (see § 905). The duration and the success of this treatment depend upon the nature of the contraction. The most favorable cases are those in which the contraction is caused by simple inflammatory swelling of the mucous membrane; those in which cicatricial strictures are present

are less favorable, and the least favorable are those in which the nasal duct is completely obliterated at some spot. Cases of the latter kind do not, for the most part, admit of a permanent cure. Even in the most favorable cases the duration of the treatment amounts to from four to six weeks, and it usually requires several months. If strictures due to cicatrices are present recurrences may set in, owing to renewed contraction of the cicatricial tissue; and, in fact, this unfortunately occurs so often that permanent cures form the exception.

Those cases which cannot be cured by treatment with sounds require, if they give rise to considerable annoyance, *extirpation of the lacrimal sac* or other radical measures (see §§ 909, 910).

Extirpation of the lacrimal sac is suitable whenever treatment by sounds is likely to be without result. This is the case where there are very extensive cicatricial contractions or complete obliteration of the nasal duct. This is still more the case if there are at the same time demonstrable changes in the bone, shown either by our coming upon bared and roughened bone in the act of sounding, or by there being an externally visible implication of the bone manifested by a sinking in of the nose (in consequence of syphilis). Furthermore, atony and dropsy of the sac, are indications for extirpation, since in these cases a normal escape of tears could not take place even after the nasal duct has been made pervious. Moreover, removal of the tear sac is very frequently done when treatment by sounds would be feasible, but the patient's external circumstances make this very protracted method of treatment impossible. This is particularly the case in patients of the working class. Indeed, many, including the translator, regard it as preferable in general to the use of sounds.

While the treatment by sounds in favorable cases restores the normal conduction of the tears, the possibility of this restoration is forever prevented by extirpation of the lacrimal sac. Hence a condition of epiphora always remains, which, however, reaches a troublesome degree only when, in consequence of irritation of the conjunctiva, there is an excessive secretion of tears, and this can be relieved by extirpation of the inferior lacrimal gland (see § 911). This seems rarely necessary. In order to obviate the possibility of this continuous epiphora, other operations, e.g., Toti's and West's (see § 910) have been devised, by which permanent drainage is maintained. In any case as an offset to the epiphora the patients are relieved of the presence of a suppurating cavity which continually exposes them to the danger of getting an ulcus serpens of the cornea, and also usually gives rise from time to time to acute phlegmons (dacryocystitis acuta).

There are cases in which the dacryocystitis is of such mild type that we do not feel justified in removing the sac, but in which the dacryocystitis may be a source of temporary danger, e.g., in the case of operations on the eyeball, in which it might give rise to infection. In such cases we may perform *incision of the sac* or *cauterization* or *ligature of the puncta* (see §§ 908 and 907).

II. Dacryocystitis Acuta (or Phlegmonosa)

521. Symptoms.—In an individual who suffers from chronic dacryocystitis, a violent inflammation may suddenly develop in the region of the tear sac. The skin in its vicinity is then reddened and greatly swollen; the swelling also extends to the lids, and even to the conjunctiva, in which there is chemosis. The inflammation is accompanied by fever and violent pain,

so that the patient is deprived of sleep for several nights. After some days the skin at the apex of the swelling takes on a yellowish discoloration, and finally is perforated, when quite a large quantity of pus is discharged. Upon this the pain abates and soon ceases altogether, and the swelling rapidly goes down. Later, there is discharged from the perforation a fluid which at first is purulent, afterward mucous, and at length perfectly clear like water. Ultimately nothing but the tears which are forced into the sac run out through the perforation, which is hence called a *lacrimal fistula.*

As long as the fistula remains open the patient is safe from any new attack of inflammation. But if the fistula closes and tears reaccumulate in the sac, recurrence of the acute dacryocystitis may ensue.

Acute dacryocystitis consists in a purulent inflammation of the connective tissue surrounding the lacrimal sac. This inflammation leads to purulent disintegration of the submucous tissue with the formation of an abscess which ruptures externally. Acute dacryocystitis is accordingly a phlegmon. Chronic dacryocystitis, on the contrary, is an inflammation of the mucous mumbrane itself, in which the purulent secretion of the latter is deposited upon the surface only. The connection between the two diseases consists in the fact that chronic precedes acute dacryocystitis and gives rise to it, for the inflamed sac is filled with decomposed secretion, and it only requires the presence of a small defect in the epithelial covering of the mucous membrane of the sac to enable the micro-organisms of the secretion to penetrate into the submucous tissue, where they excite suppuration and cause dacryocystitis.

In very rare cases acute dacryocystitis arises not from a chronic inflammation of the sac, but from a caries of the lacrimal bone. The diagnosis of dacryocystitis is easy to make from the situation of the swelling, which corresponds to the region of the sac. A characteristic feature is that the swelling extends up but little above the inner palpebral ligament. Swellings which lie mainly above the palpebral ligament are to be regarded as connected not with the lacrimal sac but mostly with the frontal sinus (discharge of an empyema beneath the skin). It is the exception for abscesses of other kinds, e.g., a furuncle in the skin or a periostitic abscess, to occur in this spot. But even in dacryocystitis the spot where the pus breaks through, does not always correspond with the position of the sac. It usually lies beneath, being quite a distance below and to the outside of it. The reason for this is that pus sinks down beneath the skin and at the same time travels outward along the inferior margin of the orbit, because the skin along this is attached to the bone by rather firm connective tissue. That such an opening in the skin which lies far below and to the outer side is a lacrimal fistula can be demonstrated by our being able to push a sound through from the opening into the lacrimal sac or by the fact that a colored liquid injected into the canaliculus flows out through the fistula.

Later on in their course, lacrimal fistulæ contract and sometimes become so minute that they are permeable by nothing but a fine bristle. The external orifice of these *capillary fistulæ* as they are called, is almost invisible. We simply notice a drop of clear lacrimal fluid appearing from time to time on the skin beneath the lacrimal sac; it is only upon careful examination that we make out the capillary opening.

522. Treatment.—If we are dealing with an acute dacryocystitis in its very inception, we may try to prevent the development of an abscess. With this end in view, we sedulously express the fluid from the sac, inject it with antiseptic solutions, and in the intervals apply a pressure bandage, which constringes it. If the inflammation has passed the initial stage, it is idle to endeavor to prevent the formation of an abscess; besides, the methods of syringing, expression, and compression, given above, could not be employed, on account of the swelling and painfulness of the parts. The only thing to

do now is to hasten the formation of the abscess, an object which is best attained by the use of moist hot compresses. As soon as fluctuation makes itself apparent, we incise the lacrimal sac and in addition relieve by sounding or better by immediate extirpation of the sac the chronic dacryocystitis that forms the basis of the condition (see §§ 908 and 909).

523. Diseases of the Canaliculi and Puncta.—In both the *puncta* and *canaliculi* contraction and even obliteration are sometimes observed, the consequence of which is epiphora. The most frequent cause giving rise to these conditions is injury of the mucous membrane of these structures due to sounding. To remove them we must endeavor to enter the canaliculus with a conical sound and thus dilate it, or, if necessary, we must slit it up (see § 904). Occlusion of the canaliculi may also be produced by foreign bodies or by *concretions.* The latter are of a gray or greyish-green color and of friable or hard consistence, and prove to be conglomerated masses consisting of a fungus, the streptothrix Fœrsteri. The sporotrichon may also form concretions in the canaliculi in cases of sporotrichosis (Bedell—cf. § 477). In some cases cystoid dilatation of the canaliculi has been found. This arises from an obliteration of the canaliculus at both extremities and the accumulation of fluid in its cavity, so that the tube is gradually distended till it forms a cyst.

In the puncta there can be frequently made out a change of position of such a character that the lower punctum is turned outward (forward) instead of looking upward (*eversion of the punctum*). This represents the very first stage of an ectropion—a condition which bears within itself the germ for its own development (see § 439).

524. Congenital Anomalies of the Tear Passages.—These are rare and without special significance. Absence of the puncta and canaliculi, supernumerary puncta, abnormal length of the canaliculi have been described. The congenital lacrimal fistula is a capillary fistula, differing from the acquired form in that it is found in childhood and is associated with perfectly patent tear passages. For congenital atresia, see § 518.

525. Diseases of Lacrimal Gland.—It is extremely rare for the lacrimal gland to be the seat of disease. Among such diseases belong: 1. Inflammation (*dacryoadenitis*). This may occur from metastasis, e.g., in parotitis epidemica (mumps of the lacrimal gland). Dacryoadenitis may go on to resolution; in other cases, suppuration of the gland with discharge of the pus externally has been observed, and a fistula is left communicating with the gland. The treatment is symptomatic. A chronic bilateral enlargement of the lacrimal and the parotid and other salivary glands marks *Mikulicz's disease.* This may or may not be associated with the signs of a pseudoleucæmia or of a true, rapidly fatal leucæmia with generalized lymphatic enlargements. According to some the disease is often, according to others rarely due to tuberculosis. It is often associated with pus infection, and other causes have been assigned. The eye symptoms include swelling and drooping of the lids, so that the palpebral fissure is reduced to a narrow slit, and failure of the lacrimal secretion due to sclerosis and destruction of the gland (Lane). It is said that arsenic is of service. 2. *Neoplasms.* These are mostly mixed growths implicating both the epithelial and the connective tissue portions of the gland. If the former element predominates we call the tumors adenomata, cylindromata, or carcinomata; if the latter, they are termed fibromata, myxomata, lymphomata, or sarcomata. Tumors require extirpation of the gland (see § 911). 3. Cystoid dilatation of a duct of the gland—a condition called *dacryops.* This may also require extirpation of the gland. 4. *Atrophy* of the gland in xerophthalmus (§ 507).

526. Epiphora and Absence of Lacrimal Secretion.—Disturbances in the function of the lacrimal apparatus find expression either under the form of epiphora or of absence of the tears. *Epiphora* is an exceedingly frequent symptom, and is either based upon an increase in the secretion or a hindrance to the discharge of the tears. The former occurs physiologically in weeping, and also in the presence of all sorts of irritants affecting the terminal expansion of the trigeminus and its vicinity. Among these are cold wind, air rendered foul by smoke, etc., foreign bodies in the conjunctival sac, inflammations of the eye and its adnexa, affections of the nose, and neuralgia of the first and second branches

of the trigeminus. Furthermore, irritation of the retina by strong light causes increased tear secretion. Interference with the conduction of tears into the nose may have its cause either in some hinderance to the normal closure of the lid or in anomalies of the lacrimal passages. Among affections producing the former condition are paralysis of the orbicularis, shortening or ectropion of the lids, notching of the border of the lid, and even simple eversion of the lower punctum: to the latter belong all the affections of the lacrimal channels which have been treated of in this chapter. In old people it is an extremely common thing for the eyes to water especially out in the open air in cold weather, without there being any discoverable lesion of the conjunctiva or the tear passages. For this senile epiphora instillation of the collyrium adstringens luteum (see § 457, note 3) is the best remedy. If we find such an epiphora without discoverable cause in younger persons, there is probably an excessive irritability of the mucous membrane of the nose which excites increased secretion of tears in a reflex way. In cases of epiphora for which no other cause canbe found, we must, then, examine the nose carefully and treat it according to the indications.

The opposite conditions, namely, *abolition* of the lacrimal secretion, is extremely rare. It is found in xerophthalmus, in consequence of occlusion of the excretory ducts of the lacrimal gland: also in paralysis of the trigeminus, and in facial paralysis when the lesion is situated very high up in the nerve. It is hence supposed that the fibres destined for the innervation of the lacrimal gland start from the brain along with the facial, and run with the nervus petrosus superficialis major to the nasal ganglion of the trigeminus, by the second branch of which they reach the gland through the nervus subcutaneus malæ (Goldzieher). A purely nervous disturbance lies at the bottom of those cases in which persons declare that formerly they wept a good deal, but now are unable to weep even when very much distressed.

CHAPTER XXII.

DISEASES OF THE CORNEA.[1]

I. Inflammation of the Cornea.

527. General Character.—Every inflammation of the cornea (keratitis)[2] begins in an infiltration of its substance with wandering cells (Fig. 230). With these an excessive amount of fluid permeates the tissues. Consequently the affected spot becomes cloudy and swollen, a fact which is often evidenced by an elevation of the surface over it. The overlying epithelium loses its smoothness, and this no matter whether the infiltrate is superficial or deep. Accordingly the clinical signs of this stage (stage of the primary infiltrate) are: cloudiness, loss of lustre, and at times elevation (never depression) of the surface. The color of the clouded portion is a whitish or yellowish, not bluish gray; in markedly purulent processes it is whitish yellow. The subsequent fate of the infiltrate varies. It may go on to resorption or suppuration.

(*a*) In the case of *resorption* the exudation gradually disappears from the cornea. In favorable cases, the affected portions regain their transparency and smoothness, and become perfectly normal. In other cases, especially when the inflammation is more intense or of longer duration, the substance of the cornea although not destroyed in toto, loses some of its elements (stroma or cells) which have to be replaced by a process of repair. The replacing substance, at least in the later years of life, is not corneal tissue, and hence an opacity is produced. But as the destruction of normal tissue and the process of repair keep pace with each other, there is no apparent loss of substance. These forms of keratitis are hence known under the name of *non-suppurative* or better *non-ulcerative* keratitis.

(*b*) The infiltration is *purulent*, i.e., leads to rapid melting away of the corneal tissue en masse through the digestive action of the pus corpuscles of which the infiltrate consists. By the extrusion of the products of this disintegration, there is formed a loss of substance visible to the naked eye; in other words there is formed a corneal *ulcer*. In certain cases such a loss of substance is produced by the primary toxic action of the germs themselves. The forms of inflammation in which there is an evident loss of substance are grouped together under the name of *suppurative* or better *ulcerative* keratitis.

The subdivision of keratitis into ulcerative and non-ulcerative keratitis corresponds most fully to practical requirements. Every ulcerative keratitis, since it is associated with destruction of corneal tissue, leaves behind it a permanent opacity, which in many cases injuriously affects the visual power. On the contrary, so long as purulent dissolution of the cornea has not taken place—that is, in non-ulcerative keratitis—a complete

[1] This chapter is largely but not entirely remodelled on the revision of Salzmann.
[2] From κέρας, horn.

restoration of its transparency, and with it of the normal power of vision, is possible, and, in fact, often does take place. Moreover, the above subdivision also corresponds to the essential characters of the corneal inflammations. For it is not merely a matter of accident, nor something that depends altogether on the degree of inflammation, that determines whether an infiltrate in the cornea goes on to suppuration or to resorption On the contrary, the forms which tend to the production of suppuration ordinarily present from the very beginning characters differing from those borne by forms in which there is no progress toward suppuration, so that these two categories are distinct not only in their consequences but also in their clinical aspect. The suppurative keratitides develop as a rule from the entrance of pyogenic bacteria into the cornea from its surface (ectogenous infection). They have their origin, therefore, generally in external morbific agents and in the majority of cases, only one focus of disease is present, which starts at some point on the surface and from this spreads continuously either along the surface or into the depth of the cornea. The deep forms of non-suppurative keratitis, on the contrary, are caused as a rule by constitutional disease. The morbific agent is carried to the cornea by the circulation of blood or lymph, and since it exerts its action on many points of the corneal parenchyma at once, many distinct foci of disease are produced. Hence the cloudiness in the cornea can be resolved by the loupe into numerous small spots which are the separate infiltrates, which lie in the middle and deep layers of the cornea.

FIG. 230.—INFILTRATION OF THE CORNEA IN PHLYCTENULAR KERATITIS. (After Hayashi.)

The phlyctenule which is two days old consists of a deposition of leucocytes in a series of rows between the superficial lamellæ of the cornea, which in attenuated form are still to be recognized between the cells. Over the right side of the accumulation, Bowman's membrane has a gap, and the spot in the infiltrate lying just beneath this point no longer shows any nuclear staining. Still more widespread is the necrosis in the epithelium which for almost the entire extent of the accumulation is transformed into a mass destitute of recognizable cell nuclei. Thus the disintegration of the phlyctenule is preparing the way for the formation of an ulcer.

528. Vascularization.—In keratitis we very often observe the development of new blood-vessels in the cornea. In one set of cases this vascularization is an important factor in the *process of repair.*

The inflammatory irritation emanating from a diseased spot on the cornea produces first a dilatation of the vessels at the corneo-scleral margin, but if the irritation lasts longer it produces vascularization of the cornea, offshoots growing out from the marginal vessels toward the inflamed area. Thus now the blood, which combats the noxious agency, is brought to the battle-ground directly and can therefore develop its action without loss of time. When a corneal ulcer is spreading steadily, and at length we see the new-formed vessels extend at one spot to the border of the ulcer, we know that here no further progress of the ulcer is to be apprehended, the vessels opposing an effective barrier against the disintegration of the corneal tissue. But often the vessels arrive too late to prevent the destruction of the tissue, the loss of substance having occurred before the vessels reached the diseased spot. Then the function of the vessels consists in bringing the material for repair—i.e., for filling in the loss of substance by means of cicatricial tissue in loco. Since it takes a certain time for vessels to develop, then the more slowly the inflammation runs its course, the more likely are we to see the vessels appearing in the progressive stage of the inflammation and limiting the latter. In cases with rapid course the vessels do not make their appearance until the end of the process and then simply initiate repair.

In the case of a corneal ulcer the vessels designed to effect repair are superficial vessels—and in the case of deep ulcers, deep vessels also—which at the outset grow out in a radial direction from limbus to ulcer, and later

when the latter is healing spread irregularly over its floor; After the loss c substance has been filled in, the vessels gradually disappear, so that a cor neal scar contains fewer of them as it grows older. Nevertheless, th blood-vessels never entirely disappear from the larger scars.

Persistence of vessels is noticed particularly in scars with anterior synechiæ the vascularization in this case being effected by the prolapsed iris.

Repair can be effected without vascularization of the cornea. We often see ulcer of the cornea heal without the formation of a single new vessel in it.

In other cases the formation of new vessels accompanies the *progres of the inflammatory process,* and hence belongs, like the exudation itself, to the clinical picture of the inflammation. The best example of this i afforded by the vascular form of parenchymatous keratitis (§ 574); anothe variety is seen in pannus (§§ 469, 483).

It is very important to determine the situation of the vessels in the cornea, that is, whether *superficial* or *deep*—since by this fact alone we car often diagnosticate what sort of keratitis we are confronted with. The type of superficial vascularization is afforded by pannus, that of deep vascularization by keratitis parenchymatosa.

The signs which enable us to distinguish the two kinds of vasculariza-tion from each other may be contrasted as follows:

Superficial Vessels (Fig. 231)	Deeply Situated Vessels (Figs. 232, 233)
Spring from the net-work of marginal loops of the limbus, and can therefore be followed from the cornea into the limbus, and thence on to the vessels of the conjunctiva.	Spring from the vessels of the sclera close to the margin of the cornea, and hence appear to come to an end suddenly at the limbus, as they disappear behind the latter to enter the sclera.
On account of their superficial position are clearly visible and well-defined, and have a vivid-red color.	Are not distinctly recognizable, or are even unrecognizable, except as a diffuse red coloration, and have a dirty red (grayish-red) hue; the reason for these appearances being that the vessels are veiled by the clouded layers of cornea lying in front of them.
The vessels branch in an arborescent fashion.	The vessels either form fine twigs running broom-fashion—i. e., parallel to each other—or else diverge like the hairs of a paint-brush.
The corneal surface is uneven, because the vessels raise up the epithelium lying directly over them.	The surface of the cornea is lusterless, it is true, but not uneven.

529. Irritative Symptoms of Keratitis; Implication of Neighboring Organs.—Keratitis is almost always accompanied by inflammatory symptoms. These are not by any means necessarily in proportion to the gravity

of the condition. Cases in which they are slight or absent—cases constituting what are called *torpid* or *asthenic* ulcers—may be very dangerous. The inflammatory evidences of keratitis are—

1. *Pain* and *photophobia* with consequent lacrimation and blepharospasm.

2. *Hyperæmia.* Since the inflammation is seated in a non-vascular tissue, the hyperæmia must originate in the vessels of the adjoining parts, i.e., in the marginal network at the limbus and in the vessels of the iris. We hence find (*a*) *ciliary injection* and generally also injection of the anterior conjunctival vessels (see § 448) and (*b*) hyperæmia with irritation of the *iris*, causing discoloration of the latter and miosis. To these may be added injection of the cornea from the formation of new vessels (§ 528).

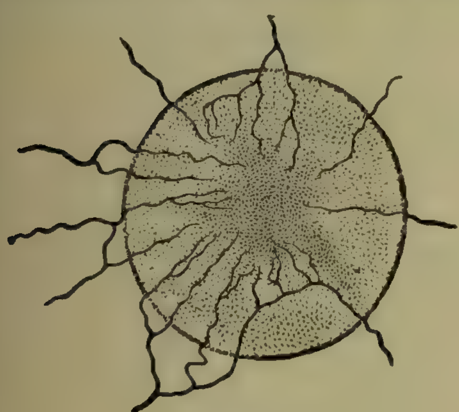

Fig. 231

Fig. 232.

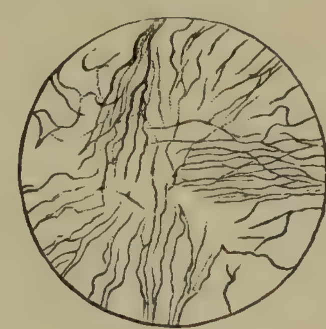

Fig. 233.

Fig. 231.—Superficial Blood-vessels in Pannus. Magnified 2×1.

Fig. 232.—Deep-Lying Blood-vessels in Parenchymatous Keratitis. Magnified 2×1.—Recent case with brush-like branching.

Fig. 233.—Deep-Lying Blood-vessels in Parenchymatous Keratitis. Magnified 2×1.—Old case with broom-like branching of vessels.

3. *Exudation.* This is derived from the same sources as the *hyperæmia.* In violent inflammations of the cornea these are associated with an extensive conjunctival injection an *œdema* of the bulbar conjunctiva and even of the lids. The pus corpuscles poured out by the vessels of the corneoscleral margin, enter the cornea, and travel toward the focus of infection. In this way is formed what is called the *invasion ring* (Fig. 242). If the epithelium is deficient, pus corpuscles also enter from the conjunctival sac. In one of these two ways the corneal *infiltrate* is produced.

Exudation from the iris produces, as in the case of a primary iritis, posterior synechiæ, fibrous membranes on the anterior surface of the iris, and pupillary membranes. If the ciliary body is implicated, deposits may be formed on Descemet's membrane. In a severe suppurative keratitis, the exudate produced by the iris is purulent, forming then a yellowish mass (*hypopyon*[3]) in the bottom of the anterior chamber. The hypopyon, being fluid, is limited above by a horizontal line, and for the same reason is seen to change its place and seek the deeper portions of the chamber when the head is moved. In other cases the mass is gelatinous, and then when seen from in front often has a border that is convex upward, or it lies quite like a solid ball at the bottom of the chamber and does not change its position with the movements of the head. The amount of the pus varies greatly, ranging from a scarcely perceptible crescent lodged in the lowest

[3] From ὑπό, beneath, and πύον, pus.

sinus of the chamber up to masses of such size that the entire chamber is filled by the hypopyon. Hypopyon may disappear by resorption, this process taking place the more rapidly the more fluid the hypopyon is.

Hypopyon being derived from the iris is free from germs and hence produces no reaction in the surrounding tissues. The rapidity with which it is absorbed varies exceedingly. A hypopyon of considerable size may disappear almost completely in twenty-four hours, in other cases it may remain so long that it becomes organized.

Fibrous exudates may be replaced by cicatricial tissue and thus cause closure of the pupil and anterior synechiæ.

Iritis associated with keratitis is purely symptomatic and disappears with the subsidence of the primary process in the cornea.

4. *Impairment of sight* is produced in keratitis by the cloudiness, unevenness, and loss of polish of the cornea, and in severe cases by the presence of exudations in the anterior chamber and the area of the pupil.

530. Etiology and Classification.—Inflammations of the cornea comprise (a) *primary* keratitis, which develops in a perfectly healthy eye, from some cause (traumatism, disease) affecting the cornea directly; and (b) *secondary* keratitis, which is consequent upon some other disease of the eye, especially some disease of the conjunctiva (§ 96). Most forms of keratitis are *ectogenous*, i.e., derived from injury or infection from without; some are *endogenous*, i.e., derived from some agent introduced into the cornea through the blood, (§ 99).

The classification, however, into *ulcerative* and *non-ulcerative* keratitis (see § 527) is the most practical one; and, particularly in the case of the latter variety, we also as a matter of practical convenience distinguish between the superficial and the deep forms. Such a classification lends itself best to clinical differentiation.

II. Ulcerative Keratitis in General

531. Character and Course.—The most frequent form of suppurative keratitis is the *corneal ulcer* in which the process of purulent disintegration begins in the most anterior layers of the cornea (Fig. 234). In this way a superficial loss of substance is produced which is recognizable as a depression in the corneal surface. In the beginning, the loss of substance represents only those parts which are most markedly infiltrated, and hence are the first to break down. Surrounding these are layers, which likewise are infiltrated with the exudate, although not to as high a degree. Hence both the floor and the walls of the ulcer are still infiltrated, gray and uneven, and the wall ends in a sharp edge. The ulcer in this stage is called a *foul* (coated, infiltrated, or *progressive*) ulcer. Later on, the infiltrated portions forming the floor and the wall of the ulcer, so far as they are incapable of living, are also cast off; but those parts of the corneal tissue which have retained their ability to live, remain, are freed by a process of resorption from the exudate which infiltrates them, and become once more transparent. Thus, the ulcer has, it is true, become somewhat larger than before, but has *not* surpassed the limits of the original infiltrate.

This sort of course is found especially in phlyctenular and simple traumatic ulcers. In many other cases, the disease does not thus remain confined to one spot but spreads superficially, so that new portions of the cornea are successively involved in the infiltration and therefore also in the ulcera-

tion. The advance may take place in all directions at once. Thus the whole cornea may be involved, and in the most severe cases may be destroyed en masse. Very often, on the other hand, the disease spreads in certain directions only, so that peculiar forms of ulceration, linear or branched, are produced. The advancing side presents a particularly gray or even yellow cloudiness. Sometimes the ulcer keeps advancing steadily in one direction, while on the opposite side it heals just as fast, so that apparently it goes creeping over the cornea (*serpiginous*[4] ulcer).

532. Perforation of the Cornea.—Beside spreading superficially the ulcerative process may also extend into the depth of the cornea. As soon as it has penetrated so far that the floor of the ulcer has become quite thin, the floor protrudes, because the thin layer of tissue yields to the intra-ocular pressure. This protrusion often fills up the depression formed by the ulcer.

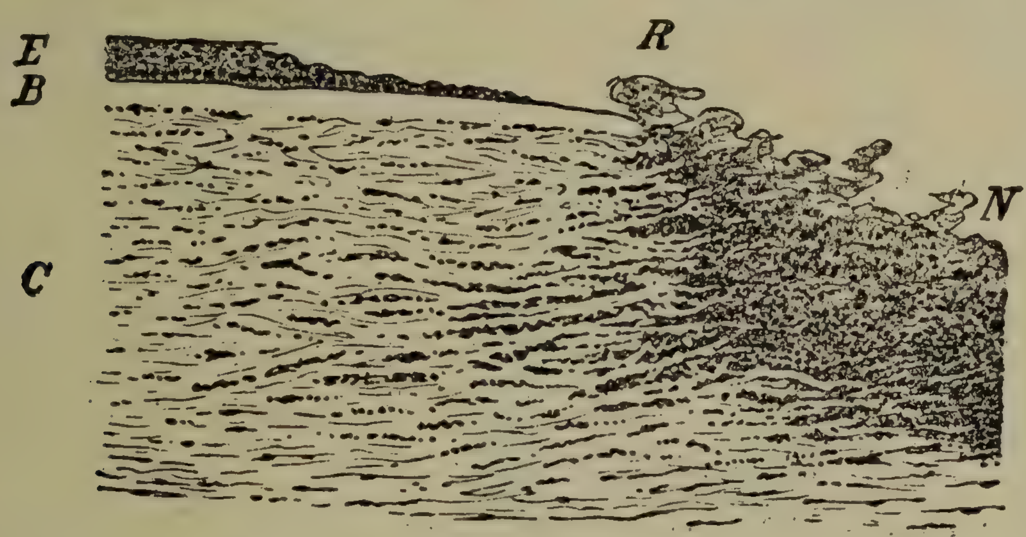

FIG. 234. PROGRESSIVE MARGIN OF A CORNEAL WALL AFTER OPHTHALMIA NEONATORUM (after Salzmann) Magnified 75×1.

E, epithelium, broken off near the margin of the ulcer. *B*, Bowman's membrane cut off short at *R*, the edge of the ulcer. *C*, stroma of the cornea. The infiltration with pus cells increases steadily as the ulcer is approached, and the lamellæ become constantly thinner. Finally the entire structure is converted into a necrotic mass (*N*) lining the floor of the ulcer.

Hence the fact that an ulcer suddenly becomes shallower while the attendant symptoms of irritation persist is a sure sign of impending perforation. Often but by no means always, the thin wall left separating the air from the anterior chamber is Descemet's membrane, which owing to its physiological constitution not only offers resistance to destruction by suppuration but also even though difficultly withstands the intra-ocular pressure. Then there appears in the floor of the ulcer a transparent vesicle surrounded by a clouded gray ring. This condition is called a *keratocele*[5] (hernia membranæ Descemeti, descemetocele). In elderly people in whom Descemet's membrane is considerably thicker than in the young a keratocele lasts longer and is hence more often noticed; in such cases it may even happen, although but rarely, that it lasts for weeks and finally is fixed in place by cicatrization (keratectasia ex ulcere; Fig. 271). In young persons and in children a keratocele lasts but a short time and hence often escapes observation.

Ordinarily when the floor of the ulcer has become so thin that protrusion has taken place, it soon ruptures. This *perforation* of the cornea

[4] From *serpere*, to creep.
[5] From κέρας, horn, and κήλη, hernia.

results usually from some momentary increase of intra-ocular pressure, such as is produced by bodily exertion, coughing, sneezing, pressure of the lids in blepharospasm, or by pressure of the fingers carelessly made in the attempt to open the eye for purpose of examination. The perforation produced by such causes may entail very damaging results.

At the moment of perforation the patient suddenly experiences violent pain and feels a hot liquid (the aqueous humor) gushing out of the eyes, after which, not infrequently, the severe pains previously present subside. The anterior chamber empties at once; the iris and lens are pressed against the cornea so that the anterior chamber is abolished; the eye feels quite soft. The pupil contracts even if it had been previously dilated with atropine.

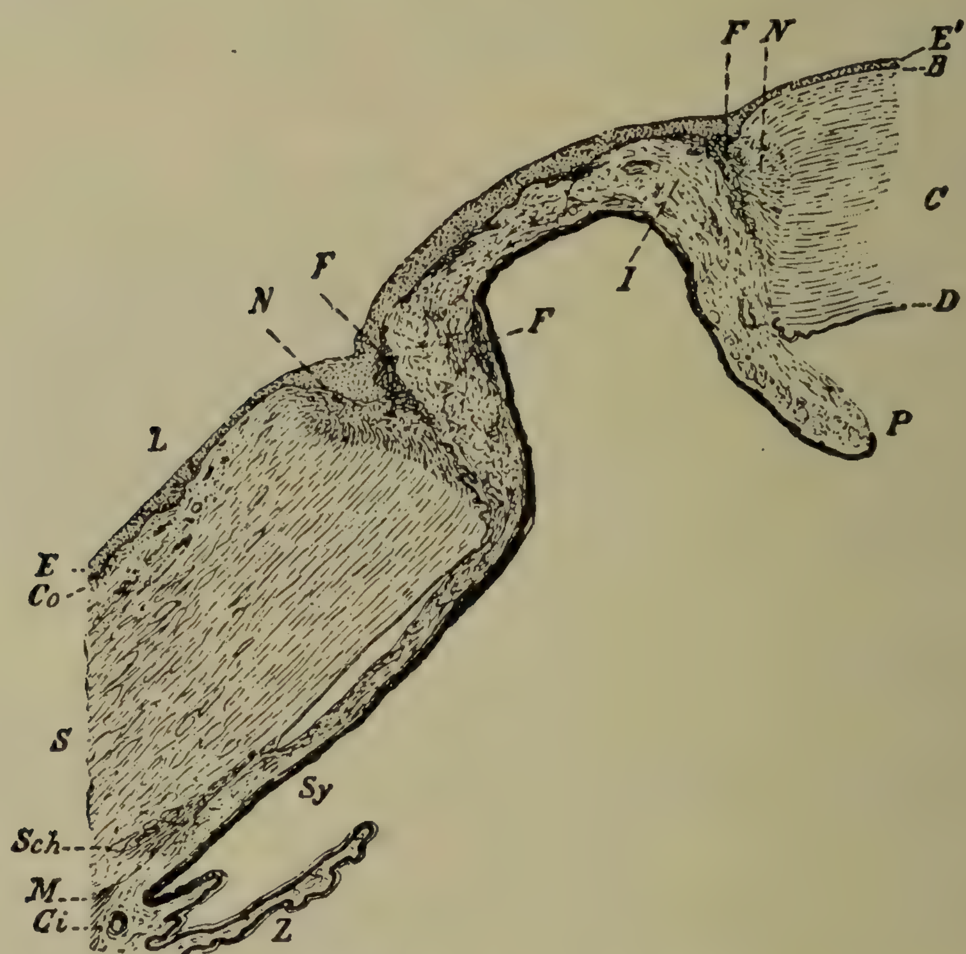

Fig. 235. Partial Prolapse of the Iris in Process of Healing (after Salzmann). Vertical Section Magnified 25×1.

The case was one of ulcer caused by lagophthalmus. Perforation one week before. The prolapsed iris, *I*, is slightly thickened and is attached by fibrin (*F*) to the walls of the ulcer in which the formation of scar (*N*) has already begun. Thickened epithelium, *E*,*E*, forms a continuous covering for the prolapse. The periphery of the iris is adherent to the ligamentum pectinatum, forming the peripheral synechia, *Sy*. *B*, Bowman's membrane; *S*, stroma of cornea; *D*, Descemet's membrane turning forward at the edge of the perforation. *P*, border of the pupil. *L*, limbus. *Co*, conjunctiva. *Sc*, sclera. *Sch*, Schlemm's canal, *M*, beginning of the ciliary muscle. *Ci*, circulus iridis arteriosus maior, indicating the point where the root of the iris was originally attached. *Z*, ciliary process.

Except when it is very small and also occupies the centre of the cornea, the opening formed by the perforation is occluded by the iris. In this way it becomes possible for the anterior chamber to be restored within a very short time, although, to be sure, the iris at the site of the perforation remains permanently connected with the cornea. If the perforation is quite small, the iris simply applies itself to it from behind and there becomes solidly adherent. In case, however, the perforation is larger, the iris, as a rule, is driven into it by the escaping aqueous humor, and thus a *prolapse* or *hernia* of the iris is produced (Fig. 235). This is represented by a hemispherical

prominence which, while recent, has the gray or brown color of the iris. Soon, however, this color is changed because of a layer of gray exudation (F, Fig. 235) which covers the prolapse like a cap and may be removed with a forceps. When the prolapsed portion of the iris is much stretched, the proper color of the iris is lost and the prolapse looks black, because of the retinal pigment on its posterior surface, which appears through the thinner stroma. This is particularly often the case in large prolapses of the iris. The extent of the prolapse of the iris is proportional, first of all, to the size of the perforation. In the worst cases the perforation may comprise the whole cornea, which has suppurated throughout; in that case the iris prolapses almost in its entirety, and the protrusion consists almost wholly of iris together with a small rim of intact cornea (so-called *total prolapse* of the iris, Fig. 236.) But the way in which the prolapse occurs has also an influence upon its size. If the perforation takes place with great force (e. g., while the patient is straining hard), or if the patient behaves in a restless manner after it has taken place, a relatively larger portion of the iris will be driven into the perforation.

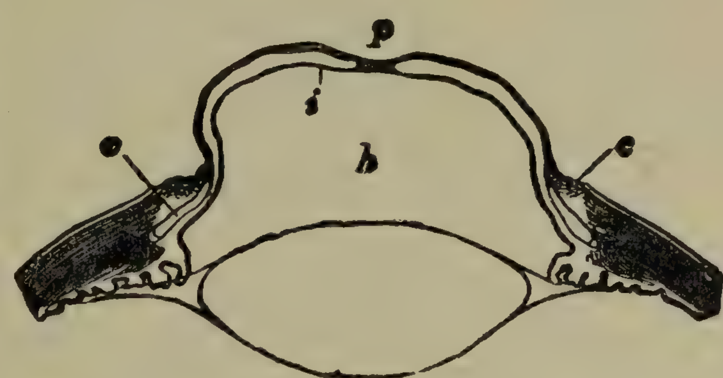

Fig. 236.—Total Prolapse of the Iris (Schematic).

Only the marginal portions, c, of the cornea are preserved, and these are still partially infiltrated. Between them bulges the iris, which is driven strongly forward and which consequently is thinned, so that the pigment, i, upon its posterior surface shines through it and gives the prolapse a blackish hue. The pupil, p, is closed by a membrane, which is continuous with a thin layer of exudate that covers the entire iris. The space, h, between the iris and the lens is the enlarged posterior chamber. Of the anterior chamber only the shallow, slit-like annular space, v, is left. This no longer communicates anywhere with the posterior chamber (seclusio pupillæ).

In a total prolapse of the iris, the pupil is always occluded and is generally closed by a plug of exudate (p), while in partial prolapses the pupil remains free. This is a difference of great importance as regards the future fate of the eye.

533. Accidents in Case of Perforation.—In perforation of the cornea we sometimes see—

(*a*) *Luxation of the Lens.*—After the escape of the aqueous humor, the lens, to reach the cornea, must be pushed forward through the entire depth of the anterior chamber (2.5 mm.), a movement associated with a considerable degree of stretching of the zonula of Zinn. If the forward movement takes place very suddenly, or if the fibres of the zonula have been rendered fragile by disease, the zonula ruptures. In consequence the lens may become tilted, or, if the perforation is large enough may protrude through the opening (phakocele) or may even be expelled from the eye. Expulsion of the lens is more likely to happen in children, in whom the lens capsule is frail and readily ruptures, allowing the lens matter, which in them is soft, to exude through the perforation. More common than prolapse of the lens is prolapse of the vitreous, which takes place if the lens escapes ahead of it or if there was no lens in the eye before the accident.

(*b*) *Intra-ocular hæmorrhages* are the result of the rapid diminution in tension, by virtue of which an increased quantity of blood flows into the

vessels of the interior of the eye, which have been thus suddenly relieved o external pressure, and causes their rupture. Hæmorrhage takes place if the perforation occurs very suddenly, or if the eye beforehand was under a abnormally high tension. The latter is the case in glaucomatous and staphylomatous eyes, in which, furthermore, there is generally also a degeneration of the vessels associated with an increased fragility of their walls The hæmorrhage may be so great that the entire contents of the eyeball are extruded by reason of it; nay, more, the patient may almost be in danger o bleeding to death.

(*c*) Suppuration starting from the cornea may be carried over into the deeper parts of the eye and lead to the destruction of the latter by *severe irido-cyclitis*, or even by *panophthalmitis*. This occurs mainly in the case o extensive destruction of the cornea, especially if, at the same time, the suppuration is of a particularly virulent character, as in gonorrhœal conjunctivitis or in ulcus serpens.

534. Healing of Non-Perforating Ulcers.—When there has been no perforation, healing begins with the regeneration of the epithelium (Fig. 237) This takes place very quickly. Defects involving the epithelium only may be covered with new epithelium in the course of a day. Even in the case o ulcers in the progressive stage, the epithelium strives to cover the defec although it cannot keep its place there, so long as inflammation and exfoliation are going actively on. If, however, disintegration takes place slowly and with but slight inflammatory symptoms (as, for example, occurs in the case of atheromatous ulcers), the ulcer even while in the progressive stage becomes covered with epithelium. But ordinarily the epithelium pushes its way over the defect only in the place where the debris covering the floor is cast off from the edge of the ulcer. As this takes place, the edge o the ulcer loses its cloudiness and becomes rounded off and the inequalities on the floor of the ulcer become filled up. The floor thus becomes transparent, smooth, and gives a mirror-like reflex. The ulcer is called a cleansed or *regressive* ulcer. At the same time the inflammatory symptoms begin perceptibly to abate, and any exudate that may be present in the anterior chamber undergoes absorption.

Accordingly the contrast between a progressive and a regressive ulce is in typical cases very marked. It may be indicated in the following summary way:

Progressive Ulcer	Regressive Ulcer
Inflammatory symptoms marked.	Slight.
Edge of ulcer sharp and infiltrated so that the defect is surrounded by a cloudy areola.	Edge rounded off and free from infiltrate; i.e., the cloudiness, if present at all, does not extend beyond the limits of the defect.
Floor of the ulcer uneven, coated, dull.	Floor smooth and shining.

Under the epithelial covering, *cicatricial tissue* develops. This spring from the stroma of the cornea and gradually fills up the defect until th

epithelium is elevated to about its former level. Since this cicatricial tissue is opaque, the cloudiness returns in the area occupied by the ulcer, but it does not spread beyond the limits of the original defect in the cornea. Furthermore, the inflammatory symptoms keep on diminishing, and the surface remains smooth. By these signs the advent of cicatrization is distinguished from a recurrent infiltration.

The cicatricial tissue consists of cells and a fibrous interstitial substance. The cells, if there has been no perforation of the cornea, are derived from the fixed corneal

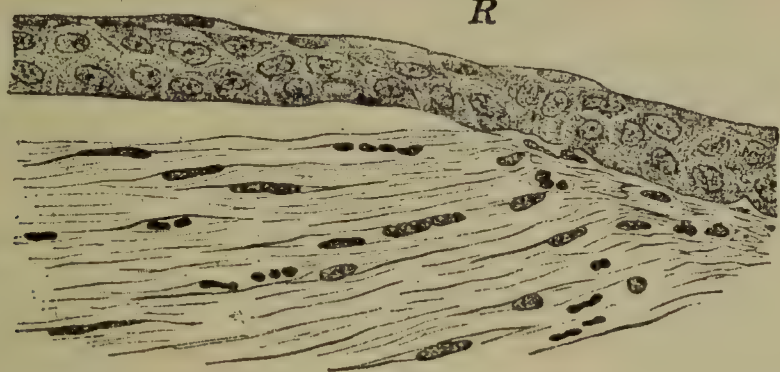

FIG. 237.—MARGIN OF A REGRESSIVE ULCER (after Salzmann) Magnified 400×1.

The edge (*R*) of the ulcer is indicated by the cessation of Bowman's membrane, *B*. The epithelium, *E*, has pushed its way over this edge and has rounded it off. Beneath the epithelium there can be seen on the floor of the ulcer a thin layer of cicatricial tissue, *N*, characterized by its rather finer fibrillation. The stroma (*C*) of the cornea, where it is preserved, contains still some multinuclear leucocytes. The nuclei of the fixed corneal cells are somewhat increased in number and, near the ulcer, are shorter and thicker.

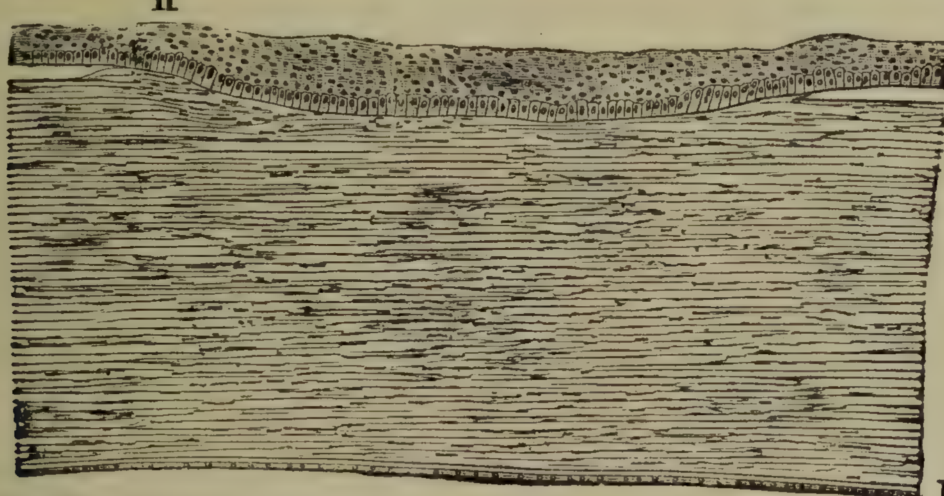

FIG. 238.—DEPRESSED SCAR (FACET) OF THE CORNEA. Magnified 90×1.

In the area of the facet the epithelium *E* shows a shallow depression; Bowman's membrane, *B* is wanting here, and so are the superficial layers of the cornea, *C*. The larger portion of the loss of substance is filled with epithelium only; only at *n* there is a layer of connective tissue between epithelium and corneal parenchyma. Corresponding to this connective tissue there was a delicate semi-circular opacity which marked one edge of the scar. In its other parts the scar was almost entirely transparent and could be recognized only by the flattening of the surface of the cornea. *D*, Descemet's membrane, with its endothelium.

cells (keratoblasts). In recent scars the cells predominate, in older scars the interstitial substance. In the interstitial substance the regular arrangement of fibres which characterizes the stroma of the cornea is lacking. Bowman's membrane is not regenerated, so that the epithelium lies directly upon the cicatricial tissue, from which it is separated by a wavy line (Fig. 238).

The scar tissue, when recent, appears uniformly gray and cloudy. Later when examined with a loupe it assumes a finely fibrous appearance. As time goes on it gets more like the normal corneal tissue, and its cloudiness lessens (the scar "clears"). A true *clearing* of the scar in the sense that the

cloudiness vanishes completely occurs only in infants (e.g., in the scars left by ophthalmia neonatorum). In older children and still more in adults the clearing is incomplete, so that the ulcer leaves a permanent opacity. Moreover, the defect is rarely filled so completely that the surface of the cornea is restored to the normal. Even if ordinary examination shows no defect, examination with the keratoscope (§ 225), shows a slight flattening (*facet* of the cornea, Fig. 238).

The clinical signs of a healing of this sort are these. The cornea shows an area of cloudiness but with lustrous surface and either with no depression or with but slight flattening and unevenness. The eye is free from irritation. The interference with vision depends on the site of the scar.

We may recapitulate in the following statement the distinctive signs of a non-perforating ulcer in its three stages of development:

If the surface over the clouded area is dull, we are dealing with a recent affection, and in that case, if there is no loss of substance, with an infiltrate, but if a loss of substance is present, with a foul ulcer. In both cases the inflammatory symptoms are generally marked. If the surface is lustrous, the affection is an old one, and, if a loss of substance is present with slight inflammatory symptoms, is a clean ulcer; but, if no marked loss of substance is visible and there is no evidence of inflammation, we are dealing with a cicatrix.

In certain cases the scar, instead of being as above stated either flush with the surrounding surface or somewhat depressed, bulges (keratectasia ex ulcere, see § 614). This happens when the scar is so thinned as to give way readily before the intra-ocular pressure.

In other regards the changes found are not always as schematic as here indicated. Thus the distinctive marks of an ulcer may be obscured if the eye is swimming with *tears* which fill up the defect in the cornea and give the latter an apparently smooth and lustrous surface. It may be necessary to keep the eye open awhile—a thing sometimes difficult to do on account of the photophobia—in order to dry off the surface enough to make a correct diagnosis. On the other hand, opacities that are present upon the cornea may be old, and yet the cornea, because of a coincident increase of tension, may look dull and stippled.

Again, the intensity of the inflammatory symptoms is not always a sure guide. Thus in a condition like phlyctenular keratitis, we may find the symptoms increasing although the ulcer is healing, simply because there is another ulcer in process of development. On the other hand certain forms of ulcers, quite dangerous in character, are associated with but slight irritation.

Careful examination of the outline and color of the opacity will help in the diagnosis. Thus, in the case of a cicatrix, the outline of the opacity is commonly better defined than in the case of a recent opacity due to inflammation. The color of the latter form of opacity varies from gray to yellowish white and to yellow; cicatrices, on the contrary, present rather a pure white, or, if thin, a bluish-white hue.

To determine whether an ulcer is covered with epithelium we may use *fluorescein* (§ 389). The progressive ulcer very quickly is colored green or if there is considerable clouding gets a yellowish tinge; while an ulcer which has received a coating of epithelium remains unstained. In very slight defects, the staining also may be very slight, simply a punctate stippling of green showing. In deep ulcers going down to Descemet's membrane only the sides of the ulcer stain with fluorescein, since Descemet's membrane itself does not stain (Butler). This method is especially useful in the case of corneal abrasions and more particularly in recurrent erosions. Scarlet red and collargol act in the same way as fluorescein.

535. Healing of Perforating Ulcers without Incarceration.—Perforation usually produces an immediate favorable effect on the ulcerative process, in the sense that the ulcer ceases to enlarge and tends to heal. The healing of a perforating ulcer in which there has been no prolapse of iris or in which the prolapse has been properly excised, takes place as follows. The perforation opening is finally closed by a firm fibrinous plug. The anterior chamber is then restored. This, however, takes some time, and until then the lens remains in contact with the posterior wall of the cornea, and often retains permanent marks of this contact in the shape of a permanent opacity at its anterior pole (anterior capsular cataract; see § 642). The permanent healing of the ulcer is effected by cicatricial tissue which develops from the stroma of the cornea and little by little replaces the exudate. The posterior endothelium of the cornea also takes part in the process (Hanke). These scars which thus involve the whole thickness of the cornea are much grayer

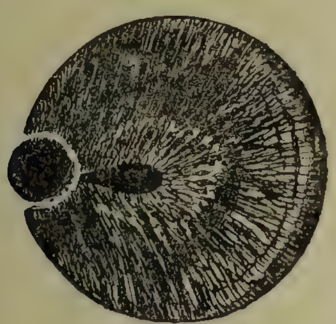

FIG. 239.

FIG. 240.

FIG. 239.—SCAR AFTER PERFORATION OF A MARGINAL ULCER WITH INCLUSION OF THE IRIS. The scar is situated very peripherally, partly in fact in the sclera, a thing which can happen only as the result of ulcers due to phlyctenular conjunctivitis. The dark central portion of the scar corresponds to the incarcerated iris, the white margin to the solid scar-tissue lying between the iris and the border of the former perforation aperture. The pupil is pear-shaped, with its pointed end displaced toward the scar, and corresponding to this the iris on the opposite side is put on the stretch. Consequently, the pupillary zone and the ciliary zone are broadened, and furthermore the zone of peripheral crypts, which otherwise would be concealed behind the limbus, becomes visible under the form of a dark stripe at that portion of the periphery of the iris opposite the scar.

FIG. 240.—SCAR WITHOUT INCLUSION OF THE IRIS. Here there was also a phlyctenular ulcer which had perforated. The prolapsed iris had been excised in the proper way. Consequently, it is no longer connected with the scar, but over an area corresponding to the scar shows a coloboma. The pupil is permanently dilated because of the gap made in the sphincter pupillæ.

and more opaque than those found in non-perforating ulcers (see Fig. 240).

Sometimes the perforation does not close completely by cicatrization, so that a minute aperture remains permanently (*fistula of the cornea*). This appears under the guise of a small dark point, surrounded by opaque, whitish, cicatricial tissue; the anterior chamber is absent, the eye is soft. If the fistula persists for a long time, the eye gradually perishes. Sometimes the fistula closes but only by a thin membrane which protrudes in the form of a vesicle. When this happens an increase of tension is apt to set in, that may lead to a renewed rupture of the occluding substance, which is still but slightly resistant. An alternation of this sort between an open fistula with softness of the eyeball, gradual closure of the fistula, and a consequent steady increase of pressure until the cicatrix ruptures anew, may be repeated for a long time, until finally a severe inflammation or an intra-ocular hæmorrhage occurs, which leads to the atrophy of the eye, and thus puts an end to the process. In other cases the cornea flattens, the eye grows softer and softer, and at length goes blind from detachment of the retina.

Fistulæ of the cornea occur mainly as a sequel of perforations that lie opposite the pupillary margin of the iris, so that the iris cannot block the opening completely, but simply lies with its pupillary border in the perforation orifice. In this case often there is is no solid connection present between the iris and the cicatricial tissue, minute intervals remaining between the two. Hence, corneal fistulæ do not as a rule present wide canals lined with epithelium, and yet the scar-tissue filling the perforation opening is not dense, but is permeated by fissures through which the aqueous keeps oozing until it reaches the exterior surface of the cornea (Czermak). The origin of other fistulæ is that the prolapsed iris splits apart under the strain produced by the pressure of the aqueous, and the opening thus formed never closes solidly again. Lastly, in very extensive prolapses of the iris, it may happen that in the process of cicatrization a fistula is left at the spot corresponding to the pupil.

536. Healing of a Perforating Ulcer with Inclusion of the Iris.—If there is a *partial prolapse* of the iris (Fig. 235) a fibrinous exudation glues the prolapse to the wall of the ulcer and causes a temporary closure of the opening. The anterior chamber is then soon restored. After this the epithelium (*E*, Fig. 235) shoots out and covers the opening over. Under this covering, cicatrization in the true sense of the word takes place. In this process the walls of the ulcer (*N*) including both the tissue of the cornea and the prolapsed iris, take part. From these structures connective-tissue cells grow out and in consequence the fibrinous exudate is replaced by cicatricial tissue. If the iris is but slightly prolapsed it takes but little part in the process, sending out perhaps simply some vascular offshoots. If the prolapse is large and there is a considerable degree of inflammation of the whole eye, the prolapsed iris is also actively inflamed. Its vascular layer then is converted into a sort of granulation tissue, so that the prolapse loses its original iris color and becomes grayish-red. As this granulation tissue is converted into cicatricial tissue, gray bands form on the surface, which grow broader, fuse, and contract, thus making the prolapse constantly flatter and flatter. This cicatricial tissue is continuous with the cornea along the wall of the opening, and hence attaches the prolapse firmly in the latter. Cicatricial tissue developed from the corneal endothelium also forms on the inner surface of the cornea, so that the adhesion between the latter and the iris always spreads beyond the limits of the opening.

The outcome of this process is a scar which is seated in the cornea and which on ordinary clinical examination would seem to be a scar derived from corneal tissue. This, however, it is in part only, the rest being a portion of the iris which has been transformed into a cicatrix. With this cicatrix the remainder of the iris lying in the anterior chamber is solidly united. Such an adhesion of the iris to a corneal cicatrix is called an *anterior synechia*.[6]

In the process of cicatrization the vascular layer of the iris is converted into cicatricial tissue but the pigment epithelium on the posterior surface of the iris and the muscle fibres of the sphincter pupillæ are preserved intact. If then the cicatricial layer happens to be thin, the pigment layer shows through, making the scar look slate-colored. About this darker area the portion of the scar representing the wall of the perforation stands out as a white ring (Fig. 239).

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[6] From συνέχειν, to connect. The term *leucoma adhærens* (from λευκός, white) is also employed to denote a cicatrix of the cornea with anterior synechia, especially if dense and white.

Owing to the fact that the iris is drawn forward to the scar, the pupil loses its round shape and is drawn in toward the site of the adhesion. To what extent this is the case depends upon where the perforation is situated and what part of the iris is prolapsed. In peripherally situated ulcers the pupil is drawn strongly toward the site of the perforation; it has the shape of a pear, the tapering end of which is directed toward the site of the synechia (Fig. 239). If, however, the perforation is near the centre of the cornea (as in Fig. 235), the pupillary portion of the iris becomes engaged in it in healing and in this case the distortion of the pupil is slight, or entirely absent.

In ulcers with inclusion of the iris, which have healed smoothly, the adhesions affect not only the prolapse itself but also the periphery of the iris, thus forming a *peripheral synechia* (Fig. 235, *Sy*; cf. also § 839). This may occur not only in the sector occupied by the prolapse, but may extend a considerable distance beyond, even to the opposite side. It can readily be understood from this why eyes with anterior synechiæ are predisposed to develop secondary glaucoma (§§ 848, 849).

The sort of cicatrization above described is the rule in the case of small prolapses of the iris. Rarely do these produce a staphyloma (§ 617).

The cicatrization of a *total prolapse* of the iris is effected in the same way as in the case of a partial prolapse. Now, however, the pupil, being involved in the prolapse is blocked, first by fibrinous exudate, afterward by cicatricial tissue, so that a *seclusio pupillæ* (§ 687) is produced. The subsequent course depends upon the intra-ocular pressure. If this rises, a *staphyloma* of the cornea develops (§ 617); if it sinks, the scar and the remnants of the cornea are flattened out (*applanatio corneæ*), or if all the cornea except the marginal rim is destroyed, the scar representing the totally prolapsed iris shrinks down into a small perfectly flat cicatrix which takes the place of the cornea, (*phthisis corneæ*).

III. Ulcer of the Cornea

537. Varieties.—The general characters and course of corneal ulcers have been given in the sections just preceding and the general etiology has been described in § 96. We need therefore now simply name the separate varieties with their differential characteristics.

A. The **secondary** ulcers of the cornea have already been mentioned in connection with the conjunctival diseases which cause them. They include—

1. The *catarrhal* ulcer (§ 451). This is marked by its situation at the margin of the cornea, its crescentic shape, its comparatively slight tendency to extension, and its usually benign course.

Several crescentic ulcers may be present in the same eye at different parts of the circumference of the cornea; nay, more, by their confluence an annular ulcer may be formed, completely encircling the cornea. In the latter case the annular opacity which is left has a great resemblance to the arcus senilis corneæ. In cases where such an annular ulcer has penetrated deeply, the very detrimental result of a permanent ectasia of the cornea has been observed. For by stretching of the floor of the ulcer the base of the cornea at the point where the ulcer is situated is pushed forward, and the whole cornea assumes an oblique position. If the ulcer completely surrounds the cornea, the latter

may give way before the intra-ocular pressure and move forward en masse. In this case the region of the cornea inclosed by the annular ulcer lies like a watch glass on top of the marginal portions of the cornea (keratectasia ex ulcere, § 614). Other forms of ulceration observed in catarrhal conjunctivitis are the clear facetted ulcer (see No. 3 below), minute deep ulcers like gimlet holes, and minute superficial ulcers in a circle midway between the centre and the circumference of the cornea. These last occur specially in children and heal quickly (Butler).

2. The ulcers of *phlyctenular conjunctivitis* as well as those of *conjunctivitis ex acne rosacea* are small, superficial, and generally, although not necessarily, marginal. In the progressive stage they show a whitish infiltration and have little disposition to extend laterally, but rather tend to burrow deeply. They hence often, form crater-shaped depressions and quite often in spite of their small size, cause perforation (Fig. 239). A special form is the vascular fasciculus (fascicular keratitis; see § 483).

3. *Trachomatous* ulcers (§ 469) are either marginal like the catarrhal ulcers or occur with pannus, in that case lying beneath the lower edge of the pannus. Subsequently the pannus may grow over the floor of the ulcer. Rarely ulcers form within the pannus. These forms of trachomatous ulcer have nothing characteristic in their shape or course.

The *central, non-irritative* ulcer occurring in trachoma and also in Koch-Weeks, pneumococcus, and mixed infections develops generally in the centre of the cornea. It is distinguished by the absence of accompanying symptoms of irritation, so that often the disturbance of vision is the only thing that calls the patient's attention to his trouble. Objectively, the ulcer is distinguished by the fact that even during the progressive period it is but very slightly clouded, so that it scarcely gives any evidence of its presence except the loss of substance that it produces; it may therefore be very easily overlooked if we do not carefully test the corneal reflex. It has a tendency to fill up incompletely with cicatricial tissue, so that a central facet remains which causes very great deterioration of sight by the production of irregular astigmatism.

4. The ulcers in *gonorrhœal* and *diphtherial* conjunctivitis (§§ 461 and 478) are marked by their severity, their rapid extension and their purulent character. They often cause total destruction of the cornea and consequent total prolapse of the iris and staphyloma of the cornea. As the infiltrated tissue disintegrates very rapidly, and as the yellowish-gray cloudiness of the floor and walls of the ulcer is slight in comparison with the real gravity of the disease, the perforation often occurs before the tyro expects it. The prognosis of these ulcers is extremely bad. Of much milder type are the multiple superficial ulcers found in metastatic gonorrhœa (§ 461).

B. The **primary** ulcers of the cornea are—

5. The *infectious marginal ulcer* (zur Nedden). This is mainly characterized by its etiology, being caused by zur Nedden's bacillus.

This condition is quite frequent in the Rhine country; elsewhere it is found rarely or not at all (although this may possibly be due to the difficulty of demonstrating the bacillus). The clinical symptoms are quite like those of the catarrhal marginal ulcers, but there is no conjunctivitis, since the bacillus is pathogenic only for the cornea. Less commonly ulcers develop in the central part of the cornea. The prognosis is good.

6. *Small marginal* ulcers of the cornea often develop in elderly people, especially of the male sex, without any discoverable cause. They begin

with pretty violent symptoms but are scarcely of the size of a pin's head, and heal rapidly without penetrating deeply. Recurrences are frequent so that many people have attacks one or more times every year. The uratic diathesis appears to be a frequent cause of these ulcers, and treatment directed against this diathesis and consisting of the proper dietetic regulations or the use of mineral waters often prevents the recurrence.

7. *Traumatic* ulcers may originate in a superficial defect caused by a wound, abrasion, burn or chemical corrosion, the extrusion of a foreign body, etc. They may also be caused by the cumulative effect of repeated slight irritations, e.g., those due to ingrowing lashes, the pressure of cicatrices or small growths in the lids, concretions of the conjunctiva, etc. They usually occupy the zone of the cornea that lies in the palpebral fissure, and are generally small, round, shallow, and only moderately infiltrated. If trichiasis is the cause, the ulcer is usually combined with rather marked vascularization of the cornea. Traumatic ulcers in general usually run a benign course. But we must not forget that quite inconsiderable injuries give rise often to a very different, dangerous type of ulcer (ulcus serpens; see § 550).

538. Etiology.—Ulcers of the cornea are among the most frequent affections of the eyes, and special significance attaches to them because the opacities that they leave very often impair the sight. Ulcers of the cornea, if we except those resulting from phlyctenular conjunctivitis are found much oftener in adults, and specially in elderly people, than in children. It seems that in the later years of life the cornea is less well nourished and is hence more disposed to disintegrate than in youth.

The results of *bacteriological examination* in some cases of corneal ulceration are negative; in other cases we find the same bacteria that are familiar to us as occurring in the conjunctival sac. This should not surprise us, since the cornea certainly is most often infected by invasion from the conjunctival sac. A perfect correspondence between the individual variety of corneal ulcer present and a definite morbid germ, so that from the appearance of the ulcer one could diagnosticate the germ with certainty, exists only for the keratitis produced by mold fungi and to a less extent for ulcus serpens, which, at least in the majority of cases, is caused by the pneumococcus. The pneumococcus, however, may also cause ordinary ulcers of the cornea and particularly the clear facetted ulcer, which is likewise produced by severe inflammation caused by the Koch-Weeks bacillus or mixed infections (Butler). Marginal ulcers such as occur in conjunctival catarrh are often produced by the diplobacillus of Morax-Axenfeld or the allied bacillus of Petit, sometimes also by zur Nedden's bacillus. According to Butler, except in mixed infections, the Morax-Axenfeld diplobacillus is not very often a cause of corneal ulceration. In any event, the bacterial findings in the case of corneal ulcers vary greatly.

Some ulcers, e.g., the marginal ulcers of old people (§ 537) and marantic ulcers (§ 559) are due to constitutional causes; and in others that are not produced directly in this way the depressed general state of the patient often affects the course unfavorably and, unless attended to, may lead to destructive changes in the cornea. This is particularly the case with the ulceration of the cornea occurring in marasmic infants affected with ophthalmia neonatorum. Here ulceration may progress in spite of all local measures, and every effort must be made to reinforce the patient's resisting power by general treatment. Again, many believe that deficient nerve power is at the bottom of many cases of ulceration following vesicle formation (herpes of the cornea, recurrent erosion ; see §§ 564 and 591), and in others hypothyroidism seems to be a factor.

539. Prognosis.—In view of the very varying course run by ulcers of the cornea the physician who is called to a patient with an ulcer of the cornea must determine not only with the diagnosis but also the prognosis ; he should be able to predict to the

patient to what extent his sight will suffer permanent impairment, in order that such impairment may not afterward be charged against the medical treatment. The prognosis for vision depends upon the situation, the extent, and the density of the opacity which the ulcer has left behind it. Small opacities, even when dense, are generally less injurious to vision than those which are less dense but extensive (§ 610). It is hence less serious for an ulcer to extend into the depth of the tissue than upon the surface. If an ulcer is progressing in the direction of the centre of the cornea, every millimeter of advance causes additional injury to the vision, while an extension toward the corneal margin is almost a matter of indifference. No further disintegration is to be apprehended at those portions of the margin of the ulcer to which vessels have already penetrated, and, so too, a portion of the cornea, covered by pannus, is protected against suppuration in gonorrhœal conjunctivitis. In every instance the ulcer is arrested at the limbus, as it never makes its way into this or into the adjacent sclera. The only exception to this is formed by those ulcers which not infrequently develop from the nodules of phlyctenular conjunctivitis situated in the limbus (Fig. 239 and 240). Even extensive suppuration of the cornea, as in gonorrhœal conjunctivitis, ulcus serpens, etc., always leaves a narrow rim of cornea intact, which, to be sure, is often not of sufficient size to render possible an iridectomy for the restoration of vision. It must be remembered that the course and hence the prognosis of keratitis are sometimes much affected by the patient's general condition, and the keratitis is not completely or at least rapidly relieved until the proper constitutional treatment is applied.

540. **Treatment.**—Ulcers of the cornea are very amenable to proper and energetic treatment. They hence in general afford a favorable prognosis if they come under treatment early; in the great majority of cases it is possible to put a stop to their progress, and produce regular cicatrization. The treatment varies according to the stage.

Causal Treatment.—Recent ulcers that are still *foul* (progressive) require, most of all, the consideration of the *causal indication.* In traumatic ulcers any foreign bodies that are still present must be removed. Cilia which are directed against the cornea must be epilated; papillomata of the edges of the lids, when causing trouble in the cornea, must be removed. In the numerous cases in which the ulcer of the cornea is caused by a conjunctival lesion, the treatment of the latter forms, as a rule, the most important part of our therapeutics, and under it, moreover, the ulcer advances toward recovery. Hence, in corneal ulcers resulting from catarrh, trachoma, or gonorrhœal inflammation of the conjunctiva, we must by no means desist from cauterization of the latter, if it is required by the conjunctival trouble. So too, in cases of diplobacillus conjunctivitis, we continue using zinc sulphate (see § 453) after ulcers have developed, and with good effect on the latter. The only precaution that must be observed is that when caustics are applied they should not come into contact with the cornea itself—a contingency which can be avoided by carefully washing off from the conjunctiva any excess that may be present. Furthermore, we should cauterize with the silver solution only, and not with the copper stick, which is too irritating. Moreover, as long as progressive ulcers are present in the cornea, no irritating collyria, such as the collyrium adstringens luteum and the like, should be instilled, as they would then come into contact with the cornea.

To fulfill the causal indication, we must in many cases apply constitutional and hygienic treatment. Thus, if the disease has a malarial basis as in many cases of den-

dritic keratitis, we must use quinine; in herpetic and other cases in which there is evidence of nerve disturbance, we use arsenic (which, indeed, seems helpful in a variety of corneal conditions). In some intractable forms thyroid extract may be required, although this is more often indicated in non-suppurative keratitis. Fresh air and good diet, careful regulation of the bowels, and, in general, measures to promote nutrition and prevent accumulation of waste matter are indicated in all cases, but particularly in the marantic ulcers (§ 559), the uratic ulcers (§ 537), and the ulcers of phlyctenular conjunctivitis. In the last-named condition minute doses of calomel are of service (see § 488), and in ulcers generally urotropine, 1 gm. every 3 hours, is beneficial and may render cauterization or paracentesis unnecessary (Kilpatrick). Finally, we should look for and remove any latent focus of infection in the teeth, tonsils, nasal cavities, etc.

541. Bandage and Atropine.—The *indicatio morbi* requires in most cases the application of a *protective dressing* (see § 116). This as a rule, is to be kept on until the ulcer gets clean and becomes lined with an epithelial covering which protects the cornea against exterior influences. When the floor of the ulcer is thinned and shows a tendency to bulge, a *pressure dressing* must be applied and kept up until the freshly formed cicatrix is sufficiently strong to offer resistance to the intra-ocular pressure.

A contraindication against the bandage is furnished by a profuse secretion, because the latter would be retained in the conjunctival sac by the closure of the lids, and would remain in constant contact with the ulcer. For this reason, in ulcers resulting from conjunctivitis the bandage must be very often dispensed with. In quite small children, also, the bandage is generally useless, since it soon gets displaced; and a badly applied bandage is more hurtful than any exposure of the eye could be.

Next to the bandage *atropine* plays the most important part in the treatment of ulcers. It combats the inflammation of the iris, hence diminishes the general state of irritation, and so reacts favorably upon the ulcer itself. It also often markedly relieves the pain and photophobia. It must be instilled as often as is necessary, in order to keep the pupil steadily dilated. In cases where the evidences of iridic irritation (ciliary irritation, photophobia) are slight, it may be omitted.

In such cases or in cases that promise to be transient, atropine may tentatively be replaced by homatropine.

542. Antiseptic Applications.—With these two remedies alone—the bandage and atropine—we attain our object in mild cases. For those ulcers, however, which, from the purulent hue or from the great infiltration of their surrounding parts, show a rapidly *progressive* character, we must employ still other remedies. Some of those most used are—

Moist hot compresses (see § 118) applied every day for an hour or more, the dressing being left off each time for the same period.

In some cases *phototherapy*, *actinotherapy* (§ 119) or *iontophoresis* (§ 120) with zinc sulphate have been useful.

Eyebaths of *magnesium sulphate* (8 per cent or stronger) used for five minutes every two or three hours are serviceable especially in trachomatous infections (Kilpatrick).

Very finely powdered *iodoform* sprinkled upon the ulcer or applied in 10-per-cent ointment.

Absolute *alcohol* or tincture of *iodine* or a solution of iodine in vasogen applied to the ulcer with a cotton-tipped swab. This very effective though very painful application can be used with considerable freedom, as it rarely does injury to the intact cornea. *Formaldehyde* (1:50) may be similarly applied (Wilmer).

In phlyctenular and other superficial ulcers, powdered *calomel* dusted into the eye or in the latter stages 1-per-cent *yellow oxide of mercury ointment* (see § 488).

Collargol ointment (one per cent). This is non-irritating. It stains the denuded portion of the ulcer brown (thus serving like fluorescein to indicate its limits and the progress of treatment).

Subconjunctival injections (see § 135) of a mercury oxycyanide solution or a 10-per-cent salt solution made beneath the bulbar conjunctiva.

543. Cauterization.—If in spite of these remedies the ulcer is evidently spreading, we must proceed to cauterize the ulcer with the actual cautery. For this purpose we use a small sharp-pointed cautery iron, or the galvano-cautery loop, or Paquelin's thermo-cautery, making the application lightly to the ulcer wherever it shows a gray coating. In the case of extensive ulcers it is not necessary to cauterize the entire ulcerating surface, but it is sufficient to destroy the most infiltrated portion of its margin, at which an advance of the ulcerative process is to be expected. Before cauterizing we make the cornea insensitive by repeatedly instilling a 4-per-cent solution of cocaine.

The introduction of the actual cautery, which was first proposed by Martinache, was a great step forward in treatment; for the ulcers against which we were often powerless before, namely those which are deeply infiltrated and rapidly progressive, are just the ones that are usually arrested at once by this means. The application of the cautery is painless when cocaine is employed, and does not, as might be supposed, cause any marked irritation of the eye. On the contrary, after its application the pain often ceases instantly, while the other symptoms of irritation abate. In private practice, when one has no other means to resort to, the head of a probe or of a knitting needle, heated red-hot may be employed for cauterization. The chief thing is to make the cauterization extensive enough. Perforation of the thinned floor of the ulcer can, with care, be easily avoided; should it occur, however, it has no bad results beyond what the perforation itself gives rise to, since the hot point is cooled at once by the outpouring aqueous humor. An opacity remains permanently at the cauterized spot; but since we only cauterize such places as would otherwise go on to purulent disintegration, the final opacity is not greater than it would have been in any case.

For the actual cautery may be substituted other forms of heat, such as instillations of very hot water on the cornea, the application of hot air, chauffage (Weekers), or the thermophore (Shahan) (see § 118). Or we may employ a chemical caustic, e.g., *nitric acid* or 95-per-cent *carbolic acid.* To apply the former we may use a wooden tooth-pick soaked in the acid and then dried (Jackson). We must be careful that neither agent comes into contact with the sound cornea.

544. Paracentesis.—Another potent remedy for combating rapidly spreading ulcers is *paracentesis* of the anterior chamber (§ 941). People were led to do this operation by observing that ulcers after spontaneous

perforation generally went on to rapid healing (§ 535). In a similar manner artificial perforation—i.e., puncture of the cornea—performed early prevents the extension of the ulcer and its threatened rupture. This is better than waiting until the ulcer spontaneously perforates the cornea, because in the latter case the ulcer keeps enlarging superficially and thus produces a more extensive opacity, and, furthermore, because after ulcerative perforation of the cornea a prolapse of the iris almost always develops, leading to the formation of an anterior synechia, while with a properly performed puncture this is not the case.

If perforation is imminent, and we prefer not to bring it about artificially by puncture, we take care to keep the patient quiet in bed and wearing a light pressure bandage in order that the perforation may take place gradually, and that as little as possible of the iris may be driven into the opening.

For *transplantation* of the conjunctiva in rapidly advancing ulcers, see § 917.

545. Treatment of Perforating Ulcers.—When perforation of the cornea has taken place, treatment has to aim at the following objects: in the first place, that the iris shall not adhere to the cornea, or at least shall do so to the smallest possible extent; in the second place, that a firm and flat (not ectatic) cicatrix shall be formed. The two objects are attained as follows:

1. If the *perforation is quite small* the iris does not prolapse into it, but simply becomes applied to its posterior orifice. In such cases, rest, bandage, and atropine suffice for the treatment. There then remains only a punctiform adhesion of the iris to the corneal cicatrix, and this is often subsequently drawn out into a thin filament. In particularly favorable cases no anterior synechia at all may be produced; for instance, if the iris, before it has become firmly adherent to the site of perforation, is pushed away from it again by the reaccumulating aqueous.

2. If—in the case of a perforation of greater size—the *iris has prolapsed*, it should be excised. A replacement of the iris into the anterior chamber (reposition) in most cases would be impossible of performance, and even if it should succeed, would have no lasting results, since the iris would continually prolapse again. For the performance of *excision* we first make the cornea insensitive by means of cocaine. Then with a sharp-pointed instrument (conical sound) we separate on all sides the adhesions of the prolapse to the perforation opening, draw the iris as far as possible out of the wound with the forceps, and snip it off close to the cornea (Leber). If the iris still projects anywhere into the opening, it must be freed with a spatula and pushed back into the anterior chamber. If the operation has been successful, the iris ought no longer to be attached anywhere to the margin of the aperture; on the contrary, there should be a coloboma of the iris (Fig. 240) with free pillars, as after a regular iridectomy. In this way we obtain a firm cicatrix without inclusion of the iris.

3. Excision of prolapse should *not be done* if (*a*) it is very large, for then there would be left a large gap in the cornea which would take a long time to

close and would entail danger of injury to the lens or infection of the eye; (*b*) the ulcer is still advancing. In this case we wait until the perforation has produced its customary favorable effect on the progress of the ulcer. Prolapses 4 or 5 days old can be excised as well and as completely as those just formed. (*c*) If the prolapse is over a week old as by that time it is so solidly adherent that it cannot be detached. (*d*) If infection would be likely, e.g., if there is a purulent conjunctivitis, a dacryocystitis, etc.

In cases where excision cannot be done, and especially in *old* or *very extensive prolapses* of the iris, we confine ourselves to the attempt to transform the prolapse into a firm and flat cicatrix. In many cases a pressure bandage applied for a long time accomplishes this end. If we cannot attain our object in this way, as is particularly the case when the prolapse is constricted at its base like a mushroom, we must produce flattening of the prolapse by repeatedly puncturing it or by excising a small portion. In the case of a very bulging total prolapse of the iris, it is advisable to split it transversely, and then, after opening the anterior capsule, to expel the lens. If there is a remnant of sound cornea left broad enough for the performance of an iridectomy, this operation is an excellent means for securing a flat cicatrix.

4. In *keratocele*, rest, the application of a bandage, and ultimately the puncture or cauterization of the protruding vesicle, are required.

5. In *fistula of the cornea*, in order to effect its closure, everything must be avoided that might temporarily increase the ocular tension, and thus force the fistula open again just as it is closing. For this purpose we order rest in bed, with the application of a light bandage to both eyes, while at the same time we instill a miotic (eserine or pilocarpine) in order to diminish the pressure in the anterior chamber. An iridectomy has a very good effect, but this can be performed only when the anterior chamber has been, at least to some extent, temporarily restored. If these measures fail, we must remove the cicatricial margins of the fistula either by excising or by cauterizing them (with a blunt galvano-cautery or thermo-cautery point) so as to fill the fistula up with new, firm scar tissue. Here again, however, if we are going to perform cauterization, there must be some remnant, even though a shallow one, of the anterior chamber present, as otherwise we should singe the anterior surface of the lens. In fistulæ that we cannot close by these means, we often succeed by removing the epithelium covering the fistula and sewing over the orifice a flap taken from the adjacent conjunctiva (§ 917) or we excise the fistula and the surrounding cicatricial tissue with a corneal trephine and implant in the orifice a plug of healthy cornea or of fascia lata.

546. Treatment in Regressive Stage.—The treatment of ulcers in the regressive period, or *period of cicatrization*, should aim at filling the loss of substance completely with a resistant cicatrix, and at rendering the latter as transparent as possible. For the attainment of both objects irritants are employed. We begin cautiously with the weaker remedies, passing gradually, if these are well borne, to the stronger ones. One of the mildest of irritants is powdered calomel; more energetic is the action of the yellow-

precipitate ointment (from 1 to 4-per cent), the collyrium adstringens luteum (§ 457, Note 3), tinctura opii crocata, and dionin. Calomel and the yellow ointment can also be used with advantage in the earlier stages of corneal ulceration (§ 542). In applying the yellow-precipitate ointment, we insert it into the conjunctival sac with a brush or glass rod, and then by rubbing it about with the upper lid perform a sort of massage upon the clouded cornea. Another irritant remedy that is recommended is the application of hot steam or hot air (see § 118). It is advisable to continue the application of these irritants for a long time in order to secure the greatest possible clearing up of the cornea, but in so doing the remedies must be changed from time to time, as otherwise the eye gets accustomed to them and they lose their efficacy.

547. Ulcus Rodens.—The ulcus rodens of Mooren (*Mooren's ulcer, chronic serpiginous ulcer* of Mayou) is a superficial ulcer which develops from the margin of the cornea (usually the upper margin) with marked inflammatory complications. From the sound portion of the cornea it is limited by a gray, clouded margin, which is evidently undermined. This latter symptom is characteristic of ulcus rodens. After a short time the ulcer begins to grow clean and to cicatrize, becoming covered with vessels from the limbus. Just when one supposes the process to be nearing complete recovery, a relapse sets in with a return of the symptoms of irritation, and in this the ulcer pushes its way forward somewhat farther in the cornea. So the disease goes on with discontinuous attacks and intervening remissions, until the ulcer has covered the entire cornea. The latter is thus everywhere deprived of its superficial layers, and hence remains permanently clouded throughout its entire extent, so that vision is very greatly diminished. In some cases however, the clouding is slight. The ulcerated area, as Mayou remarks, shows but little tendency to fill up with new tissue. Perforation of the cornea in this affection has never been observed. This rare disease attacks elderly people, and not infrequently invades both corneæ either simultaneously or in succession. It was regarded as incurable but can usually be controlled by the actual cautery applied in a thorough-going way to the undermined edges of the ulcer. A few cases have been cured by tuberculin injections or by iontophoresis with zinc sulphate.

548. Keratitis Marginalis Superficialis.—This condition has many traits in common with ulcus rodens. It is a rare disease, found in persons in middle life. A quite superficial ulceration spreads over the cornea, starting from its margin. It does not, however, start from all parts of the margin at the same time, nor does it advance uniformly. Consequently the ulcerated marginal zone of the cornea is demarcated from the transparent central portion by a sinuous border formed by a fine gray line (Fig. 241). This variety of keratitis drags on for a long time—sometimes for years—periods of intermission alternating with relapses, which are associated with moderate symptoms of irritation. It is distinguished from ulcus rodens in that the ulcer is extremely shallow, and hence the cornea within its limits shows only a very faint and grayish opacity. Moreover, the edge of the ulcer, scarcely visible in any case, is not undermined. The ulceration never reaches the centre of the cornea, so that the extremely faint opacities that remain do not interfere with sight. A frequent result of keratitis marginalis superficialis is that the conjunctiva is drawn up over the cornea in the form of a pseudo-pterygium (see § 504).

IV. Ulcus Serpens (Hypopyon Keratitis)

549. Character.—Ulcus serpens is a grave form of keratitis which starts in the centre of the cornea and spreads thence to the periphery. It tends rather to spread superficially than to cause perforation, but in bad cases the whole cornea may be destroyed. The inflammation has a markedly purulent character, evidenced by the intense yellowish-white

color of the infiltrate and the regular development of hypopyon. Because of its situation and character it always entails a considerable impairment of sight and may cause complete blindness.

550. Etiology.—Ulcus serpens originates in infection of the cornea by the pneumococcus. Such infection presupposes, first, a lesion of the corneal epithelium, which in the normal state protects the cornea against the entrance of micro-organisms. This lesion in most cases is caused by an *injury* of the cornea. The injuries which lead to the formation of the ulcus are, as a rule, very slight, consisting in a simple scaling off of the epithelium, such as is caused by a rough cloth, a leaf, or a branch grazing the cornea, small foreign bodies flying into the eye, and scratches produced by contact with slivers of wood in those engaged in wood-chopping or fragments of stone in those engaged in breaking stone. Even when a typical ulcus serpens appears to originate spontaneously, it is probable that there has been an antecedent injury, since such slight injuries of the cornea as these are readily overlooked by the patients. Cases in which an epithelial lesion has apparently developed spontaneously include an ulcus serpens which occurs in herpes of the cornea (from spontaneous rupture of the vesicles) and the kind that very often occurs in eyes rendered blind by glaucoma. Even here slight injury acting on an insensitive eye may be the real cause. In exceptional cases severe perforating injuries, and likewise operation wounds, may also give rise to an ulcus serpens. Associated with the injury, and constituting the second factor in the production of ulcus serpens, is the presence of a chronic lesion of the conjunctiva (catarrh or trachoma), or an infection of the lacrimal sac (present in about one third of the cases of serpent ulcer), by which the infecting secretion is furnished. It is only in rare cases that the infection is brought from without, e.g., by a foreign body.

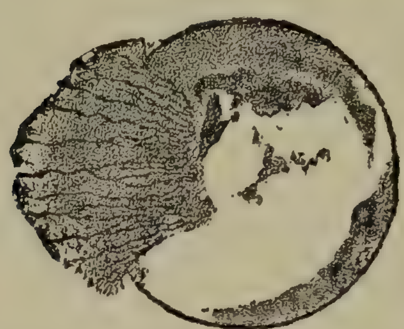

FIG. 241.—KERATITIS MARGINALIS SUPERFICIALIS.
At the inner margin of the cornea is a pseudo-pterygium.

Typical ulcus serpens attack adults exclusively, and especially those belonging to the working class. These are more frequently exposed to injuries of all kinds, and, besides, more often suffer from neglected affections of the conjunctiva and lacrimal sac than do members of the well-to-do classes. Great heat favors the formation of ulcus serpens, which is hence much more frequent in the hot season than in winter. For this reason reapers are not infrequently affected with the disease, since in cutting the grain they scratch their eyes with its awns, and, besides, do their work during the hottest days of the year. Stone masons and coal miners also are particularly apt to be attacked by ulcus serpens.

Pneumococcus ulcers, running a malignant course occur though rarely, in children. They do not, however, have the typical appearance of ulcus serpens, for they form large infiltrates which often are so greatly distended as to project above the surface of the cornea. They are uniformly gray or are yellow like pus throughout, and rapidly break down.

Ulcus serpens also occurs in *acute infectious diseases*, such as smallpox, scarlet fever, measles, typhus, etc. The form that results from *smallpox* is most frequently observed. In this case it makes its appearance not at the height of the disease but in the stage of desiccation, and, in fact, sometimes even in patients who have already left their beds. These variolous ulcers are found in children as well as in adults, and not infrequently affect both eyes so that total blindness may be produced by them.

At the time when smallpox was very widespread it formed one of the most frequent causes of blindness, so that about one third of all cases of blindness were produced by it. Since smallpox, owing to the introduction of vaccination, has become less prevalent, the blindness due to it has correspondingly diminished. Thus, in France, before the introduction of vaccination, 35 per cent—and after its introduction 7 per cent—of all the blind lost their eyesight by reason of smallpox (Carron du Villards). In Prussia, before the introduction of compulsory vaccination, 35 per cent—after its introduction 2 per cent—of all the blind people in the country were rendered so by smallpox.

Since the ulcus serpens in variola develops such a length of time after the stage of eruption, it obviously cannot be regarded as a smallpox pustule that has been localized upon the cornea. Such pustules do occur, but only in the conjunctiva. It is true they may then, if they lie near the margin of the cornea, give rise to a suppurative infiltration of the adjoining portions of the latter, but not to the typical picture of the centrally situated ulcus serpens. When the latter develops in the stage of desiccation of variola, it must be attributed, like a traumatic ulcus serpens, to an infection of the cornea from without. There is no lack of opportunity for such infection to take place, since the free border of the lids is a favorite seat for variolous pustules, which thus can come into direct contact with the cornea. The infection could not be due to the germ causing smallpox, for it occurs at a time when immunity against smallpox has already been effected. True variola of the cornea can be produced in animals (Paul), but the changes in this case affect the epithelium only.

In typical ulcus serpens the pneumococcus is found so extremely often (Uhthoff and Axenfeld) that ulcus serpens has been called by the name of pneumococcus ulcer of the cornea. Exceptionally, it is true, other bacteria such as the diplobacillus described by Petit, the streptococcus, the bacillus pyocyaneus, the actinomyces (Elschnig) have been found. But the clinical picture of these cases differs in character and course from typical ulcus serpens.

551. Symptoms and Course.—A non-infected erosion of the cornea remains clear and heals in a day or two without leavng a trace. An infected erosion remains unhealed and its floor becomes clouded and gray. Already by the end of three or four days one can appreciate that an ulcus serpens is imminent from the intensified cloudiness of the rim surrounding the grayish area, which itself has begun to assume a disc-like shape. This initial stage of the disease, however, is very rarely seen, because generally no attention is paid to the condition until it has lasted a week or more. By this time, then, the infiltrate appears as a grayish-white or yellowish disc, which occupies nearly the centre of the cornea. The opacity of the disc is greater at its edges than in the centre and generally the edges them-

selves show a particularly well-marked gray or yellow opacity in one special direction. The disc is surrounded by a delicate gray area, and frequently fine, radiating, gray striæ extend from the margin of the disc into the transparent part of the cornea. The surface of the cornea over the disc is dotted and often at the beginning is raised some distance above the level of the surrounding parts. Soon, however, this spot is seen to be depressed although not with abruptly depressed edges, as in the case of an ulcer, but rather under the form of a shallow dimpling. Moreover, the rest of the cornea that is not occupied by the serpent ulcer proper is less lustrous, being covered with a delicate uniform cloudiness. These changes are always associated with a violent iritis. The aqueous is turbid, the iris is discolored and is fastened by posterior synechiæ to the lens capsule, the pupil is closed by exudate. Uniformly a hypopyon is present, forming one of the characteristic marks of the disease. Corresponding to the severity of the inflammation is the violence of the irritative symptoms: slight œdema of the lids, intense injection of the conjunctival and ciliary vessels, photophobia, and pain, which latter often reach a very considerable height. Nevertheless there are also torpid cases, which are associated with very slight symptoms of irritation.

The subsequent *course* consists in the enlargement, both superficially and in depth, of the serpent ulcer. The superficial enlargement takes place chiefly in that direction in which the margin is marked by a specially dense opacity—an opacity which not infrequently looks like a yellow crescent placed upon the serpent ulcer. Inasmuch as the anterior lamellæ of the cornea within the region occupied by the ulcus serpens keep constantly breaking down, there is formed an extensive ulcer, the floor of which is coated with pus. Ultimately this may spread till it reaches the limbus.

While the ulcus serpens is thus progressing the accompanying iritis keeps on increasing pari passu, and the hypopyon keeps growing until it fills the greater part of the anterior chamber.

The *hypopyon* at first consists simply of pus and has a horizontal upper border which however, does not change its place on movement of the head, because it is held in place by fibrinous exudation. As the hypopyon increases, its upper border no longer remains horizontal, but becomes convex upward. Furthermore, the portion in contact with the cornea is higher than that in contact with the iris, so that the hypopyon appears like a stream of pus flowing down from the centre of the ulcer (cf. Fig. 245, *d*). This appearance the older writers called *onyx* (unguis) from its resemblance to the lunule of a finger nail and thought it due to a settling of a layer of pus in the lamellæ of the cornea itself. This, however, does not occur. The purulent masses always lie in the anterior chamber, but now are no longer pure pus but a quite consistent fibrino-purulent exudate. We see this best when we open the anterior chamber. The exudate then does not flow out, but must be picked out in clumps with a forceps. These clumps are adherent to the posterior surface of the cornea at the centre of the ulcer, and this gives a high hypopyon its peculiar shape. Owing to the similarity in color it is often difficult to distinguish between the hypopyon and the infiltration of the margin of the ulcer.

If the hypopyon is absorbed or is removed, we often observe in the region occupied by the middle of the ulcer a mass of pus, the color of which, intensely yellow in the centre, fades off toward the periphery of the ulcer.

This mass cannot be removed by paracentesis. It lies, in fact, in the cornea itself (§ 554) and constitutes the so-called *posterior abscess.*

552. Perforation.—The posterior abscess is the precursor of perforation. The pus-colored spot protrudes more and more, until all of a sudden the anterior chamber is abolished, the hypopyon disappears, and if the perforation is not right in the centre of the cornea, the discolored iris prolapses. In ulcus serpens, perforation is not therefore preceded by a keratocele (cf. § 554). Furthermore, the perforation and also the prolapse of the iris are small, as a rule. Only in particularly bad cases do we get a total prolapse of the iris.

Often enough the perforation opening is so situated that the iris cannot prolapse. Nevertheless, anterior synechiæ are formed since the iris everywhere is covered by exudate, and hence adheres to the surface of the cornea, as soon as the anterior chamber is abolished.

In many cases the perforation starts the ulcer on the road to healing; in other cases the ulcer progresses in spite of the perforation and destroys the whole cornea; and it may even happen that the interior of the eye becomes infected so that panophthalmitis (§ 755) is produced.

Sometimes as a result of treatment—more rarely without this—an ulcus serpens heals *without perforating.* In that case the marginal infiltration disappears, the hypopyon becomes less and soon vanishes altogether, the edges of the ulcer become rounded, its floor regains its lustre, but remains for a long time somewhat depressed, since the defect is filled only very gradually with cicatricial tissue. New vessels may form in the cornea, both superficial and deep, but these may be but sparsely present or be altogether absent.

553. Results and Prognosis.—Even in those cases which heal without the formation of anterior synechiæ and with simple deposition of cicatricial tissue, the sight is considerably affected because of the central site of the cicatrix and the fact that even if it is thin and allows the light to pass, it yet forms a facet, producing marked astigmatism. If the ulcer has gone deep and especially if it has perforated, the scar left is dense and white. If anterior synechiæ have formed, secondary glaucoma often develops. Posterior synechiæ are almost always left and in bad cases also occlusio pupillæ and often cataract. In the worst cases the eye is made completely blind through staphyloma of the cornea or by atrophy of the eyeball.

The prognosis is doubtful as long as any part of the ulcer is still in the progressive stage. Even in the most favorable cases the sight is considerably affected, although it may be helped by operation. Nor in stating the prognosis must we forget the possibility that a secondary glaucoma may develop later.

554. Morbid Anatomy.—Leber has shown how suppurative keratitis is produced. Trauma alone cannot do it. Such a condition is excited by the toxins produced by germs. These toxins exert upon the cell protoplasm an irritant action, when but slightly concentrated, and, when more concentrated, a paralyzing and ultimately fatal effect. When pus cocci are introduced into the cornea by inoculation they first increase in number within the corneal tissue. Then the cornea for a certain distance about the colony of

cocci dies, because the toxic substances excreted by the cocci are present within this are in a state of strong concentration. Accordingly, the colony of cocci now lies in th centre of the necrotic area (Fig. 242). In the meantime violent inflammatory symptom have made their appearance in the eye. The toxic substances by diffusion have reache the margin of the cornea, and there cause dilatation of the vessels and increased per meability of the vessel walls, entailing as a necessary consequence increased diapedesis o the blood-plasma. In addition to this diapedesis of serum an emigration of white blood corpuscles also takes place from the vessels. This is effected by active movements of th leucocytes, which, irritated by the toxic substances, emigrate toward the focus of inflam mation (*chemotaxis*). The leucocytes, however, do not make their way into the necroti district itself, the pus cells that are found there being such as have emigrated from th conjunctival sac. In fact, the leucocytes derived from the margin of the cornea ar paralyzed at the border of the necrotic area owing to the great degree of concentration o the toxic substances at this spot. Thus it happens that a constantly increasing numbe of cells are arrested at the margin of the necrotic spo and die there. In this way is produced the *infiltratio* (or *migration*) *ring*, which is apparent to the nake eye. Now leucocytes have the property of dissolvin by a kind of digestive action, tissues in which they ar present in large quantities. They effect by this mean the exfoliation of the necrotic area, and give rise to *delimiting suppuration*. The inflammatory phenomen in the cornea, consequently, appear under the guise o a process having a definite purpose to subserve, th principal end and object of which are to eliminate th necrotic area, and with it the morbific agents that i contains. But besides this the pus corpuscles, a experiment has shown, have the additional property o directly inhibiting the growth of germs, so that the oppose the diffusion of those organisms that may hav chanced to grow out beyond the necrotic mass.

Fig. 242.—Inoculation Keratitis. (After Leber.) Magnified 3 ×1.

Surface section through a rabbit's cornea into the centre of which a dilute suspension of staphylococcus aureus has been injected three days before. In the middle of the cornea is seen the mass of proliferating cocci surrounded by a necrotic zone. This latter is bordered by a broad migration ring, adjoining which below there is a second one, narrower and not complete.

A like migration takes place on the posterior surface of the cornea also. First the endothelium o Descemet's membrane over the necrotic area become detached and a clot of fibrin is precipitated from th aqueous upon this portion of the posterior wall of th cornea. Then leucocytes migrate into the clot, s that soon a plug of pus can be seen on the posterior surface of the cornea at the site o the inoculation.

The experiments by which these processes have been demonstrated were made on animals. Ulcus serpens in man follows exactly the same course. The changes occurring in the first stages of the condition are shown in Fig. 243, taken from one of Fuchs' specimens. *Older cases* (Fig. 244) show a flat loss of substance extending down to the middle layers of the cornea and having walls rising steeply and projecting like an embankment. The projection here is caused by an *infiltration* which extends some distance in the middle layers of the cornea at the level of the floor of the ulcer. At the margin of the ulcer, this infiltration is very intense; along the floor it is very much less marked and may be absent altogether. In the latter case the floor is formed of swollen non-nucleated corneal lamellæ, which sometimes quite fill up the ulcer. Moreover, the deep layers of the cornea, whereever they underlie the ulcer, have no cell nuclei and must therefore be regarded as necrotic.

If, as the disease goes on, the infiltration disappears, the epithelium at once regenerates (Fig. 245, *b*) and grows over the floor of the ulcer. But as the infiltration may recur, we often find in an ulcus serpens the signs of an advance and a regression combined.

The *exudation in the anterior chamber* arises from the vessels of the uvea, for which reason it often contains pigment granules. It consists of pus corpuscles and a framework of fibrin. At first it collects in the angle between cornea and iris at the bottom of

the anterior chamber (hypopyon, Fig. 245, *d*). The upper surface of the hypopyon is not absolutely horizontal, but slants up to the posterior surface of the cornea, and is continuous with an extremely thin layer of pus corpuscles, which coats this whole posterior surface and which is one of the causes of the faint cloudiness, present outside of the ulcer proper.

Right opposite the centre of the ulcer on the posterior surface of the cornea is a rather large mass of pus (*c*, Fig. 245) agglomerated into balls by fibrin, and generally united with the hypopyon. This exudation erodes Descemet's membrane, splits off portions of it, and finally perforates it. Then these masses of pus enter through the opening, and form an infiltrate of more or less lenticular shape in the posterior layers of the cornea. This is the *posterior abscess*. This then is connected with the hypopyon but is separated from the marginal infiltration by the necrotic mass forming the floor of

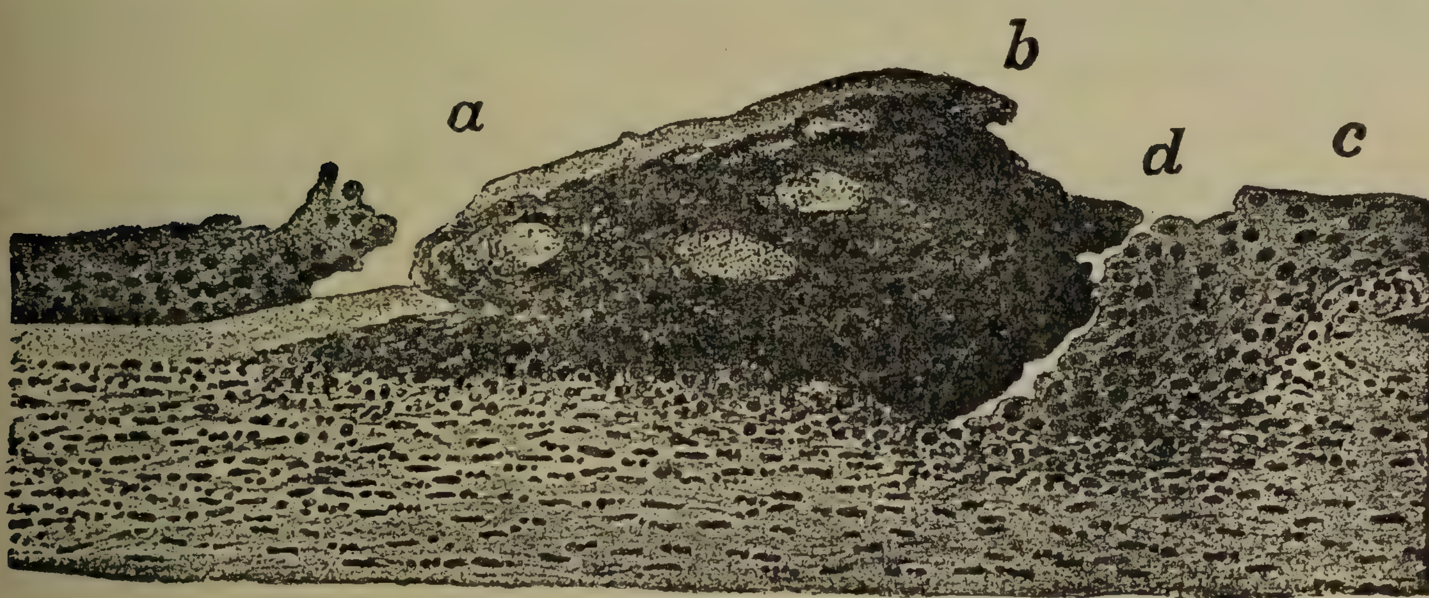

FIG. 243.—RECENT ULCUS SERPENS. Magnified 355 × 1.

An ulcus serpens developed from an erosion, the latter showing a faint gray cloudiness and, on the 3rd to the 4th day after it had started, getting a more pronounced gray border. Since such an early stage of ulcus serpens could not otherwise be had for anatomical examination, Fuchs inoculated the cornea of an eye that was destined for enucleation with a culture of pneumococci which had been made from a case of ulcus serpens. The eye was enucleated 3 days later; at the site of inoculation there was a gray infiltrate which rose somewhat above the level of the adjacent cornea and which had a more pronounced gray border like a recent ulcer. The cross section shows a plug consisting of the swollen uppermost layers of the cornea. In this lie only a few poorly stained (necrotic) pus corpuscles, and great quantities of pneumococci, which in particular quite fill up the large gaps visible in the plug. Bowman's membrane over the plug is also necrotic. At *a* the swollen masses push through the membrane, out upon the anterior surface of the latter, while the sharp edge of the plug juts forward like a wedge beneath the membrane. On the other side, Bowman's membrane is wanting between *b* and *c*. Through this gap the epithelium *d* grows down into the deeper parts, and separates the necrotic plug sharply from the living corneal tissue. The lamellæ of the cornea at the lower side of the plug are infiltrated with pus corpuscles whose number increases toward the border *a*. At this spot both in the necrotic border itself, and in the surrounding infiltrated lamellæ of the cornea, still living pneumococci are present. Hence at this side an extension of the suppuration would be expected to take place.

the ulcer. But as these necrotic layers of the parenchyma keep softening and melting away, perforation of the cornea finally takes place. Hence in ulcus serpens Descemet's membrane is perforated before the corneal stroma.

The *pneumococci* are present in the infiltrated wall of the ulcer, but not in the hypopyon nor in the posterior abscess.

555. Treatment—In consideration of the rapid progress which an ulcus serpens usually makes, and which threatens the entire cornea with destruction, a particularly prompt and energetic interference is required.

The first thing to do is to *remove the lacrimal sac*, in case it is diseased; for, as long as this supplies a source of infection, other methods of treatment must be illusory.

The next thing in order is to disinfect the conjunctival sac as thoroughly as possible. For this purpose we may use with advantage a one-

per-cent *collargol* ointment. With this we should apply the remedies ordinarily used for corneal ulcers, viz. *atropine*, a *bandage*, and *hot moist compresses*. But as soon as the margin of the ulcer shows a purulent infiltration, we must proceed without delay to attack the corneal infection directly. The following remedies may be employed.

1. *Cauterization* with thermo-cautery or galvano-cautery. This is applied to the infiltrated edge of the ulcer including a little of the sound cornea adjoining. If the hypopyon has risen to the centre of the ulcer or above or if there are clumps of pus lying in the anterior chamber back of the ulcer, so that there is danger of an early perforation of Descemet's membrane, it is well to perforate the cornea by applying the cautery point to the floor of the ulcer and evacuate the anterior chamber; if, however, the hypopyon is not of great amount, we refrain from doing this, as it will disappear spontaneously if the progress of the ulcer is arrested.

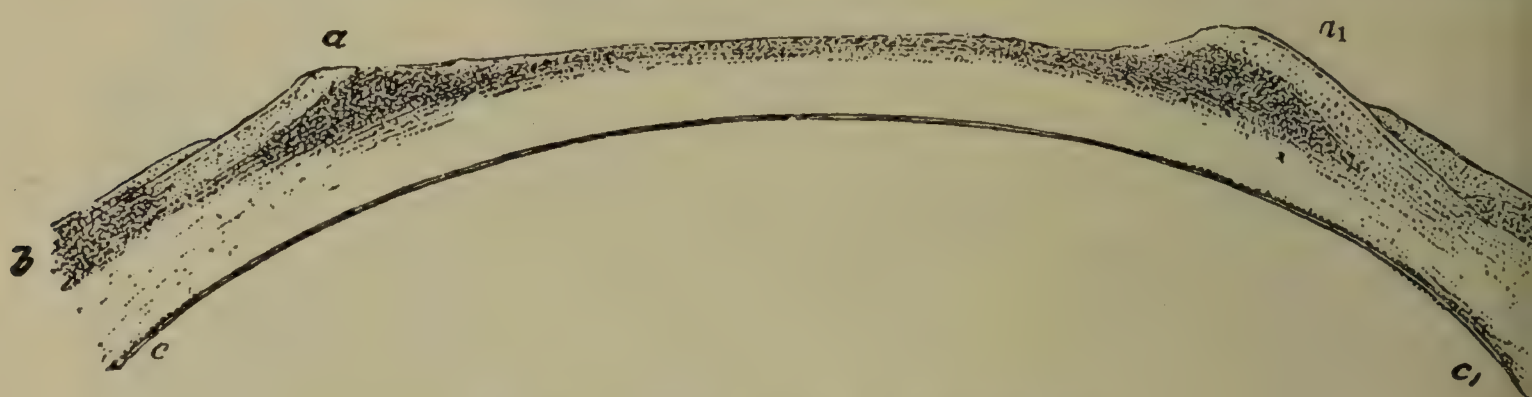

FIG. 244.—ULCUS SERPENS. Magnified 10×1.

The ulcer is large, is still covered with pus in every part, and has a yellow border a a_1, surrounding it on all sides. This border is elevated because the anterior lamellæ of the cornea are raised by the subjacent infiltration. Layers that are infiltrated with pus form the floor of the ulcer. The layers that succeed these deeper down show, to be sure, no infiltration, but neither do they show any living corneal corpuscles, so that the cornea here is necrotic in its entire thickness. At the margin of the cornea there is marked infiltration beneath the limbus, b b_1, and under this lie living corneal lamellæ presenting a moderate infiltration which only towards the most posterior layers c c_1, becomes more marked. A posterior abscess is wanting in this case.

2. The application of *intense heat* directly to the cornea (by steam, hot air, chauffage, or the thermophore, see § 118). With these applications the destruction of tissue is better limited to the area involved than by the cautery. Birch-Hirschfeld has obtained good results by the application of *light* of a short wave length (light passed through a quartz lens with a uviol filter).

3. Cauterization with *zinc sulpnate* (Éperon). A cotton-tipped probe dipped in 20-per-cent solution of the sulphate is applied to and underneath the edge of the ulcer. If the solution reaches the sound cornea it does no harm as it affects only the parts not protected by epithelium. The solution should be boiled before using. Zinc sulphate has also been successfully applied by means of iontophoresis.

4. *Serotherapy*. About 50 cc. of Romer's antipneumococcic serum are injected beneath the skin of the abdomen and some non-carbolated serum is dropped on the ulcer. If the ulcer is in the progressive stage a local reaction is produced, both infiltration and hypopyon increasing. If the treatment is successful the infiltration begins to subside in 24 to 48 hours after the injection. In this case the latter may be repeated in two or three days.

But if no improvement is produced by the first injection, cauterization should be performed by one of the methods just given.

5. *Optochin.* A one-per-cent solution of optochin hydrochloride is applied to the ulcer and afterward a ½-per-cent solution is dropped into the eyes every 2 hours.

6. In extensive ulcers, especially if the other methods fail, the cornea should be split (*Saemisch's* or rather *Guthrie's* incision; see § 941). This is done not for the purpose of removing the hypopyon but to secure the advantages of perforation (see §§ 535, 552). It has the disadvantage that in consequence of it extensive adhesions form between the iris and cornea, which may produce secondary glaucoma and staphyloma of the cornea.

In view of the virulence of ulcus serpens it is important that the general practitioner should be informed of the necessity of putting even the seemingly mildest cases at once under the treatment of a specialist. He may at the same time as a preventive measure apply the zinc sulphate solution, as above prescribed, since it is possible for even a tyro to do this without damaging the eye.

Fig. 245

Fig. 245.—Ulcus Serpens. A, front view. B, vertical cross section. Magnified 2.5 ×1.—C, longitudinal cross section. Magnified 20 ×1. In this, as in most of the cases of ulcus serpens that have been examined anatomically, the eye was affected with absolute glaucoma. The ulcer occupied about the centre of the cornea; its upper, advancing border, *a*, was clearly recognizable as a yellow crescent, while the lower border, *b*, being but slightly opaque, did not specially stand out against the subjacent masses of pus which lay in the anterior chamber and extended as low down as *c*. Farther below, between the cornea and the iris, is seen the hypopyon, the upper border of which is convex (A, *d*). Owing to the glaucoma, the iris has, rather peculiarly, become adherent by its periphery to the cornea. *e*, posterior abscess in the cornea.

Prophylaxis of ulcus serpens is possible only in the sense that one removes all infectious conditions of the conjunctiva and lacrimal sac which may give rise to it. Particularly important in this regard is excision of the

sac in a case of persistent dacryocystitis (§ 909). In smallpox also timely prophylaxis may prevent great damage.

During an eruption of smallpox the lids are much swollen, and hence are not opened by the patient, and even the physician generally neglects to look at the eye from time to time. In that case, when the swelling of the lids goes down during the stage of desiccation and the patient opens his eyes again, the morbid process in the cornea is often already in progress, and we are just so much behindhand in undertaking the treatment. Horner, therefore, is right in demanding that a physician treating a smallpox patient should prevent the agglutination of the lids by applying a pledget smeared with ointment, should examine the eyes every day, and should cleanse the conjunctival sac with antiseptic solutions. Careful watching will enable us to recognize the very commencement of the corneal disease, which in these early stages presents the most favorable conditions for treatment.

556. Keratomycosis.—This variety of keratitis produced by mold fungi (Fig. 246) presents even upon external examination a clinical picture differing from that of the ordinary ulcus serpens. There forms in the central portion of the cornea an infiltrate which later undergoes superficial disintegration, and is distingushed by its peculiar, dry, crumbly surface. About this area a gray or yellow annular line of demarcation forms, which gradually deepens into a gutter and ultimately leads to the exfoliation of the inclosed portion of cornea, which in the meantime has become necrotic. The latter being thus detached en masse from the cornea, cicatrization of the resultant loss of substance ensues. Hypopyon is present, but the irritative symptoms are slight, and the whole course is usually very chronic. Atypical forms are observed, including cases which resemble a fascicular keratitis (Axenfeld). The prognosis is better than in ulcus serpens, although like the latter the condition leaves opacities of the cornea. Examination of the sequestrum shows it to be permeated by the mycelium of the aspergillus fumigatus (occasionally by other mold fungi). It is probable that, as a general thing, this fungus is carried into the cornea by the foreign body that caused the original injury. Treatment consists in the removal of the sequestrum containing the fungi, and subsequent cauterization.

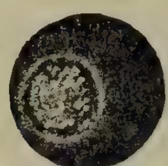

Fig. 246.—Keratomycosis Aspergillina. Natural size.

The prickly fruit of a horse chestnut had fallen into the eye of the patient, a woman thirty-five years of age, fourteen days before. The fungus mass is surrounded by a ring of demarcation but is still solidly connected with the underlying parts. Three small colonies of fungi have grown almost up to the ring of demarcation.

557. Keratitis Disciformis (Fuchs).—This is a keratitis that is allied to ulcus serpens and consists in the development in the middle layers of the cornea of a gray, disc-shaped opacity. In the centre of the disc a small more deeply clouded speck is commonly observed (Fig. 247). The periphery of the disc is sharply delimited by a border of deeper gray, which in many cases is made up of concentric lines (Fig. 248), or may be surrounded by one or more rings representing the residua of a preceding larger opacity (Kraupa). This disc-shaped infiltrate never becomes yellow nor leads to disintegration of the cornea; only exceptionally a small loss of substance develops over a circumscribed area. The irritative symptoms are marked only at the beginning of the disease; later they may be absent altogether. Hypopyon is absent or scanty. The course is protracted, as it takes one or more months for the eye to become free from congestion and for the infiltrate to be transformed into a corneal opacity, which is permanent. In the course of the disease scattered, superficial or deep-seated blood-vessels often develop and extend into the infiltrate. The cause of the disease, just as in ulcus serpens, is an infection of the cornea from without, the central grayer speck representing the point of entry of the bacteria. The epithelial lesion that precedes the infection may often be attributed to a slight injury, especially a contusion of the cornea by a missile, and in some cases to an antecedent herpes corneæ febrilis; but in very many cases the cause remains unknown.

Schirmer has observed a typical keratitis disciformis when the cornea had been infected by vaccine virus (*keratitis post-vaccinolosa*). This occurs most frequently in

persons who in nursing children that have been recently vaccinated have acquired a vaccine blepharitis (§ 420), from which the cornea has become infected. These cases represent, we may say, a milder form of the ulcus serpens of variola and are instructive in two regards. They show that without doubt the ulcus serpens of variola does not originate, as was formerly supposed, in a metastatic way, but by infection from without, probably from the lid margin. They further prove that keratitis disciformis is closely related to ulcus serpens; is, we may say, a milder form of the latter, in which the inflammation does not go on to suppuration—a fact which probably depends on the slighter virulence of the micro-organisms that have entered the cornea.

Older authors, properly recognizing the relationship of keratitis disciformis to ulcus serpens (which they called abscessus corneæ), gave the former the name of *abscessus siccus*, to denote an abscess in which suppuration does not develop.

Keratitis disciformis has a certain resemblance to keratitis profunda (§ 581), in which also a deep-seated gray, non-ulcerating cloudiness develops in the centre of the cornea. But this is composed of gray striæ and specks and merges gradually into the transparent cornea. In keratitis disciformis, on the contrary, the opacity appears uniformly gray and only on strong magnification is resolved into very minute clear-cut, white, closely packed dots; moreover, it is very sharply demarcated from the healthy cornea by the gray circular line along its border. Keratitis disciformis should not be confounded with keratitis centralis annularis (see § 575). From a macula of the cornea it is distinguished by the fact that the surface of the cornea is stippled, instead of being smooth and lustrous.

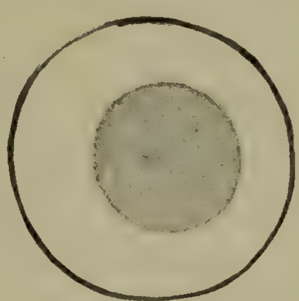

Fig. 247.

Fig. 248.

Fig. 247.—Keratitis Disciformis. Due to an erosion of the cornea.

Fig. 248.—Keratitis Disciformis. Due to herpes febrilis corneæ. The two maculæ at the inner lower margin of the cornea are on the site of a herpes eruption.

The opacity does not tend to spread much, and perforation does not occur, but the disease lasts a long time and causes great damage to sight from the permanent opacity left in the centre of the cornea.

Treatment is symptomatic. If there are no irritative symptoms, dionin instillations and massage with the yellow oxide ointment may be used alternately.

V. Keratitis Associated with Dryness of the Eye

558. Keratitis e Lagophthalmo.—Keratitis e lagophthalmo originates in a defective covering of the cornea by the lids. The conjunctiva of the eyeball, wherever it lies constantly exposed to the air in the open palpebral fissure, appears reddened, and generally somewhat swollen as well. It secretes a small amount of discharge, drying upon the conjunctiva in crusts which not infrequently also cover the exposed portion of the cornea. The latter is dry on the surface, dull, slightly depressed, and at the same time clouded and gray. In the subsequent course of the disease the cloudiness becomes more and more intense, until finally disintegration of the superficial layers of the cornea takes place, with the consequent formation of an ulcer (Fig. 249). At the same time there is iritis with hypopyon. The ulcer may heal without perforation, but leave an opacity behind it, or it may perforate the cornea, and thus lead to prolapse of the iris, or even to panophthalmitis.

The *cause* of keratitis e lagophthalmo is the desiccation of the cornea in consequence of the defective closure of the lids (lagophthalmus). Owing to this desiccation the corneal epithelium becomes fissured and desquamates

in spots. Then germs migrate into the corneal lamellæ thus exposed, and produce suppuration (Fig. 250). As the lowermost portion of the cornea is the part usually uncovered (§ 442) that is also the part that undergoes dessication; but in certain circumstances other parts may be affected.

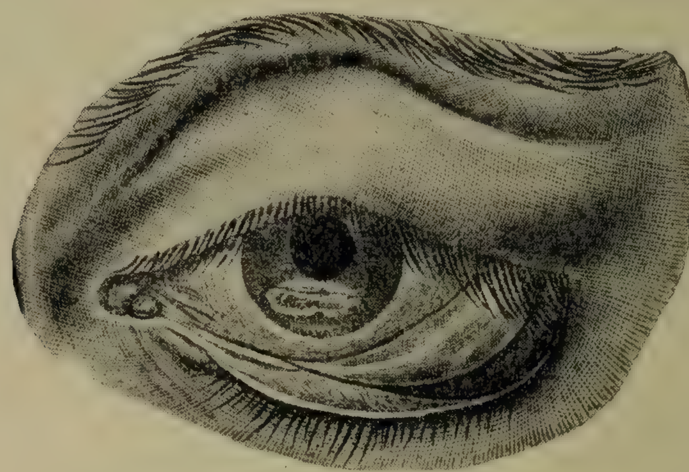

FIG. 249.—KERATITIS E LAGOPHTHALMO.

As a result of a healed caries of the upper and lower margins of the orbit, lagophthalmus had developed with ectropion of the lower lid. The skin of the upper lid, below the middle portion of the eyebrow, is drawn up toward the upper margin of the orbit and is attached to the latter by a scar one cm. long. As a result of this the free border of the lid is drawn up at its central portion, but, what is of most importance, the lid is prevented from going down, when the attempt is made to close the eyes. There is a second scar at the outer end of the lower margin of the orbit. This, indeed, is concealed by the margin of the lid, but by palpation through the lid it can be made out that there is an irregular notching of the otherwise sharp and smooth orbital margin. At the same spot, the border of the lid is attached to the bone and in such a way that the entire lid is drawn outward and downward and everted, particularly in its outer half, so that here the conjunctiva of the tarsus, being exposed to the air, is particularly swollen and reddened, and consequently appears dark in the drawing. The lower lid, being fastened down, cannot be raised when the attempt is made to close the eyes. Hence, even in sleep the lower part of the cornea between the two lids remains uncovered. This part of the cornea presents an oblong ulcer with yellowish-white border and an excavated and hence somewhat darker centre. Above and below, an areolate gray opacity adjoins the yellow margin of the ulcer.

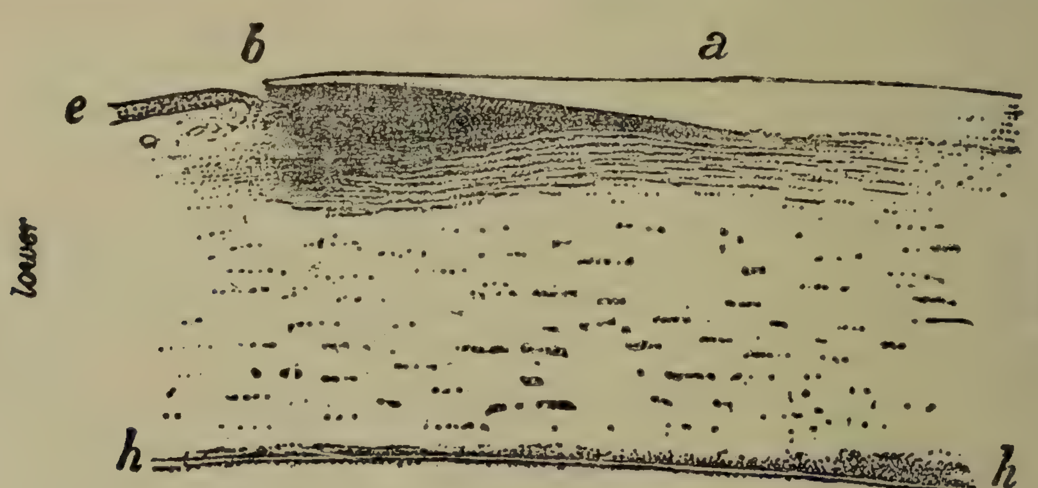

FIG. 250.—KERATITIS E LAGOPHTHALMO.

Vertical section through the lower part of the cornea. The epithelium, *e*, has been retained only in the neighborhood of the limbus, but over the dried portion of the cornea, *a*, it has been exfoliated. This portion of the cornea is necrotic, and no longer displays any stained cell-nuclei. Under it there is an infiltration of the cornea with pus corpuscles. This infiltration increases rapidly toward the lower margin of the cornea; here, at *b*, it has already caused by liquefaction of the tissue a small loss of substance, and would later lead to elimination of the entire mass, *a*. Below *b*, a less marked infiltration extends to the lower margin of the cornea. This corresponds to the gray areola which adjoins the yellow border (Fig. 249). There is furthermore an infiltration of the cornea with pus cells on the posterior surface, *h,h*. This at present is inconsiderable, but in the subsequent course would increase just like the infiltration on the anterior surface, and by growing out to meet this, would co-operate with it in producing disintegration of the cornea.

The *prognosis* depends on the nature and duration of the lagophthalmus; the *treatment* consists in taking care that the cornea shall be covered by the lids. In this way the development of a keratitis is prevented by prophylaxis, or, if a keratitis already exists, the chief condition is afforded for its cure. We must accordingly initiate the proper treatment for the cure of

the lagophthalmus (see § 442), and in the meantime, until a cure has been accomplished, take pains to effect a perfect closure of the lids by means of a properly applied bandage. In slight cases of lagophthalmus it is sufficient to keep the eye bandaged through the night only. But if the lagophthalmus is considerable, or if keratitis has already set in, the eye must be kept bandaged all the time. If the treatment is initiated early, the prognosis is good, inasmuch as the process comes to a standstill as soon as soon as the desiccation of the cornea is arrested.

559. Keratomalacia.—Keratomalacia[7] occurs only in childhood. The disease begins with night blindness (hemeralopia). In very small children who do not go about alone yet, this symptom naturally cannot be made out. In such children, the first thing that strikes us is the dryness of the conjunctiva, which next develops, and which appears under the form of triangular xerotic spots on both sides of the cornea (see § 507). The conjunctiva in these spots is covered with a fine white substance like foam, and, as the lacrimal fluid cannot moisten it, looks as if smeared with grease. The dryness extends rapidly over the rest of the conjunctiva and also over the cornea. The latter becomes dull, insensitive, and uniformly cloudy. Soon the cloudiness in the centre of the cornea increases, a gray infiltrate forming there. This spreads rapidly, takes on the yellow color of pus, and terminates in the disintegration of the cornea—a disintegration which in bad cases, may take place within a few hours. In the beginning the affected eye is not discolored; afterward, when the cornea is already greatly involved, there appears about the latter a dusky venous injection. The lacrimal secretion is not increased, but rather diminished; moreover, other symptoms of irritation, like photophobia and blepharospasm, are slight or are wanting altogether. The striking contrast between the severity of the corneal affection and the insignificance of the accompanying symptoms of irritation, together with the dryness of the eye, stamps the disease with quite a peculiar character. This affection usually attacks both eyes.

Children suffering from keratomalacia show a disturbance of the general condition, which is generally pronounced even before the outbreak of the eye trouble, and which afterward grows still greater. They become strikingly apathetic, have diarrhœa alternating with constipation, become rapidly emaciated, and often ultimately die either from exhaustion or from a complicating bronchitis or pneumonia.

The *prognosis* in very small children is bad, as in most cases they lose not only their eyes, but their lives as well. In somewhat older children the disease runs a less severe course, so that they escape with their lives and get off with smaller or larger cicatrices of the cornea.

Keratomalacia is *caused* by insufficient nutrition of the cornea. From this there results a necrosis either of the epithelium alone or of the corneal lamellæ themselves, and following this in turn is an entrance of bacteria from without and hence suppuration. The reduction in the nutrition of the cornea is only one of the symptoms of a serious disturbance of general nutrition which also shows itself in the associated hemeralopia (see § 261).

[7] Softening of the cornea, from κέρας, horn, and μαλακός, soft.

Hence, keratomalacia develops, as a rule, in consequence of enfeebling influences affecting the children, and acting detrimentally upon their nutrition. Among these influences belong insufficient or unsuitable nourishment (rearing of children by hand), severe diseases like scarlet fever, measles, typhus, etc., and particularly hereditary syphilis. In Russia the disease used to occur frequently during and after the time of the great fast, during which the mothers lost their milk in consequence of fasting. For a similar reason it was frequently observed in Brazil among the badly nourished children of the negro slaves. In Denmark in recent years it has increased considerably, because, owing to the centralization of the dairy industry, the children have been restricted to buttermilk (Rönne). In exceptional cases, children previously healthy from some unknown cause are attacked by keratomalacia, to which ensues a rapid decline of strength with a fatal issue.

In adults true keratomalacia does not occur, but probably an analogous affection are the *marantic ulcers* of the cornea which occur in persons greatly debilitated by grave liver disease (cirrhosis, carcinoma). These ulcers run a torpid course, but may produce destruction of the cornea. They have no characteristic situation or form, but sometimes look like ulcus serpens, sometimes like infiltrated marginal ulcers. They are associated with hemeralopia and xerosis and offer a bad prognosis (even as regards life). Ordinary hemeralopia with xerosis (§ 261) seems to be only a lighter form of the same disease.

The chief task that *treatment* has to accomplish is to support the child's strength by means of fitting nourishment. In addition we must try to stimulate the vitality of the tissue of the cornea, a thing best performed by means of moist warm compresses placed upon the eyes. If the apathetic little patients do not close their lids properly, the corneæ must be protected from desiccation by bandaging the eye.

Young rats fed on food devoid of fat-soluble vitamines die, the cornea in the meantime becoming first xerotic and then undergoing suppuration. The more mature the animal, the less likely is this result to occur. It seems not unlikely that the keratomalacia of children depends on a similar dietetic error and that the administration of fat-soluble vitamines, particularly cod-liver oil is therefore indicated.

560. Keratitis Neuroparalytica.—In this affection, which occurs in consequence of paralysis of the trigeminus, the cornea becomes dull and slightly cloudy. Then, beginning in the centre of the cornea, a depression shows itself in the epithelium as if the latter in that spot had been exfoliated. This depression spreads more and more peripherally until finally the only portion of epithelium that is left of normal thickness, is a strip 2 to 3 mm. broad at the margin of the cornea. This gives the cornea quite a peculiar appearance, such as is found in no other disease of it. In the meantime the cloudiness of the cornea also has increased. This is most marked in the centre and there is uniformly gray; toward the margin it gradually decreases and may be resolved by the magnifying glass into separate maculæ. Subsequently the cloudiness becomes yellowish, hypopyon sets in, and ultimately the cornea in its centre breaks down into pus. Thus a large ulcer

forms, which cicatrizes with inclusion of the iris, and generally with flattening of the entire cornea. Not all cases, however, run so severe a course; the keratitis may get well without the occurrence of any purulent disintegration of the cornea, although there nearly always remains an opacity of considerable dimensions, and often, besides, a flattening of the cornea. Lighter cases, however, are seen in which, with moderate protection, the cornea does not undergo considerable exfoliation or, even if it does, regains its epithelium rapidly without the formation of a lasting opacity (Chance, Theobald).

The course of the disease is slow, and is characterized by the slightness of the associated symptoms of irritation. There is, indeed, marked ciliary injection but no lacrimation, since the secretion of the lacrimal gland, due to reflex action, is diminished or abrogated. Owing to the coincident paralysis of the trigeminus, pain obviously is altogether absent.

Unless the cornea can be adequately protected, the *prognosis* is unfavorable, for, whether the formation of ulcers does or does not take place, the disease leads, almost without exception, to the production of a dense opacity over the entire cornea, and hence to an almost complete annihilation of the visual power.

The *treatment* consists in the use of hot moist compresses and atropine. To protect the insensitive cornea against injury it should be covered by a median tarsorrhaphy (§ 924), the lids being closed for weeks, months, or even years (de Schweinitz), and thereafter being protected by an appliance like the watch crystal bandage (Buller's shield—see § 464), which does not interfere with the movements of the lids, keeps the eye moist, and does not come into contact with the cornea. Besides, we may try electricity, or, following Nieden's recommendation, strychnine (3 to 5 mg. by hypodermic injection beneath the skin of the temple). If it is possible to remove the cause of the paralysis we do so.

The *cause* of neuroparalytic keratitis is a paralysis of the trigeminus. The lesion causing the paralysis may affect the nerve in any part of its course. Thus it has been repeatedly observed after injections of alcohol into the Gasserian ganglion or after removal of the latter or of the nerves emanating from it. Yet keratitis does not occur in all cases of trigeminal paralysis. For example, it is not found with herpes zoster, even though this is associated with anæsthesia over the whole district supplied by the first branch of the nerve.

The paralysis produces a disturbance of nutrition which makes itself first apparent in the corneal epithelium. If in a case of trigeminal paralysis in which the eye is still healthy, we hold the lids apart for one or two minutes with the fingers and thus prevent moistening of the cornea, small depressions, which soon enlarge and become confluent, appear in the epithelium. A healthy eye does not show these pits at all or does so only after a long time. These pits prove a diminished resistance of the corneal epithelium to desiccation and consequently a disturbance of its nutrition which was present before the desiccation began. Diminished moistening is, therefore, certainly more injurious for such eyes than for normal ones, and in fact we often actually see a genuine keratitis e lagophthalmo in cases of trigeminal paralysis when the eye has been exposed more than ordinarily to desiccation. For the same reason, too, bandaging of the eye is indicated and often has a good effect even in neuroparalytic keratitis at the outset. But, neuroparalytic keratitis may develop even when the cornea is kept permanently covered. Desiccation, therefore, is to be regarded only as a favoring factor, not as the final cause

of keratitis neuroparalytica. Such a cause is found in a disturbance of nutrition of the cornea (and particularly of its epithelium) produced by the loss of innervation. As a result of this disturbance of nutrition the resisting power of the cornea is so reduced that the latter is thrown into a diseased condition by external influences which are so slight that a normal eye would not have been injured by them. (Similarly in hemiplegia a bedsore sometimes develops with extraordinary rapidity on the side which is paralyzed, and is absent on that which is not, although it is exposed to the same pressure as the other.)

But neither a trophic disturbance, nor dessication, nor the injuries to which the cornea is exposed by its insensitiveness suffices to account for the purulent liquefaction of the cornea. This can only be the result of a secondary infection by pyogenic germs which find a ready avenue of entry in the epithelial defect produced by the paralysis.

561. The Three Forms of Dessication Keratitis Contrasted.—The three affections of the cornea just described—keratitis e lagophthalmo, keratitis neuroparalytica, and keratomalacia—have been frequently confounded with each other. Thus the keratitis e lagophthalmo, which makes its appearance in patients during the death agony, has been regarded as a keratitis neuroparalytica, i.e., as caused by the reduction of the nervous energy. Conversely, some have explained keratitis neuroparalytica and keratomalacia as produced by desiccation of the cornea, and in this way have placed them in the same category with keratitis e lagophthalmo.

The three diseases in fact have some common features. Among these are the dryness which the eyes exhibit, and also the insignificance of the irritative symptoms in comparison with the severity of the keratitis, an insignificance shown in the absence of increased lacrimal secretion, of blepharospasm, and often also of pain. And yet the *dryness of the eyes* in these three forms of keratitis is to be referred to very different causes.

(*a*) In keratitis e lagophthalmo an actual desiccation from evaporation exists. It affects only the exposed portion of the cornea, and may be relieved by closing the lids. The desiccation in this case is the one cause of all the subsequent changes.

(*b*) In keratomalacia the cornea is not actually dry, but only looks so, because the lacrimal fluid does not adhere to its surface. This dry appearance is present even when the eye is swimming in tears or when it is kept constantly closed; evidently, bandaging is of no effect against this sort of dryness. It is caused by the fatty character of the epithelial surface, which consequently is not wetted by the tears.

(*c*) In keratitis neuroparalytica there is neither real desiccation of the cornea, as in keratitis e lagophthalmo, nor a peculiar fatty condition of its surface, as in keratomalacia; on the contrary, the eye looks dry simply because, in spite of the marked inflammation of the cornea, the lacrimation, which we usually see under these circumstances in other cases, is absent. The secretion of the lacrimal gland is in fact diminished or altogether abrogated; nevertheless, the moistening of the eye is quite sufficient, as indeed it is after extirpation of the lacrimal gland.

The *absence of marked symptoms of irritation*, which characterizes these three varieties of keratitis, is accounted for in the keratitis e lagophthalmo of very ill patients and in keratomalacia by the general depression of the vital forces, and in keratitis neuroparalytica by the insensitiveness of the eye. The irritative symptoms, which in other cases are put in action through reflex impulses originating in the sensory nerves, are absent in the case of paralysis of the trigeminus.

The three forms of keratitis are hence, in spite of their external similarity, entirely different from each other, and can be readily differentiated by the clinical picture which they present. Keratitis e lagophthalmo occupies, as a rule, the lowermost part of the cornea. Keratomalacia begins in the centre of the cornea, and is found only in children who are the subjects of a rapid decline of nutrition. Finally, keratitis neuroparalytica is characterized above all by the rapid exfoliation of epithelium over the whole extent of the cornea, and does not occur except in conjunction with a trigeminal paralysis which can be diagnosticated at once.

VI. Deep Purulent Keratitis from Posterior Infection

562. Ring Abscess.—This usually occurs after perforating injuries of the cornea, and also after operations (especially cataract operations). It is ushered in with violent inflammatory symptoms. No matter where the corneal wound that gives rise to it is situated, the abscess develops a day or two after the injury in the central portions of the cornea as a yellow ring which is concentric with the limbus and is separated from it by a slightly cloudy marginal zone, 1 to 2 mm. broad. The ring itself has about the same width; the central portions of the cornea inclosed by it are again less cloudy and simply gray, not yellow. In the next few days, however, the yellow coloration spreads over the entire cornea; the latter disintegrates completely, and generally panophthalmitis ensues. Here accordingly we have to deal with an affection of the cornea of a peculiarly fulminating course, so that it is best to enucleate at once such an eye affected thus with annular abscess. Annular abscess may be caused by various bacteria; most often perhaps by the bacillus proteus fluorescens (Hanke) and the bacillus pyocyaneus.

Anatomical investigation shows that the primary cause of the abscess is a purulent endophthalmitis, originating in an ectogenous infection of the anterior segment of the eye (§ 98). By the toxic action of the germs an extensive necrosis is set up in the middle and posterior layers of the cornea. The yellow ring is simply the line of suppuration which demarcates the necrosis. That panophthalmitis is the inevitable outcome is inherent in the very nature of the infection

563. Keratitis Pustuliformis Profunda (Fuchs).—This rare condition, also known as ulcus internum (Bietti), posterior corneal ulcer (Klein), and deep central infiltrate of the cornea (Reis), occurs mainly in old people and chiefly in men. It is marked by the presence of yellow, puriform deposits, placed very deeply in the cornea. Sometimes a single, large, usually central deposit occurs; more often there are several, the size of a pin's head and variously disposed. The deposits are generally surrounded by a grayish, hazy, often punctate opacity. The condition is associated with a severe iritis, which, indeed, probably constitutes its real starting point. It is also accompanied by hypopyon, deposits on Descemet's membrane and, at times, vitreous opacity. There is great pain. The deposits never break down to form ulcers. Occasionally they undergo resolution, but generally they are transformed into deep and dense permanent opacities, and in bad cases flattening of the cornea or even atrophy of the eyeball may ensue. Usually but one eye is affected. In some cases syphilis seems to be the cause; in other cases the origin is unknown; but in any event the immediate cause must be a toxin, derived from the inflamed iris and acting on the cornea from behind. This is inferred mainly from the pathological changes in the cornea, which decrease in intensity from behind forward and which in the lightest cases consist in destruction of the posterior endothelium and proliferative inflammation of the hindmost layers of the cornea. In the severer cases, Descemet's membrane and the deeper layers of the cornea are also destroyed. There are marked changes in the iris and a massive exudate composed of polynuclear cells in the anterior chamber. The disease runs a very chronic course, and treatment, which must be aimed at relieving the symptoms, has very little effect.

The name *ulcus corneæ internum* has been given by E. von Hippel and others to an ulcer on the posterior surface of the cornea opening into the anterior chamber. Such a condition cannot be diagnosticated clinically, and, although it has been held that anatomical studies prove its existence in some cases, the evidence is not conclusive (Salzmann).

VII. Superficial Forms of Non-Ulcerative Keratitis

(*a*) *Conditions Predominantly Affecting the Epithelium*

564. Keratitis with Formation of Vesicles.—Vesicles on the cornea are generally small and multiple, and the anterior wall is very frail, being formed simply of the epithelium of the cornea, which is lifted up from Bowman's membrane by a limpid liquid. It is the exception for larger isolated vesicles (bullæ) to occur having the same frail character. The bullæ with a more resistant wall are a degenerative phenomenon, and will be considered under the head of dystrophies (§ 597).

The development of the vesicles is associated with marked irritative symptoms, such as ciliary injection, lacrimation, photophobia, and sharp pain. The latter without doubt is due to the traction made on the intra-epithelial nerve terminals. The vesicles very soon rupture, so that in most cases we do not see the vesicles themselves at all but only the epithelial defects left after they have collapsed. Then remains of the vesicle wall are found adhering to the edge of the epithelial defect and forming loose filaments that can be pushed about by moving the lids (cf. § 568). In spite of the violent irritative symptoms there is an undersensitiveness of the cornea.

There are the following varieties of keratitis with vesiculation:

565. (*a*) **Herpes[8] Febrilis Corneæ (Horner.)**—In febrile disease, especially of the respiratory organs (most frequently in epidemic influenza, next oftenest in bronchitis, pneumonia, ordinary influenza, etc.), less frequently in other febrile diseases, like typhoid fever, intermittent fever, etc., small vesicles (herpes facialis) often make their appearance on the lips, the alæ of the nose, the eyelids, the ears, etc. At the same time an eruption of small transparent vesicles, which are associated with violent symptoms of irritation, may occur upon the cornea. These are scarcely the size of a pin's head, and are often disposed in rows or in groups. The vesicles very speedily rupture, leaving small abrasions, the floor of which shows a faint opacity. Generally these abrasions soon heal, so that after two or three weeks the disease is over, without leaving any lasting opacity of the cornea. In severe, and especially in neglected cases, however, large corneal ulcers which not infrequently have a branching form (keratitis dendritica, see § 569), may develop from the small abrasions.

There is no doubt that the vesicles upon the cornea are entirely analogous to those that develop upon the skin. Just as the latter are generally present only upon one side of the face, so also the affection of the eyes is usually unilateral in its development, and is, moreover, confined to the same side as the vesicles upon the face. With careful treatment the prognosis is good, as in that case the disease generally gets well without leaving any opacity. The treatment is purely symptomatic, for the most being confined to the use of a protective bandage and of atropine. In accordance with the

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[8] From ἕρπειν, to creep.

probable neuropathic origin of many of these cases, arsenic is often indicated (Verhoeff). Iontophoresis with zinc sulphate (½-per-cent solution) sometimes helps (Zahn).

Experiments by Gruter and others indicate that herpes febrilis is an infectious condition caused by a specific micro-organism or virus.

566. (b) **Herpes Zoster[9] Corneæ.**—This is one of the symptoms of herpes zoster ophthalmicus—that is, zoster which is localized in the region of distribution of the trigeminus (see § 415). The cornea participates in the morbid process by forming small vesicles, which generally are arranged in groups and rupture speedily, as in the case of herpes febrilis. From the latter, however, herpes zoster is distinguished by running a considerably severer course, since the irritative symptoms persist after the rupture of the vesicles, the parenchyma of the cornea becomes deeply clouded at the spots where the vesicles were situated, and iritis is added to the other symptoms. Sometimes a regular keratitis profunda develops (§ 581). It takes a longer time for the opacities to disappear, nor is it always the case that they disappear completely. The insensitiveness of the cornea to touch is especially marked in herpes zoster. Reduction of the intra-ocular tension not infrequently exists so long as the inflammation is still recent; in other cases increase of tension sets in. The prognosis of this variety of herpes, from what has just been said, is less favorable than that of herpes febrilis; the treatment is the same. Iontophoresis with quinine is said to reduce the pain (McNab).

Other Forms of Vesicular Keratitis.—Apart from the forms above described, the formation of vesicles upon the cornea is further, in rare cases, observed under special conditions—e.g., as a result of the action of various, and particularly of corrosive, substances upon the cornea, after burns, after a cataract operation beneath the bandage, etc. Cases also occur in which, without known cause, there develop upon a perfectly sound cornea, vesicles or bullæ the formation of which we are inclined to attribute to nervous influences, as we do also in herpes febrilis and herpes zoster. Cases of this sort are usually characterized by periodical recurrence. One old lady for twelve years suffered from occasional attacks of inflammation in her eyes, which otherwise were sound. The inflammation occurred once or twice a year, and affected sometimes one eye, sometimes the other. It was associated with violent pain, great photophobia, and profuse lacrimation. In the first days of the attack the only things found were œdema of the lids, great ciliary injection, and a cornea covered with minute elevations, as if it had been strewn with sand. Then a large transparent bulla developed upon the cornea, after the rupture of which the inflammatory symptoms rapidly abated and the epithelial defect healed without leaving a trace behind.

The slit lamp shows that vesiculation of the cornea is not infrequent in conditions of corneal œdema; further, that vesiculation on the external surface (*keratitis vesiculosa externa*) may be associated with the development of vesicles on the internal surface (*keratitis vesiculosa interna*), produced by elevation of the corneal endothelium (Koeppe).

567. Non-vesicular Affections of the Corneal Epithelium.—Under the name of *keratitis epithelialis punctata* Koeppe describes a condition marked by the presence of discrete or confluent, grayish spots in the epithelium of the cornea, producing considerable impairment of the sight, but, only moderate irritation and injection. The surface of the cornea is smooth, lustrous, and not elevated, and there is no involvement of the subepithelial layer. Probably identical is the *keratitis epithelialis guttata* of Kraupa.

[9] = girdle-eruption, from ζωστήρ, girdle. We also speak of it as zona ophthalmica.

Cocaine opacity of the cornea, produced by weak (one or two-per-cent) solutions is marked by a delicate, diffuse, grayish haze in the epithelium, which may last som hours. If repeated doses of a stronger solution (4-per-cent or over) are used, discret opaque spots may form in the epithelium. Such spots are also produced by the instilla tion of a 2-per-cent solution of *holocaine.* On the other hand, atropine, homatropine, an pilocarpine produce no opacity of the corneal epithelium, and eserine does so to only very slight degree. In elderly subjects *fluorescein* sometimes produces a faint greenis tinging of the apparently unbroken cornea (Koeppe).

568. Filamentary Keratitis.—In the case of epithelial defects of the cornea we ofte observe fine filaments which adhere by one end pretty firmly to the edge of the defect while the other end, which is often swollen to a club shape, hangs down free. The fila ments consist sometimes of tough mucus, sometimes of epithelial shreds, representing th remains of the wall of a vesicle. Occasionally they occur after corneal erosions o wounds. A very characteristic appearance is presented when such filaments ar

Fig. 251.

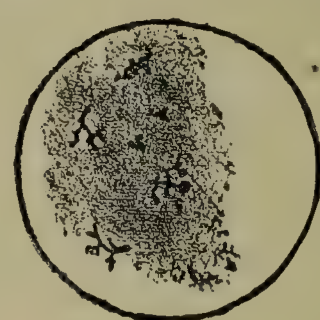


Fig. 252.

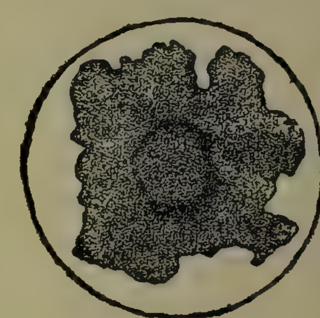


Fig. 253.

Fig. 251. Keratitis Dendritica.—To the left of the large branching opacity lies a group o faint, small maculæ representing the remains of an herpetic vesicle.

Fig. 252. Keratitis Stellata.—In the nasal half of the cornea which is moderately cloude are four large and two very small ulcers showing a stellate branching.

Fig. 253.—Large Superficial Ulcer Arising from Dendritic Keratitis.—The central gra ring represents an earlier stage of the ulcer, which since has advanced nearly to the margin of the cornea

attached to the erosions left after herpes corneæ, for then the cornea often seems to b covered with a great number of shaggy, gray, movable filaments. This condition i called *filamentary keratitis* (Leber, Uhthoff, Fischer).

569. Dendritic Keratitis.—In this condition there develops in the most superficial layers of the cornea a linear, usually zig-zag opacity, and from the angles of the zig-zag branches jut out, forming a tree-like figure. The surface over the opacity presents slender furrows, which are bounded by gray, somewhat raised edges. The branches are scarcely a millimetre in breadth, but at their points of junction and at their ends often show a knoblike enlargement.

Generally only one such branching opacity is found (Fig. 251). But sometimes several smaller foci with stellate branching occur (*keratitis stellata,* Fig. 252). In the later stages the branches of the tree often widen and coalesce to form a very shallow, irregularly lobulated ulcer (Fig. 253).

Under the slit lamp the lesions of dendritic keratitis are resolved into grayish, solid or hollow (vesicular) corpuscles situated in the epithelium and apparently in rela- tion with the terminal branchings of the corneal nerves. Beneath these epithelial lesions, even before the latter break down into ulcers, very faint inflammatory infiltrates are found in the most superficial layers of the stroma. In the case of the most recent lesions these infiltrates may be absent. By a sort of budding-out of the original epithelial foci the tree-like figures are formed (Koeppe).

The disease develops with irritative symptoms—photophobia, lacrima- tion, and the feeling as if there were something in the eye—, and these with

the condition itself may last for months. Finally the furrows become covered over, only the broadened spots remaining as little gray maculæ. At length these too disappear, so that no actual scar is left. In other cases, the inflammation suddenly assumes a purulent character, the tree figure disappears and is replaced by an irregularly shaped ulcer, whose edges take on a gray or yellow infiltration, hypopyon develops, and the process of disintegration spreads rapidly over the whole cornea. This sudden change, of course, depends on an infection of the ulcer by pyogenic germs. Extensive opacities result from this complication.

Dendritic keratitis was first described by Kipp, who pointed out that in America at least it is caused in nine cases out of ten by malaria. In Europe it is said to have the same causation as herpes febrilis.

For *treatment* we use atropine, disinfection, and a bandage. It is especially important that the bandage should be worn steadily, because the feeling that there is a foreign body in the eye is thus best relieved, and under the protection of the bandage the epithelium regenerates more quickly. It is very important also to protect the cornea from infection. If this is avoided the outlook is good even when the disease lasts a long time. For the relief of the pain, dionin may be applied but not cocaine; for the detrimental effect of the latter on the corneal epithelium would be especially marked in a disease like this which itself is associated with damage to the epithelium. Cases, like those occurring in America, which are due to malaria are relieved by quinine.

(b) *Conditions Affecting Especially the Subepithelial Layers*

570. Folds and Ruptures in Bowman's Membrane.—These appear as slightly curved or as straight, singly or doubly contoured lines, covered usually by smooth epithelium (Schirmer, Fuchs, Vogt). They occur with explosive injuries, phthisis bulbi, and extensive scars of the cornea, and are usually associated with similar folds in Descemet's membrane (cf. § 589).

Of similar nature is **Superficial Linear Keratitis.** This is a condition also called *letter-shaped keratits* (Haab) on account of the figures formed on the cornea. It is characterized by an acute onset with pain, congestion, and the formation in the cornea of a number of gray superficial epithelial ridges, usually running vertically and tapering at the ends. The ridges present a series of denser spots or nodes, which often stain with fluorescein, although the ridges themselves do not. The tension is usually markedly lowered, and the sight greatly impaired. The attack subsides in a few days or one or two weeks, leaving a grayish opacity, which at length disappears. If, however as often happens, there are several attacks, a permanent opacity is left which may render the eye partially or completely blind. The ridges on the cornea differ from those of dendritic keratitis in that they are multiple, straight, and have no bud-like outgrowths from them. They are caused by a wrinkling and elevation in Bowman's membrane with the formation of new fibrous tissue in the underlying substantia propria (Spicer and Greeves). The condition sometimes develops after injury, at other times without assignable cause. The treatment is symptomatic.

571. Keratitis Punctata Superficialis.—This condition, also called *keratitis subepithelialis punctata*, begins with the symptoms of an acute conjunctivitis. Either at the same time or not till some days or weeks afterward there are observed extremely minute dots which can be seen only with

a magnifying glass and which within a few days grow to form faint gray round spots (Fig. 254). These are sometimes only ten to twenty in number, sometimes very abundant—upward of a hundred. As in the case of herpes febrilis these are often arranged in groups or in short rows. In every case, however, the marginal portions of the cornea are the part least covered by the spots. The spots lie in the most superficial layers of the cornea, which latter looks dull because the epithelium over the spots bulges out in the form of a nodule. The irritative symptoms soon vanish, but the spots, as well as the punctate look of the corneal surface, generally remain for months almost unchanged, and then very gradually disappear. Whether the sight is little or much affected depends on the number and situation of the spots. Keratitis punctata superficialis is found most frequently in young people, and affects sometimes one, sometimes both eyes. It often begins with a catarrh of the air passages just as herpes febrilis corneæ does, but is distinguished from herpes by the absence of true vesicles and of the superficial losses of substance which develop from the latter; and for this reason it is but rarely associated with the development of ulcers. In the initial stage, atropine and hot applications are used; in the regressive stage, the yellow-oxide ointment and massage.

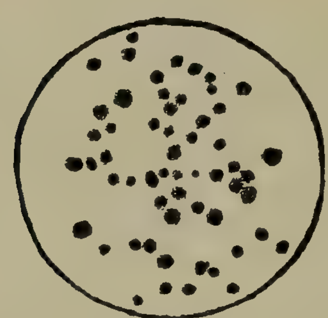


Fig. 254.—Keratitis Punctata Superficialis.

Quite like keratitis punctata superficialis is the *keratitis produced by chrysorobin*, but in this the epithelium over the discrete infiltrates becomes necrotic and forms minute ulcers (Koeppe).

572. Pannus.—Pannus consists in the new formation of a tissue resembling granulations beneath the epithelium of the cornea. Pannus is to be looked upon as an affection of the conjunctival layer of the cornea (conjunctiva corneæ, see § 9), and in every instance is simply one of the symptoms of a conjunctival disease—that is, either a trachomatous or a phlyctenular conjunctivitis (see §§ 469, 483 for particulars).

Not to be confounded with true pannus are the superficial new-formed vessels developing in connection with ulcers or as a result of trichiasis and the dystrophy known as pannus degenerativus (see § 597).

VIII. Deep Forms of Non-ulcerative Keratitis

573. These forms have as a common characteristic the development of an infiltrate in the middle and deep layers of the cornea, an infiltrate, however, which shows no tendency toward purulent disintegration, but—generally not till after existing quite a while—disappears again by resorption. When this takes place, the cornea in favorable cases clears up again completely, while in other cases opacities of a varying degree of intensity are left, and are sometimes even accompanied by flattening of the cornea. In keeping with the deep position of the infiltrate in the cornea, the uveal tract, and especially the iris and ciliary body are almost always implicated. The principal varieties are parenchymatous keratitis, sclerosing keratitis, and keratitis profunda.

(*a*) *Parenchymatous Keratitis*

574. Invasion.—Parenchymatous (or interstitial) keratitis may begin in the centre or at the margin of the cornea. If the disease invades the *centre* of the cornea first, we see small, dim, gray maculæ making their appearance in this situation, and lying in the middle and deep layers of the cornea; the surface of the latter is lustreless and dull. The number of maculæ gradually increases, so that they keep extending farther and farther toward the margin; but they are always massed most thickly in the centre, where they frequently become confluent. Since even between the maculæ the cornea is not clear, but shows a fine diffuse cloudiness, the entire cornea may in severe cases get to look uniformly gray, like ground glass. As soon as the opacity of the cornea has advanced somewhat farther, vascularization begins by the penetration of vessels into the cornea from different spots upon the corneal circumference. We see the vascular trunks coming out from beneath the limbus, because—as opposed to the vessels in pannus—they arise from the deeply situated vessels of the adjacent sclera (Figs. 232 and 233). They branch in tufts, like a brush, in the deep layers of the cornea, and often appear quite indistinct and of a dirty-red or grayish-red color, because covered by the clouded superficial layers of the latter.

In the much more frequent cases in which the disease begins at the *margin* of the cornea, the first thing we see is an area of marginal cloudiness over which the cornea has lost its lustre. The cloudiness is deeply situated, and when regarded with the naked eye looks uniformly gray, but with the magnifying glass can generally be resolved into separate maculæ or dim parallel streaks. Soon similar areas of cloudiness appear at other spots of the corneal margin, and then push their way forward concentrically from all sides toward the centre of the cornea. Simultaneously with the appearance of the marginal opacities the corresponding portions of the limbus become injected and the vessels of the corneal margin begin to grow out. The formation of vessels, as far as it originates in the network of marginal loops in the limbus, soon comes to an end, so that the limbus advances only a little way out upon the cornea, where it looks red and swollen ("epaulet-like" swelling of the limbus). While this swelling soon goes down again, the deep vessels, which come out from beneath the limbus, grow farther and farther into the cornea, and follow closely upon the opacity which advances in front of them; it looks as if they were pushing the opacity before them. These vessels have, as in the case of those of the first form, the characters of deeply situated vessels, shown by their broom-like branching and by their dull, dead, grayish-red hue.

When parenchymatous keratitis has attained its acme, the cornea is often so opaque that we scarcely recognize the iris through it. At the same time it loses its lustre completely, so that it looks as though smeared with grease (with the magnifying glass we recognize numerous minute elevations of the epithelium, which make the surface of the cornea rough, as if made of fine shagreen). Sight is so reduced that the patient can only count fingers held very close to him, or, still worse, can only recognize the movement of

the hand before his eye. Now gradually the process of recovery begins starting from the margin, where the cornea first becomes transparent again while at the same time the vessels grow constantly fewer and fewer. Th centre of the cornea remains opaque the longest, but finally clears up, too until only a fine diffuse cloudiness remains, which causes but little impair- ment of sight. This cloudiness, together with a few very minute blood-ves- sels which are visible only with the magnifying glass, can still be made ou years afterward, and are certain signs of the previous existence of a paren- chymatous keratitis.

575. Clinical Varieties.—Among the more frequently occurrin *variations* in the clinical picture of parenchymatous keratitis the followin may be mentioned:

In that form which begins with maculæ in the central portions of the cornea it no infrequently happens that the maculæ at a certain distance from the centre are particu- larly dense, and thus form a very opaque ring, which in the subsequent course con- tracts more and more toward the centre of the cornea (*keratitis centralis annularis* o Vossius). Allied to these cases are those in which the central part of the cornea become particularly opaque through the confluence of the maculæ, which form a white dis pretty sharply separated from the less opaque, marginal portions of the cornea. Case sometimes are seen in which this central opacity remained permanently after the mar- ginal portions had cleared up, and formed a dense, white, sharply circumscribed spot i the centre of each cornea, just as if there had been a deeply penetrating central ulce (Fuchs).

Sometimes the distribution of the infiltration in the cornea is such that the denses opacity occupies the lowest part of the cornea, as if the products of inflammation ha arranged themselves there in obedience to the law of gravitation. In that case th opacity is bounded above by a convex line, or it forms a triangle, the base of whic corresponds to the lower margin of the cornea, while its apex looks upward. The per- manent opacity that results from this has the greatest similarity to those triangula opacities in the lower part of the cornea which are left by an irido-cyclitis, when a exudate at the bottom of the anterior chamber has remained deposited for a pretty long time upon the posterior surface of the cornea. (See § 588.)

Just as great variations exist in regard to the density and extent of the infiltration, so they do also in regard to the *vascularization*. In many cases the cornea is so abundantly vascularized that it looks like a red cloth, in others, on the contrary, it is almost devoid of vessels, and is like white ground glass. Lying between these extremes are the numerous cases in which vessels develop from only single spots upon the corneal margin, so that simply a sector of the cornea looks red, or so that only single tufts of vessels are discoverable. Hence we may distinguish between a vascular and a non-vascular form, according to the relations of the vessels. It must be remarked, however, that even in the non-vascular form one or two vessels can generally be made out with the aid of the magnifying glass.

The vessels in parenchymatous keratitis generally lie in the *deep layers*. Yet it often enough happens that we find, especially with the aid of the magnifying glass, a few vessels also which evidently arise from the network of marginal loops or from larger con- junctival blood-vessels, and which therefore lie superficially in the cornea. All vessels tend toward the centre of the cornea, but do not generally reach it, so that here a roundish spot of the size of a millet seed or more remains unvascularized. The vascular portions of the cornea look red, and if the vessels are abundant, rise above the level of the non-vascular

centre; the latter, accordingly—which, in consequence of the marked infiltration, is gray or even yellowish gray—is depressed. We must not on this account allow ourselves to be misled and consider the depressed gray spot as an *ulcer*, for parenchymatous keratitis does not, as a general thing, lead to ulceration. Exceptions to this rule, though rare, do, however, occur. Two cases have been seen in which perforation took place in the centre of the cornea (Fuchs).

When we consider how the cases vary both in respect to the opacity and to vascularization, we comprehend why parenchymatous keratitis should present a very varying clinical picture, and hence often offer difficulties in the way of diagnosis for the beginner. But we shall generally be able to make the diagnosis with certainty if we hold fast to those symptoms which are common to all the cases—namely, the deep situation of the opacity and of the vessels, the typical increase in the infiltration up to to a certain, usually considerable, degree, and finally the absence of purulent disintegration, so that the *formation of ulcers practically never takes place.*

576. Symptoms, and Complications.—Parenchymatous keratitis is accompanied by *irritative symptoms* of inflammation, such as pain, photophobia, and lacrimation. These are sometimes very slight, sometimes violent; in general we may say that they are more pronounced, the greater the amount of vascularization with which the keratitis is associated. Furthermore, parenchymatous keratitis is almost always complicated with *inflammation of the uveal tract.* In the lightest cases there is merely hyperæmia of the iris, which makes itself apparent by the failure, complete or nearly so, of the pupil to dilate under atropine. In severe cases there is irido-cyclitis, which may lead to the formation of posterior synechiæ, the formation of deposits upon the posterior surface of the cornea, and seclusion and occlusion of the pupil. In particularly bad cases a plastic irido-cyclitis develops, which terminates in flattening of the cornea, or even in atrophy of the eyeball.

The degree in which the uvea participates in the inflammation varies greatly. Minute deposits, which are discovered in making a careful examination of the cornea with the magnifying glass at the time when the opacity is resolving, are scarcely ever wanting. Besides these, the most frequently occurring complications are posterior synechiæ and also chorioiditic foci; hypopyon, on the other hand, is extremely rare. In many cases the part that the uvea takes is so slight as not to be clinically demonstrable, in other cases, on the contrary, it is so very prominent, as compared with the process in the cornea, that what we have before us is really an irido-cyclitis—the implication of the cornea being evidenced only by the presence of a few spots of opacity in its deep layers. Thus there is a continuous series of intermediate forms between typical parenchymatous keratitis and irido-cyclitis e lue hereditaria. When both uvea and cornea are markedly involved the condition is often called *uveitis anterior.* This disease has the characters of a chronic iritis (see § 684) combined with a marked keratitis and sometimes with a deep scleritis as well. It is extremely chronic, and is undoubtedly of constitutional origin, probably often being due to tuberculosis, although in many cases the cause is obscure.

The *anterior chamber* in parenchymatous keratitis is often found to be deeper than usual, a circumstance which should not, however, be referred without further consideration to an ectasis of the cornea, a thing which occurs quite rarely. On the contrary, the cause of it ordinarily is a recession of the iris due to the increased accumulation of the aqueous—an accumulation to which both the irritated state of the uvea and the altered conditions of diosmosis may contribute.

Often it is impossible to obtain an adequate *dilatation of the pupil* with atropine, even though no evident signs of iritis are present. It seems that in these cases the atropine does not diffuse through the inflamed cornea to the same extent as it does through a

sound one, so that it does not in fact get into the aqueous in sufficient quantity to dilate the pupil.

The *intra-ocular pressure* not infrequently shows an alteration in parenchymatous keratitis. Generally, it is diminished so that the eye appears softer, although we need not therefore imagine that an atrophy of the eyeball is beginning. An increase of tension is but rarely observed, occurring for the most part only when the keratitis has induced ectasis of the cornea. In that case sometimes it does not occur until years after the inflammation has run its course. Fuchs has seen this even in those cases in which no ectasis of the cornea has been left.

Many cases of parenchymatous keratitis are accompanied by *cnorioiditis.* This is localized in the most anterior segment of the chorioid (chorioiditis anterior), which is covered with numerous—in most cases black—spots. This variety of chorioiditis would probably be set down among the most frequent symptoms accompanying parenchymatous keratitis if the examination with the ophthalmoscope, and hence the determination of the presence of chorioiditis, were not rendered impossible, while the inflammation lasted, by the cloudiness of the cornea. The making of this examination and diagnosis can be done only when the cornea has cleared up once more after the inflammation has run its course. It is also often possible to make out the existence of peripheral chorioiditis in the other, as yet uninflamed eye.

Another and rarer complication of parenchymatous keratitis is a *diffuse scleritis* in the region surrounding the cornea. This may subsequently give rise to ectasiæ of the sclera.

577. Course.—Parenchymatous keratitis always runs a chronic course. The inflammatory symptoms keep on increasing for one or two months, until the disease has reached its acme. Then the irritative symptoms very soon abate, and the process of clearing up of the cornea makes at first rapid progress. Afterward, however, it goes on more slowly again, and the centre of the cornea in particular remains for a long time opaque, so that sight is not restored until late in the disease. It takes from half a year to a year, or even more, for the cornea to gain the full degree of transparency which it can possibly have with an inflammation of the given intensity.

All the cases do not run their course in the way described. There are, for example, many lighter cases in which the changes do not go far, and which hence, too, are completed in a shorter time. Thus the process may go no further than the formation of a few maculæ, which gradually disappear again without the associated inflammatory symptoms being at any time marked. If the opacity begins at the margin of the cornea, it often remains confined to that section of it from which it originally started. In that case, if it pushes its way farther from the margin toward the centre, only a sector, and not the entire cornea, is rendered opaque. Conversely, there are also very severe cases in which dense opacities remain as a permanency. Again, owing to the inflammatory infiltration, softening of the cornea may be produced, so that the latter gives way before the intra-ocular pressure, and keratectasia develops; in this case also the cornea remains permanently opaque, and to quite a marked degree. The worst cases are those in which, by subsequent shrinking of the exudate, the cornea becomes flattened, densely opaque, and of a tendinous appearance, in which case the sight is nearly or quite lost.

Parenchymatous keratitis generally attacks both eyes, and more frequently both in succession than both at once. Sometimes there is even an

interval of several years between the involvement of the two eyes. Recurrences of the disease take place, but are not common.

The *prognosis* of the disease, from what has been said, is unfavorable as regards its duration, inasmuch as it drags on for months and years, especially if the two eyes are successively attacked. On the other hand, the prognosis in regard to the ultimate outcome must be put down as good, because in by far the greater number of the cases a good, or at least serviceable, degree of sight comes back. By holding up this prospect of recovery the physician must keep up the courage of his patient, who, because of the slow progress of the disease, is very apt to lose all hope of a restoration of his sight.

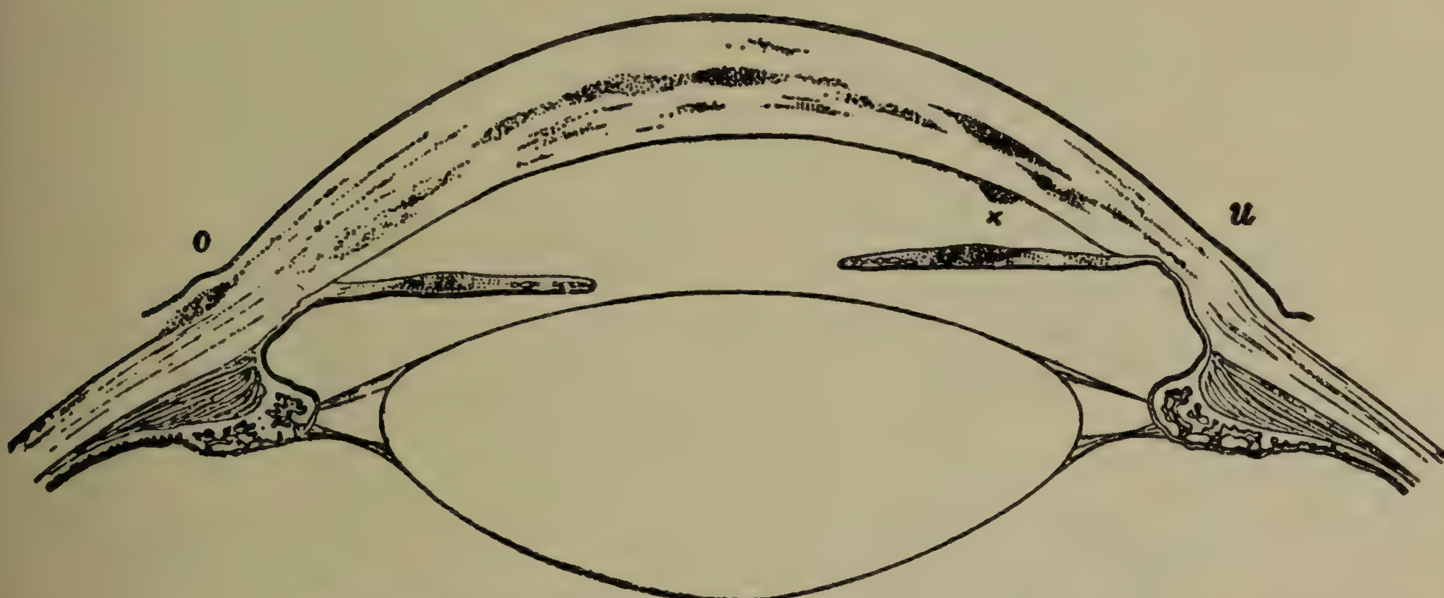


FIG. 255.—PARENCHYMATOUS KERATITIS. Magnified 7 × 1.

The patient, a girl of nineteen, had already passed through one attack of parenchymatous keratitis six years before. This recurred, the cornea became ectatic, increase of tension set in, and as the result of this the eye became entirely blind. Since the eye was inflamed and painful all the time, it was enucleated at the patient's own wish. At the time of enucleation, the cornea was densely permeated with little white spots which were situated in the middle layers of the cornea, and the latter was consequently so opaque that the iris could not be seen through it. In the preparation, contrary to the usual relations, the cornea is seen to be somewhat thicker in the centre than at the edge. To the clinically demonstrable white spots correspond small foci, consisting of uninucleated round cells. These foci are largest and most numerous in the middle layers of the cornea. At the margin of the cornea are to be seen, even in the anterior layers, files of cells accompanied by new formed vessels. At the upper margin of the cornea, *o*, there is situated a small episcleral inflammatory focus. Near the lower margin of the cornea at *x*, there is a mass of exudate (precipitate) on the posterior surface of the cornea. The iris, as is the rule when there is an increase of tension, is adherent to the cornea by its periphery, and hence is driven bodily forward, so that it no longer is in contact with the lens. The marginal portion of the iris, which is adherent to the cornea, is very much thinned by atrophy and at the lower side, *u*, is pushed somewhat into the also attenuated sclera (beginning of an intercalary staphyloma). The free portion of the iris is rather less atrophic, and both in its upper and lower part contains an inflammatory nodule, which like the nodules in the cornea, consists simply of uninuclear cells. The ciliary processes are well preserved. They are at a greater distance than normal from the margin of the lens, because owing to the increase of tension, the eye as a whole has become somewhat enlarged but the lens has not shared in the enlargement.

578. **Morbid Anatomy.**—Anatomical examination of an eye affected with parenchymatous keratitis shows small foci of inflammation which correspond to the gray specks seen on clinical inspection and which are formed by an accumulation of uninuclear leucocytes in the middle and deep layers of the cornea. Similar nodules may also be found in the anterior portions of the uvea (iris, ciliary body, most anterior part of the chorioid—see Fig. 255). The nodules sometimes contain giant cells, but yet are not typical tuberculous nodules. It may chance that disintegration of the corneal substance itself occurs at the site of the nodule, but there is not the caseation that is characteristic of tuberculosis. In the case represented in Fig. 256, instead of discrete nodules there was a uniform dense infiltration of the most posterior layers of the cornea, so that the latter looked as if transformed into granulating tissue; furthermore, numerous new-formed blood-vessels (*g*) were seen in the most posterior and the middle layers. In deep-seated infiltration, destruction of Descemet's membrane sometimes occurs in spots.

579. Etiology.—Parenchymatous keratitis is a disease of youth, appearing, as a rule, between the sixth and the twentieth year of life. It is only the exception that persons before or after this age (sometimes even after the thirtieth year) are attacked. (In 200 cases examined by Uhthoff 9 were between 30 and 40 and 4 were over 40.) The female sex suffers from it more frequently than the male. The commonest cause of the disease is syphilis, and especially hereditary syphilis. It affects thus the children of

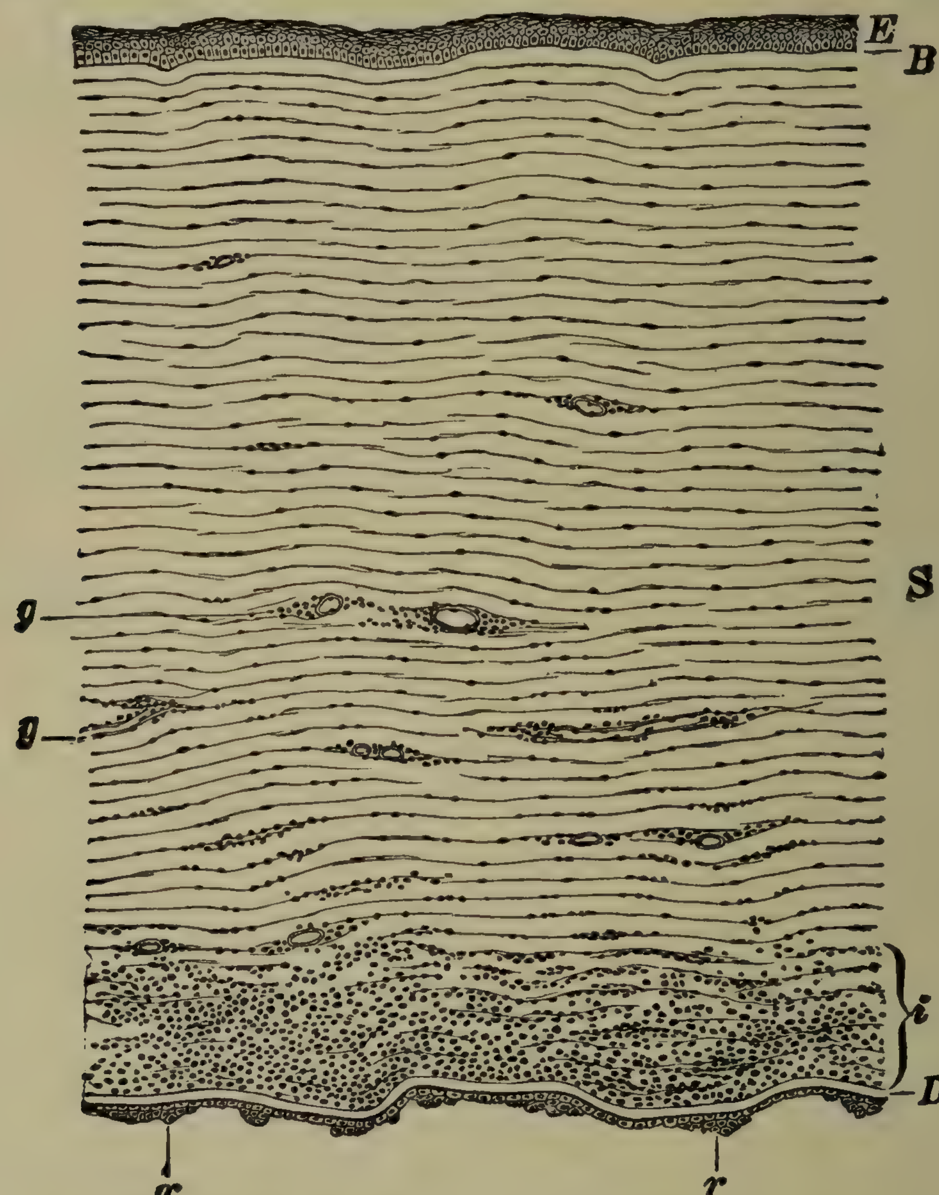


FIG. 256.—CROSS SECTION THROUGH A CORNEA WITH PARENCHYMATOUS KERATITIS. Magnified 100 X1. (After a preparation of Dr. Nordenson's.)

The stroma, *S*, of the cornea shows an infiltration, which begins in the middle layers, and keeps on increasing more and more posteriorly, so that the deepest layers, *i*, have assumed the aspect of a granulating tissue. On account of the inequality in the degree of thickening of these layers, Descemet's membrane, *D*, is undulated; upon its endothelium there are deposited in places small accumulations of round cells, *r*. In the middle and deep layers of the cornea we see the transverse and longitudinal sections of newly formed blood-vessels, *g*, *g*, while the most anterior layers, and also Bowman's membrane, *B*, and the epithelium, *E*, are normal.

syphilitic persons, but does not pass to the next generation, i.e., not affect the children of those who are themselves the subjects of a parenchymatous keratitis (Hugenin). We prove the existence of hereditary syphilis partly from the history, partly from the attendant symptoms. It is rarely necessary to get a direct *history of syphilis* in the parents; it is usually sufficient to determine by questioning whether many children have died in the family (the mortality of the children of syphilitic parents amounts, on an average, to 50 per cent), whether premature labors, and especially those in which the fetus was dead or putrefied, have not occurred, etc.

The *symptoms of hereditary syphilis* which patients with parenchymatous keratitis frequently exhibit are as follows:

1. A peculiar formation of the face and cranium. The upper jaws are markedly flat, and the bridge of the nose low or actually sunken in. Not infrequently there is ozæna or disease of the lacrimal sac, the latter in consequence of the changes in the nose. The frontal eminences are very prominent. The cornea, which is ordinarily a horizontal ellipse, has often the shape of a vertical ellipse. The intelligence of these patients is often abnormal, they being either precocious, or, on the other hand, backward in mental development.

2. The incisor teeth are abnormally shaped (Hutchinson), so that, instead of a straight edge, they show a semilunar notch (Fig. 257). This change is present only in the teeth of the second dentition, and in them most

A

B

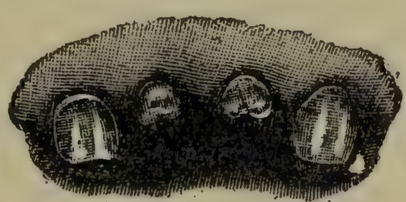


FIG. 257.—THE UPPER INCISOR TEETH IN HEREDITARY SYPHILIS. (After Hutchinson.)

A. The interspace between the middle incisor teeth is abnormally broad. The teeth themselves have a semilunar notch on thier cutting edge. The two lateral incisor teeth have somewhat convex lateral borders which converge downwards.

B. The incisor teeth are all stunted, and hence the interspaces are enlarged. The middle incisor teeth are most undeveloped and show the notching of the cutting edge. The lateral incisor teeth have convex sides which converge downwards.

frequently in the upper central incisor teeth. Often the incisor teeth are all stunted, so that they are either too small or some are altogether wanting.

3. At the angles of the mouth we may find fine cicatrices, the relics of former rhagades; so also cicatrices in the buccal and pharyngeal cavities (especially on the hard and soft palate) point to the existence of antecedent syphilitic ulcerations.

4. Numerous enlarged lymphatic glands can be made out, especially upon the neck. These are small, hard, painless, and with no tendency toward ulceration, by which characters they are distinguished from the lymph glands of scrofulous subjects, which are large and soft, and readily undergo caseation.

5. Swellings of the periosteum (tophi), which are hard and are but little or not at all painful, occur on the long bones. They are most frequently and most easily found upon the anterior border of the tibia.

6. Often there is inflammation of the knee-joint, either occurring simultaneously with the keratitis or, more often, antedating it by some years. The inflammation is ordinarily bilateral, pretty torpid, and of several months' duration, but benign, in that it gets well without leaving lasting sequelæ.

7. We frequently find hardness of hearing, which, with the outbreak of the keratitis, sometimes increases to absolute deafness.

It is important to look for all of these symptoms, for any one of them

by itself is not to be looked upon as conclusive evidence of the existence of hereditary syphilis; and, on the other hand, we ought not to expect to find all the changes above given distinctly marked in the same individual at once. If, nevertheless, a case remains doubtful, we make a Wassermann or luetin test, which in cases of hereditary syphilis usually turns out positive, and which is nearly always also positive in parenchymatous keratitis. Moreover, in a large majority of the cases the same tests prove one or both of the parents of children to be affected with syphilis (Lesser and Carsten). In a few cases this form of keratitis is observed in acquired syphilis, and we must not forget that the latter may be acquired even in childhood (e.g., through a nurse).

Lately the view has gained ground that a fairly large proportion—perhaps one tenth—of the cases of parenchymatous keratitis are due to *tuberculosis;* and we must remember that this may be so even in a syphilitic subject (see infra). In a series of 163 cases seen by Uhthoff, in which the cause could be determined, 156 were due to syphilis and 7 to tuberculosis. This probably is not far from the proportion generally obtaining, although others have found a much larger percentage of tuberculous cases.

The typical course of parenchymatous keratitis, and the participation of both eyes in it, early suggested the idea of its being due to a *constitutional cause.* Thus Mackenzie gave an excellent description of this disease under the name of corneitis scrophulosa, and at the same time gave a number of the accompanying symptoms which he looked upon as signs of scrofula. Hutchinson has the credit of having completed this series of symptoms and of having at the same time furnished the proof that they belong not to scrofula, but to hereditary syphilis. This novel view was slow in making its way. Many at first would only allow that it was true for a limited number of cases, and hence divided parenchymatous keratitis into two forms, which they called keratitis scrofulosa and keratitis syphilitica. But the more precise the knowledge became in regard to the symptoms of hereditary syphilis, the more the conviction grew that this disease lay at the root of most cases of parenchymatous keratitis, whatever form the latter might exhibit. Some succeeded in producing in monkeys and rabbits by inoculating them with syphilitic material an inflammation of the cornea which resembles the parenchymatous keratitis of man and in which, too, spirochætæ could be demonstrated in the tissue of the cornea (Scherber, Greeff).

While, however, nearly all agree that hereditary syphilis and to a less extent tuberculosis are nearly constant concomitants of parenchymatous keratitis, opinions vary widely as to the *nature of the connection.* That it is not a direct connection seems evident. For (a) spirochætæ are very rarely found in the cornea in this disease, while in undoubted luetic keratitis (e.g. in the experimental parenchymatous keratitis of animals) they are found in abundance; (b) the condition is very rare in acquired syphilis; (c) the lesions in parenchymatous keratitis are essentially degenerative (Michel, Elschnig) and do not have the characters of a syphilitic exudation; (d) antisyphilitic treatment, which is effective in cases of undoubtedly luetic keratitis, is usually ineffective in this condition, in which, moreover, the Wassermann reaction persists in spite of treatment (Hansell and Schneidemann). It seems, therefore, that parenchymatous keratitis is not the result of a direct syphilitic infection. It has been suggested that it may be an *anaphylactic phenomenon,* the cornea being sensitized at some early period of life (perhaps even in intra-uterine life) by the syphilitic virus, so that later it is readily attacked even by the attenuated virus (Derby and Walker), this second attack occurring at some time when the resistance of the system is reduced in some way. Some think that this *reduced general resistance* is itself the essential factor, and that because of it the cornea and uvea succumb to an attack that they would otherwise withstand. It is on this principle that Dunn explains the genesis of uveitis in keratitis parenchymatosa. He says that

vicious circle is established, by (*a*) the development of an autotoxæmia, originating probably in the intestinal tract; (*b*) excessive action of the thyroid gland to combat the toxæmia; (*c*) failure of the thyroid resulting in hypothyroidism and pervading uncontrolled toxæmia. According to this view, which resembles that held by Elschnig, the uveitis accompanying keratitis parenchymatosa is often due not so much to the syphilis as to metabolic toxæmia and general malnutrition due to hypothyroidism. The theory seems to find some corroboration in the occasionally successful results of treatment with the thyroid extract (§ 580). So also Panas thought the cause of parenchymatous keratitis to consist in a condition of cachexia and dystrophy and others have attributed it to general nutritional disturbance, probably of endocrine origin. But their views, so far as they exclude syphilis as at least an indirect cause, go too far. It seems probable rather that, while parenchymatous keratitis is essentially a degenerative process due to some constitutional disturbance affecting the nutrition and metabolism, the underlying cause of this very constitutional disturbance must be the syphilis or tuberculosis which is uniformly present.

Parenchymatous keratitis is one of the latest of the forms under which hereditary syphilis shows itself and is therefore rightly regarded as one of the most important and most frequent symptoms of lues hereditaria tarda. Many believe that there is no lues hereditaria tarda if we use the term in the sense that the symptoms of lues which appear in later life are really the first symptoms of the disease; they think that, on the contrary, luetic symptoms must have been present in every such case either at the time of birth or soon after, but that either these symptoms were overlooked or else the physician had failed to encounter any signs of them because he had taken charge of the case after the symptoms had gone. Thus it is supposed that parenchymatous keratitis is never really the first symptom of hereditary syphilis. But positive observations contradict this view (Fuchs).

In determining the presence of hereditary syphilis, examination of the patient's brothers and sisters may be of service, since we find in them, too, symptoms of the same disease. Moreover, it is not at all rare for two or even three of a set of brothers and sisters to be affected with parenchymatous keratitis. In considering the origin of the trouble we must remember that a woman may bear syphilitic children begotten by her diseased husband, without at the same time acquiring syphilis herself; to the syphilis of her own children she is immune.

580. Treatment.—*Local* treatment during the period of progression consists in combating the inflammation by protecting the eyes from light and by instilling atropine, which latter counteracts the complications arising from the iris. Moist hot compresses often ameliorate the symptoms of irritation and accelerate somewhat the progress of the disease. Dionin acts favorably on the photophobia and the pain. Many think subconjunctival injections (§ 135) very effective.

In the regressive period, the thing to do is to secure as thoroughgoing a clearing up of the cornea as possible. For this purpose dionin again and the well-known irritant remedies—like calomel, tincture of opium, yellow-precipitate ointment, steam, hot air, etc.—are indicated (see § 546). These, however, should be applied only when, on making cautious tests, the eye is found to bear them well—i.e., does not through their use fall into a state of renewed irritation. It is advisable to keep on with these remedies, interchanging them frequently, for a very long time—for months or years. If ectasis of the cornea threatens, it is to be combated by a pressure bandage, which, if necessary, can be combined with repeated paracentesis of the cornea.

General treatment in those cases in which syphilis is the cause must be directed against the latter. Salvarsan according to many, is helpful in shortening the process, especially if it is given repeatedly. Others have found it of little avail. It should be combined with mercurial treatment. The latter alone is ordinarily not very efficient, though it is more so in cases due to acquired syphilis. In adults we use mercury by inunction or better still by intramuscular injection. We give at periodical intervals a series of injections, using the bichloride (up to gm. 0.01) in solution every other day, or the salicylate (up to gm. 0.06), or calomel suspended in albolene or oil every ten days. In children the internal administration of corrosive sublimate is preferable. We give pills of 1 mg. beginning with one a day, increasing the dose to from six to ten pills a day, according to the age, and being mindful to pay careful attention to the condition of the mouth, in order to avoid salivation. If the Pirquet or tuberculin reaction is positive, tuberculin should be tried, and this may succeed in cases which are syphilitic as well as tuberculous, but in which treatment for syphilis has failed to help (Török; cf. § 140). In general, the best results are obtained if careful and persistent hygienic measures (fresh air and exercise, good diet, etc.) are combined with the specific treatment (Derby and Walker). In the lighter cases of parenchymatous keratitis, indeed, we may confine ourselves to a simple tonic treatment with the simultaneous employment of remedies containing iodine (cod-liver oil with iodine, iodide of iron, potassium or sodium iodides, and mineral waters containing iodine, the latter especially as a form of after treatment). Thyroid extract, gm. 0.20 to 0.30, according to age, may be used with advantage in some cases (Radcliffe, Dunn). Unfortunately, we must say that, in many cases, parenchymatous keratitis, even under the most careful treatment, runs a course that is not essentially different from what would have been the case without any treatment at all. Not infrequently we see the disease while under treatment break out in the other eye, without our being able to prevent the opacity from spreading gradually over the entire cornea in this eye also. Yet, though we may not be able to arrest the process, we can in many cases shorten it, and we can render important service by combating the complications arising from the uvea, and also in securing a more rapid and more perfect clearing up of the corneal opacities during the period of regression.

(*b*) *Keratitis Profunda* [10]

581. In this a gray opacity develops very gradually in the cornea—ordinarily in its centre—an opacity which is situated in the middle and deep layers of the cornea, and over which the corneal surface is gray and punctate, but not depressed. Seen with the naked eye, the opacity looks uniformly gray, while with the magnifying glass it may be resolved into dots and maculæ, or into gray interlacing striæ. After the opacity has remained for some time (several weeks) at its acme it begins slowly to abate, without ulceration having taken place. The development of new vessels is either

[10] Synonyms: Central parenchymatous infiltration of the cornea, keratitis parenchymatosa circumscripta.

entirely absent or is very inconsiderable. The accompanying symptoms of inflammatory irritation are sometimes slight, sometimes pretty violent. The participation of the iris is mostly limited to hyperæmia.

The disease attacks adults only and usually affects but one eye. It lasts from four to eight weeks or more but may recur. In the lighter cases the transparency of the cornea is completely restored, while in other cases diffuse opacities remain permanently in the centre of the cornea.

The *causes* of keratitis profunda are in the great majority of cases unknown. For individual cases the following causes have been given: 1. The effect of cold. Arlt has characterized such cases as keratitis rheumatica. These ordinarily run their course with marked inflammatory symptoms, especially with violent pain and photophobia. 2. Herpes zoster ophthalmicus (§ 415). 3. Intermittent fever in its chronic form of malarial cachexia (Arlt). In this, sometimes a keratitis profunda develops which is characterized by the absence of marked symptoms of irritation, and also by an unusually chronic course (Arlt). 4. Tuberculosis. 5. After injuries, especially contusions, a keratitis profunda not infrequently develops, the peculiarity of which is its comparatively rapid course and the speedy restoration of the transparency of the cornea.

Treatment consists, locally, in the application of a bandage or of protective glasses, and the use of atropine. Moist hot compresses, dionin, and subconjunctival injections of salt or mercury solutions may accelerate the progress of the case provided these are well borne. After the inflammatory symptoms have run their course, irritant remedies for clearing up the opacity are indicated. The general treatment depends upon the cause that we are able to discover for the keratitis.

(*c*) *Sclerosing Keratitis*

582. This has retained its name given it by Von Graefe, because at the spot where the infiltration is present in the cornea, dense and white opacities are left which often look like sclera. Sclerosing keratitis occurs either alone or more frequently as an accompanying symptom of scleritis (§ 625). If a scleritic nodule is situated near the margin of the cornea, there develops in the adjacent portion of the latter an opacity which is situated in its deeper layers (Fig. 278). It has approximately the shape of a triangle, the base of which is situated at the corneal margin, while the rounded apex looks toward the centre of the cornea and gradually merges into the transparent cornea. The opacity thus occupies a sector of the cornea, the base of which corresponds to the scleritic nodule. In many cases other dense opacities of rounded or irregular shape also develop at a distance from the corneal border, and even in the centre of the cornea. The opacities are gray or grayish-yellow, and gradually increase in density until the cornea at the affected spot has become completely opaque. The surface of the cornea over the opacity is stippled but not depressed; vascularization is either altogether absent or is very slight, and, when it is present, is in the deep layers of the cornea. After the opacity has reached its maximum density, a gradual retrogressive process sets in, without ulceration having taken place

at any time. The process of clearing affects the thin edge of the opacit and its apex, which looks toward the centre of the cornea; the greater portio of the opacity remains permanently and becomes ultimately bluish-whit like the adjacent sclera, into which it passes without any sharp line of de marcation. At the spot where the opacity is found, therefore, it looks as i the sclera had pushed its way into the space occupied by the cornea.

Like scleritis itself, this keratitis that accompanies it shows repeate recurrences, and it may happen that in severe cases the entire cornea i sclerosed with the exception of a small area in the centre.

The disease usually attacks young persons, especially females, an often occurs in both eyes. It sometimes runs a torpid course, but mor often is associated with marked irritation which accompanies each recur ring attack. The inflammatory symptoms are in part also caused by th complicating scleritis, and iritis. The etiolog is ordinarily the same as that of scleriti namely in young persons, tuberculosis, an sometimes hereditary syphilis, and in olde patients gout and rheumatism. Often no cer tain cause can be found for the disease.

FIG. 258.—KERATITIS MARGINALIS PROFUNDA.

The finely striate marginal zone represents the limbus, adjoining which above and on the outer and inner sides is the arc-shaped infiltrate drawn in darker shading.

Locally the same remedies are indicate that have been suggested for keratitis pro funda. The general treatment is directed t the cause of the disease, in case such a caus can be ascertained.

(*d*) *Other Forms of Deep Non-Suppurative Keratitis*

583. Keratitis Marginalis Profunda.—This rare disease generally affects old people and occurs for the most part in one eye only. There forms upon the margin of the cornea with moderate symptoms of irritation, a gray, later grayish-yellow, or even purulent-yellov opacity, which directly adjoins the sclera and hence extends under the limbus, while o the other hand it reaches for a distance of 1 or 2 mm. into the transparent cornea (Fig 258). This marginal zone of opacity generally embraces from one-third to one-half o the circumference of the cornea (most frequently the upper part), or in rare instance surrounds the entire cornea. The surface of the cornea over the opacity is somewha dull, but shows no loss of substance, and never any exfoliation of epithelium. Th limbus soon pushes forward so far as to cover the opacity with its vessels. The irritativ symptoms disappear in from one to two weeks, while the marginal infiltrate is transforme into a permanent gray opacity. This opacity bears a great resemblance to the arcu senilis, from which it is chiefly distinguished by its not being separated by a transparen zone from the scleral margin, but passing into the latter without any clear line of demar cation. Iritis does not occur in this affection and ulceration of the cornea is extremely rare. On account of the marginal situation of the residual opacity, this form of keratitis is without danger to the sight.

584. Deep Scrofulous Infiltrates occur in phlyctenular conjunctivitis under the form of extensive gray, subsequently yellow, opacities in the middle and deep layers of the cornea. They may either proceed to suppurate, or they may go on to resorption, in which case the cornea clears up—sometimes in a surprising manner. For a more detailed account, see § 483.

585. Deep Keratitis in Irido-Cyclitis.—In every irido-cyclitis the cornea is somewhat dull and opaque. But in many cases of irido-cyclitis the implication of the cornea is more pronounced, an infiltration of a gray or often later of a yellowish color being

deposited in the deeper layers. The inflammation never assumes a purulent character as in keratitis pustuliformis profunda (§ 563) and hence also no hypopyon is formed. Later, vessels develop about the infiltrate and the latter disappears, but always leaves a permanent opacity and in bad cases a flattening of the cornea. The sight in these cases is nearly or quite abolished, but that not so much from the affection of the cornea as from the irido-cyclitis.

586. Keratitis Punctata Profunda.—In irido-cyclitis resulting from acquired syphilis it not infrequently happens that isolated gray specks appear in the middle and deep layers of the cornea. These have been described by Mauthner, Hock, Purtscher, and others as *keratitis punctata syphilitica.* The condition looks something like keratitis punctata superficialis (§ 571) but is distinguished not only by the etiology, but also by the situation.

587. Posterior Annular Opacity.—Risley describes a condition associated with severe asthenopia and sometimes sick headaches, in which the centre of the cornea is occupied by a dotted ring, concentric with the pupil and enclosing a multitude of small dots. This opacity, he thinks, is situated in (not on) Descemet's membrane and is an expression of impaired nutrition due to uveal disease. It clears under the use of cycloplegics and other appropriate treatment.

IX. Non-Inflammatory Conditions Affecting the Posterior Corneal Surface.

588. Opacity Due to Tissue Contact.—When the posterior surface of the cornea is not washed by the aqueous humor, as it is in the normal eye, but an exudate or tissue is brought into apposition with it, the substance of the cornea becomes cloudy. To produce this effect, however, it is necessary that the apposition should be kept up for a pretty long time. Hence this sort of opacity is not generally found in ordinary hypopyon because the latter disappears too quickly, but is found in those more solid gray exudates which appear in the anterior chamber, particularly in scrofulous and syphilitic irido-cyclitis. Large deposits also on the posterior surface of the cornea, if they remain for a long time, usually leave behind them gray spots in the cornea. Another thing besides exudates that gives rise to this form of keratitis is the apposition of tissue to the back of the cornea, as occurs with protrusion of the iris, with cysts or other tumors of the iris that reach to the cornea, and with lenses that have prolapsed into the anterior chamber. The opacity of the cornea develops at the point where apposition took place, occurring, therefore, in the case of exudates, most frequently below. The surface of the cornea at this spot is dull, sometimes slightly uneven, and apparently gelatinous. The opacity is gray, and, after lasting a long time, becomes pretty dense, and is permeated by vessels which lie in the deep layers of the cornea. It never completely disappears, even after the cause has been removed.

The origin of the opacity is as follows: Contact of the cornea with foreign tissue alters the endothelium and perhaps also folds and detaches Descemet's membrane. Consequently aqueous is enabled to enter the cornea and produces opacification and degenerative changes there (§ 73).

589. Striate Opacity of the Cornea.—When we examine carefully an inflamed cornea with a magnifying glass, we often discover gray striæ in it. These may be short and irregular and run in the most diverse directions. This is commonly the case in keratitis profunda. At other times we see a system of parallel striæ—e.g., in the dense opacities that occur in parenchymatous keratitis and jut out from the margin of the

cornea and extending toward its centre. Again, striæ may occur that take a radiating direction, all emanating from a single point—e.g., from a corneal ulcer. The anatomical changes that give origin to the striæ are not always the same. It may be that cells, or fluid, thrust the fibres of the cornea apart and so pass on between them, taking a linear course. Such a thing can be effected artificially by injecting liquid through a puncture made in the cornea. In doing this we fill a system of parallel chinks (Bowman's tubes) which cross the successive corneal lamellæ at right angles.

Isolated, long, very delicate gray lines appear to be produced by turbid fluid filling one of the channels that lie in the substance of the cornea and lodge the nerves passing from the margin of the cornea to its centre.

Very frequently, however, striæ are produced not by exudation, but by wrinkling. This is pre-eminently the case with *traumatic striate opacity.* This condition which is often, though erroneously, called *striped keratitis,* is observed after incised wounds of the cornea, and most beautifully after the cataract operation. Within the first twenty-four hours after the operation gray striæ make their appearance in the cornea, which, starting from the wound, extend sometimes as far as the opposite margin of the cornea, and are always disposed perpendicularly to the length of the wound (Fig. 259). These striæ are particularly observable in those cases in which the lips of the wound have been somewhat contused, as, for example, those in which the delivery of the lens has been difficult. They generally pass off within the first eight days, and it is only when specially pronounced that they take several weeks to disappear. Such an opacity causes no symptoms of irritation, and does not cause the least disturbance of the healing of the wound. This proves that here we are not dealing with a real inflammation. Anatomical investigation has, in fact, shown that in these cases cellular infiltration is altogether wanting, and that there is simply a dilatation of the lymph spaces of the cornea, which are distended with fluid (Becker, Laqueur, Recklinghausen). The striate opacities themselves are referable to wrinkling of Descemet's membrane, which as a result of the incision near the corneal margin, has its tension relaxed in one direction but not in others. Byers would hence call this condition posterior corrugation of the cornea to distinguish it from the anterior corrugation found in lattice-shaped opacity (§ 599).

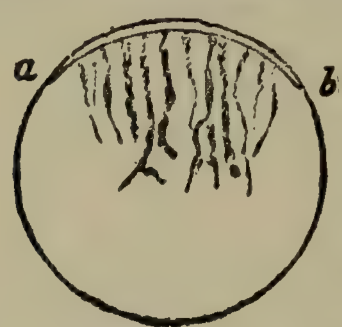


Fig. 259.—Striate Opacity of the Cornea after a Cataract Extraction.

The cicatrix, *a b*, left by the section, lies at the upper margin of the cornea.

A wrinkling of this sort may be the cause of some of the striate opacities that are observed in a true keratitis—e.g., the radiating streaks which are often seen in ulcus serpens extending out into the transparent cornea (Hess, Schirmer).

A similar striate opacity of the cornea is sometimes observed in cases of detachment of the retina which has been treated with the pressure bandage. The eye becomes suddenly very soft and the anterior chamber remarkably deep, and in the cornea fine gray striæ show themselves, which cross in different directions, so that the opacity looks like creased tissue paper. Here also, without doubt, folds in the cornea take part in producing this effect (Deutschmann, Nuel). Folds of Descemet's membrane are also seen in other conditions, e. g., after contusions of the cornea, and are then frequently associated with folds in Bowman's membrane (cf. §570).

Vesicles may also form on the posterior surface of the cornea (see page 561).

X. Injuries of the Cornea.

590. Foreign Bodies in the Cornea.—The penetration of foreign bodies into the superficial layers of the cornea is among the most common of accidents. Obviously the interpalpebral area of the cornea is the part that suffers most from injuries due to foreign bodies as from injuries in general. What is most frequently observed are small particles of iron in the cornea, particularly among mechanics of a certain sort, like locksmiths, black-

smiths, iron founders, etc. Particles of this sort, which fly off when iron is being hammered, are heated by the force of the blow so that they are thrown out as sparks. Thus they become oxidized into ferroso-ferric oxide, and under this form are found in the cornea. If the fragment of iron remains sticking in the cornea, it becomes surrounded very soon by a brown ring, because it impregnates the portions of the cornea in its immediate vicinity with iron (ferric hydrate) and so turns them brown (cf. § 602). Particles of coal are also frequently found in the cornea—for instance, in firemen or in people after a railroad journey—or of stone in stonecutters, stone-breakers, etc., or of powder or lead in gunshot injuries.

Much more infrequent, but also much more serious, are those cases in which a small foreign body has penetrated into the deep layers of the cornea.

Foreign bodies penetrating into the cornea should be removed as soon as possible (see § 940). If the foreign body is not removed in season, its expulsion by suppuration follows. An inflammatory infiltration forms about it, surrounding it in the form of a gray ring. Then the tissue of the cornea in this place breaks down, so that the foreign body becomes loose and ultimately falls out. The resulting ulcer generally becomes rapidly cleansed and heals, leaving a small opacity after it. This process of elimination takes place with marked symptoms of irritation, and especially with hyperæmia of the iris, or even with iritis, which latter makes itself evident by the formation of a hypopyon and of synechiæ. These symptoms may persist for several days after the foreign body has been eliminated or removed. Foreign bodies which are chemically indifferent, like particles of stone, coal, or lead are often tolerated by the cornea without producing inflammation and may become permanently incorporated in it. Powder grains, too, may become incorporated in the cornea, but not till after a rather long period, and in the meantime they cause considerable irritation.

For the irritation remaining after the removal or elimination of a foreign body, we use hot or cold applications, dionin etc., and if photophobia or iritis is present, atropine. Holocaine or acoine in oily solution or in ointment may be required to relieve pain, especially at night. Sometimes plain vaselin answers the same purpose. In many cases the patient is more comfortable if the eye is bandaged. For the treatment in cases of perforation or of deep infection, see § 860.

Oyster-shuckers' keratitis is a rather rare condition caused by the impact of fine bits of oyster shell upon the eye. It is marked by its acute onset, a small circumscribed, very white ulcer developing in the cornea (usually in its centre) within twenty-four hours. This is associated with considerable photophobia and irritation. The ulcer usually shows no tendency to spread, and, when small, heals without bad result. If a large area is involved or the cornea is perforated, the eye is usually lost by infection (Randolph).

591. Erosions of the Cornea.—Superficial excoriations of the cornea, which simply produce a loss of substance in the epithelial covering, are known as *erosions*. These are among the most frequent of injuries, such as one gives himself by scratching the eye with the finger nail, with a rough cloth, a stiff leaf or twig, etc. Such an injury is commonly accompanied by

pretty marked symptoms of irritation, such as photophobia, lacrimation, and especially by violent pain. Examination of the eye shows, besides the ciliary injection, a defect in the epithelium, forming an ulcer, the floor of which is perfectly transparent, so that it is only by taking the corneal reflex or using fluorescein that the loss of substance can be discovered. Healing generally takes place within a few days by a complete regeneration of the epithelium, starting from the edges of the epithelial defect; a permanent opacity does not remain. Quite a good deal of significance attaches to these traumatic erosions, from the fact that not infrequently they are the starting-point of an ordinary ulcer of the cornea or an ulcus serpens, especially if an opportunity is given for the production of infection. This latter is particularly apt to occur if there is present either a conjunctival trouble associated with abnormal conjunctival secretion, or a disease of the lacrimal sac.

It is worth remarking that sometimes *recurrences* of corneal erosion take place without any new injury having proceeded from them (Arlt). After the lesion has been to all appearances fully healed, marked symptoms of irritation set in suddenly several weeks or months afterward without known cause; and a loss of substance is again found upon the cornea in the epithelium at the site of the former injury. Such relapses may occur repeatedly. They have their cause probably in the fact that the regenerated epithelium is no longer firmly adherent to its bed (Szily), so that under the action of any insignificant cause it is again separated and cast off. This separation of the epithelium generally takes place in the form of a vesicle, which, however, ruptures so quickly that we do not get a sight of it, but only of the consequent loss of substance in the epithelium.

In this category also are to be reckoned those cases in which for months or even years after an erosion of the cornea, pain occurs when the eye is suddenly opened either in the night or on awakening in the morning. The pain passes off in a few minutes. Then if we see the patient during the day, there is either nothing at all to be discovered in the cornea objectively or at most there is found an extremely faint cloudiness which is situated in the corneal epithelium on the spot that was injured. Yet probably in this case also there are constantly recurring, very insignificant lesions of the corneal epithelium, which are produced, at the site of the former injury, by sudden opening of the lids.

Peters and Szily, Jr., in persons operated on for cataract and in eyes affected with dislocation of the lens, have observed erosions which recur very obstinately and often and are attended with great pain, and which they attribute to lesions of the corneal epithelium.

Erosions are best *treated* by applying a simple protective bandage which should be continued until the epithelium is completely regenerated. If there are marked accompanying symptoms of inflammation, which are not relieved by the bandage alone, we may instill atropine or homatropine—the latter in 2-per-cent solution. The recurrences require the same treatment, as long as a defect in the epithelium can be made out to exist. In obstinate recurrent cases the bandage may have to be applied to both eyes and kept on, day and night, for from one to two weeks. After complete restoration of the epithelial covering, it is advisable, in order to prevent further recurrences, to have the cornea massaged for some time with the yellow-precipitate ointment. For pain which recurs at night we direct the patient to introduce fatty substance (e. g., boric-acid ointment) into the conjunctival sac before retiring, and, besides, advise him in case he wakes during the night to open the eyes very cautiously—possibly not until he has first wet the margin of the lids. If, in spite of this, relapses recur we obtain solid union of the epithelium to the cornea and hence also a cure, if we scrape off the epithelium wherever it is but loosely adherent to the cornea and then paint this area with tincture of iodine or saturated aqueous solution of chlorine (Wyler).

592. Deeper Wounds of the Cornea.—The deeper wounds are usually either incised or lacerated wounds. Their margins soon after the infliction of the injury become cloudy and swollen through imbibition of fluid (tears or aqueous). In irregular, lacerated wounds, this may occur over a very great area. As the wounds heal, this cloudiness in large part disappears, although a dense opacity always remains along the line corresponding to the solution of continuity, and this opacity is very frequently associated with an irregular bulging of the whole cornea (giving rise to irregular astigmatism). Corneal wounds are particularly dangerous under two circumstances—i.e., when they are infected and when they perforate the cornea. In the former case a purulent keratitis develops which may give rise to extensive destruction of the cornea. In the latter case prolapse of the iris occurs, provided the wound is large enough. Moreover, the iris or the lens may be injured at the same time, and lastly, there exists, as in all perforating lesions of the eyeball, the danger of an inflammation of the deep parts of the eye, produced by infection and very frequently ending in the destruction of the organ.

Incised wounds of the cornea are usually linear and often rectilinear; lacerated wounds are often zigzag or stellate in shape. Clean incised wounds, e.g., those produced by operation, show no tendency to gape, become agglutinated very quickly (within a few minutes), and heal with scarcely any scar-tissue, so that there is left simply a gray line on the anterior and posterior surfaces of the cornea, the intermediate tissue being almost perfectly transparent. But corneal wounds do gape if the edges swell up, if the deeper parts (iris, lens, vitreous) get between the lips of the wound, or if the edges of the wound are irregularly shaped and overlap. In wounds of accidental origin the edges are rarely as smooth as in operation wounds and are probably always more contused. Hence they soak up tears and aqueous and so become clouded and swollen. The edges are thus raised and necessarily gape somewhat, so that they form a guttered ridge. In flap wounds the swelling may be so great that the flap is bent double.

The edges of a wound soon become agglutinated by fibrin which is later replaced by cicatricial tissue, derived from the corneal stroma and in the case of perforating wounds from the endothelium also. Bowman's and Descemet's membranes do not reunite. During the first few days the epithelium of the anterior surface of the cornea grows rapidly over the lips of the wound and down between them, this *involution of the epithelium* sometimes extending as far as the posterior layers of the cornea (Fig. 260). By reason of the permanent adhesion of the lips of the wound that takes place later, the intruding epithelium is gradually squeezed back from below up toward the surface, and the epithelial involution disappears. Sometimes, however, it is persistent. In that case the wound externally looks as though it had healed smoothly, while really only the posterior layers of the cornea are united by a firm cicatrix. Such scars may be ruptured by moderate pressure upon them; and this explains why it is that the scar left by a cataract operation sometimes—it may be years afterward—splits asunder on very slight provocation. If the gaping of a perforating wound extends clear down to the anterior chamber, the intruding epithelium may even get into the latter and produce deleterious changes there (§ 635).

As the wound heals, the cloudiness due to imbibition disappears and the ridgelike swelling flattens down, but there remains in the wound tract a white opacity produced by the cicatricial tissue. Sometimes also the curvature of the adjoining transparent portions of the cornea is altered by the cicatricial contraction so that a disturbance of sight is produced even when the wound does not lie in the pupillary area.

The *prognosis* of non-perforating is essentially better than that of perforating wounds, but in neither case can be made with certainty until

we are sure that infection has not taken place, i.e., not till several days after the injury. For the rest, the prognosis, as in the case of corneal ulcers, depends on the position and the extent of the lesion (cf. § 539).

The *treatment* of recent wounds of the cornea demands as its prime requisite that after dropping in atropine to combat any iritis that may exist, we apply a protective bandage, at the same time disinfecting the conjunctival sac, e. g., with collargol ointment. If we are dealing with a perforating wound of the cornea, the greatest possible quiet on the part of the patient (rest in bed) is requisite in order to bring about a speedy and solid closure of the wound. If the iris is prolapsed, it should be so excised, after carefully

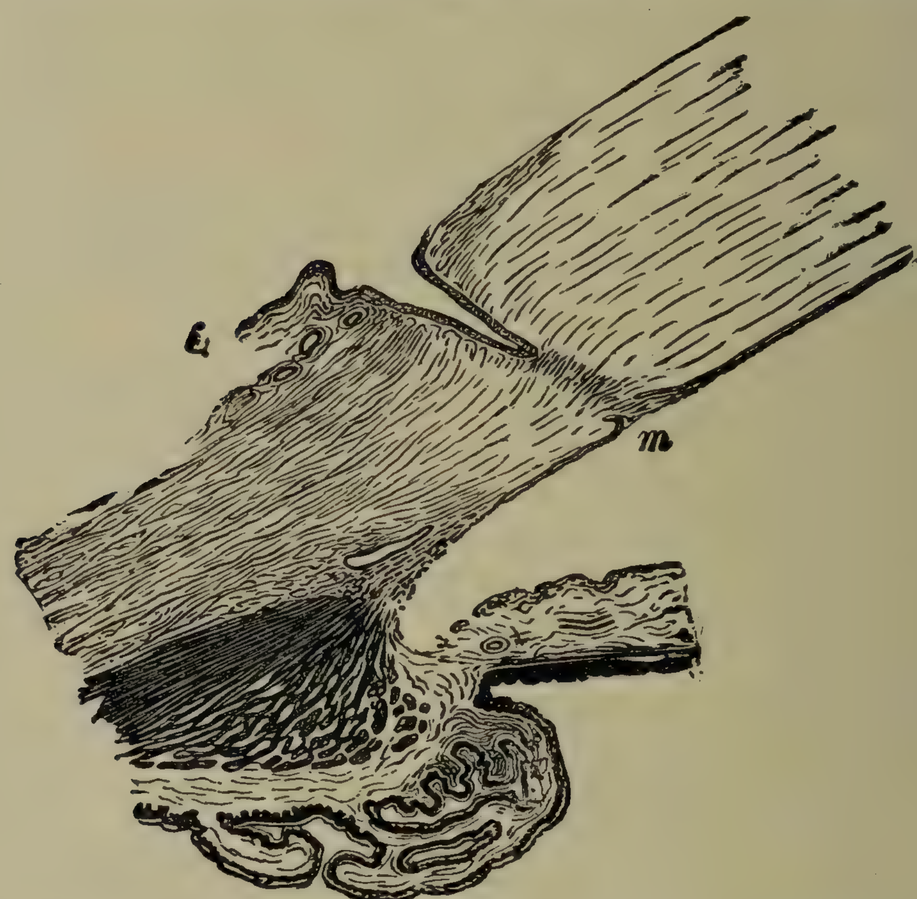


FIG. 260.—SCAR OF THE CORNEA THIRTEEN DAYS AFTER CATARACT OPERATION. Magnified 22 X 1

The section passed obliquely through the cornea and the margin of the limbus *L*. From both edges of the wound, peripheral (limbus) and central (cornea), the epithelium has grown down into the depth of the wound, which, therefore, in the living eye appeared under the form of a smooth chink running along the margin of the cornea. It is only in the deepest layers of the cornea that the lamellæ of the latter are united by recently formed scar tissue, which also pushes its way in between the cut edges of Descemet's membrane, *m*, which are curled forward. Since the operation was made with an iridectomy, there remains of the iris nothing but a stump, whose cut surface shows no trace of reaction.

separating it from the lips of the wound, so that no iris remains any longer incarcerated in the wound; according to just the same principle that holds good for prolapses of the iris of spontaneous origin. (For more precise particulars in regard to perforating wounds of the cornea, see § 853 et seq.).

To facilitate the healing of corneal wounds, Ramsay recommends that they be covered with a wafer of gelatin containing 10 per cent of collargol. In gaping flap wounds we may try to unite the edges of the wound with fine sutures, painting the sutured lips with 5-per-cent tincture of iodine or with Whitehead's varnish (a solution of 1 part of iodoform and 6 parts of compound tincture of benzoin in ether) (De Schweinitz). Unfortunately it sometimes happens that a purulent infiltration of the cornea or, in case the sutures have extended into the anterior chamber, an irido-cyclitis is started by the sutures. More favorable are those cases in which the gaping wound in the cornea is so placed that we can pass a suture through adjacent parts of the limbus and by drawing

them tightly together can approximate the lips of the wound. We may also cover in wounds of the cornea with conjunctival flaps (see § 917).

When the cornea is much lacerated the practical value of all these attempts is very questionable, even supposing that the lips of the wound heal well. The cornea remains permanently flattened and so opaque that only an inconsiderable residue of sight is left. At the same time the eye is for a long time prone to inflammation, and, besides, the danger of sympathetic ophthalmia is not altogether excluded, so that in these cases we should have done our patient a better service by enucleating at once.

593. Contusions of the Cornea.—Contusions produced by force applied to the cornea either directly or through the lids, often result in an extensive opacity which occupies the central portions of the cornea, and under the magnifying glass can be resolved into delicate gray striæ interlacing in different directions. As this opacity is situated in the intermediate and the deep layers of the cornea, it has been considered under the head of keratitis profunda (§ 581).

The striæ may in part be referable to wrinkling of Bowman's and Descemet's membranes (see §§ 570, 589), and be dependent upon the reduction in the intra-ocular pressure that often occurs after contusions of the cornea. The opacity sometimes reproduces exactly the shape of the body producing the injury. It often disappears very quickly. In other cases it lasts longer and assumes a ring shape (Caspar's *annular opacity*) (Salzmann).

After moderate contusing injuries, e.g. those caused by fragments of stone hitting the eye, a keratitis disciformis (§ 557) may develop.

Severe contusions of the cornea may cause its *rupture* (ruptura corneæ, § 854).

In new-born children there is sometimes found a uniformly dense, bluish-white opacity of the cornea which is produced by contusion of the cornea *during birth* (usually by the forceps). The opacity usually disappears altogether in the course of a few weeks.

594. Injuries of the Cornea by Caustic Agents and by Burns.—These occur simultaneously with the analogous injuries of the conjunctiva, and are produced by the same causes that these are (§ 499). In fact, in the case of such injuries of the eyeball, it is precisely the part which the cornea takes in the process that is a criterion for the prognosis—for the most harmful consequences of these injuries are the opacities which are left in the cornea. The corroded or burned cornea looks dull and opaque. The extent of the opacity depends upon the extent of the burn, but the intensity of the opacity depends upon the depth to which the corneal tissue has been destroyed. In light cases the color of the opaque cornea is gray, but in severe cases whitish. In the worst cases the cornea is all as white as porcelain, dry upon its surface, and quite insensitive; such a cornea is completely necrotic. In general it is not always easy to estimate from the character of the injury alone how deeply the destructive process has penetrated into the cornea, and hence caution is advisable in giving the prognosis.

The injury is generally followed by sharp pain. In some cases a temporary rise of tension may occur (§ 499). In the lightest cases, in which the injury has affected the epithelium only, the processes of extrusion and of healing occur very rapidly (as in the frequent cases of burns of the cornea produced by a curling iron). If the destructive process has made its way into the parenchyma of the cornea, a delimiting suppuration sets in, which leads to the separation of the eschar; the loss of substance thus produced

heals, leaving a permanent opacity. If the destructive process has at any point gone through the entire thickness of the cornea, perforation of the latter develops after the separation of the eschar. The iris then prolapses, and the resulting cicatrization unites the iris to the cornea (cicatrix of the cornea with anterior synechia). In a similar way adhesions often develop between the cornea and conjunctiva of the lids (symblepharon), provided that a loss of substance is present in the latter also. The treatment of injuries produced by burns and caustics has already received mention under the head of the analogous injuries of the conjunctiva (§ 499).

Corrosion of the cornea is most frequently produced by lime, which usually gets into the eye under the form of mortar. Corrosion with lime gives a bad prognosis inasmuch as the corneal opacity produced by it has no tendency to clear up. This behavior is explained by the microscopical findings in a corneal opacity of this sort, the latter being caused not, like ordinary opacities, by scar tissue, but by the deposition of small opaque particles which consist of calcium carbonate. For this reason this form of opacity has been called a *calcareous incrustation*. To clear the cornea by dissolving the calcium carbonate, Zur Nedden advises bathing the eye, after cocainization, several times a day for half an hour at a time in a 10-per-cent solution of neutral ammonium tartrate. The more recent the case, the better the outlook for a good result is said to be. Zur Nedden advises the same tratment for clearing up lead incrustations (§ 607).

Corrosion of the cornea may also be produced by bituminous dust, which causes corneal ulcers with iritis; dimethyl sulphate fumes, which cause cloudiness of the cornea; and substances (e.g., bits of indelible pencils) containing the basic aniline dyes, which cause ulceration of the cornea, cataract and even panophthalmitis (Stephenson). (See § 499).

XI. Dystrophies of the Cornea

595. The dystrophies are chronic disturbances of nutrition in the cornea which find their expression in the fact that very gradually an opacity develops in the cornea, which opacity steadily increases in the course of years. From inflammations in the cornea the dystrophies are distinguished —(1) by the absence of external evidences of inflammation, such as injection, pain, etc.; (2) by the continuously progressive course, while an inflammation after reaching its acme recedes; (3) by the anatomical finding which does not show an invasion of leucocytes into the cornea, but degenerative processes such as fatty degeneration (arcus senilis), the deposition of calcareous matter (zonular opacity of the cornea), hyaline degeneration (nodular opacity of the cornea), etc. From opacities in the narrower sense of the word (§ 605), i.e., opaque spots which remain after inflammations or injuries and which are stationary, the dystrophies are distinguished by their progressive course; in many cases, also, the epithelium over the opaque spot is dull, proving that a progressive process is present.

In the case of dystrophies we have to do with degenerative processes, whose cause is either old age (arcus senilis), or disordered nutrition of the eye (zonular opacity of the cornea, alterations in old scars—§ 608), or an hereditary tendency (nodular opacity of the cornea). In many cases the cause of the degeneration remains unknown.

596. Arcus Senilis (Gerontoxon[11]).—The physiological type of dystrophies is the arcus senilis which develops uniformly in the cornea in advanced life, sometimes even in middle life or even in youth, being then either the result of premature degeneration or caused by the continuous action of external irritants (pitch dust, etc.). It appears under the form of an opaque arc, first at the upper, soon after at the lower, margin of the cornea; finally the two arcs unite at the outer and inner side of the cornea to form a closed ring. The outer boundary of the arcus senilis is sharply defined, and is separated from the limbus by a strip of perfectly clear cornea; on its inner aspect, or the one turned toward the centre of the cornea, on the other hand, the cloudiness gradually shades away until it is lost in the transparent cornea. It almost never extends into the pupillary area, so that it practically never causes any interference with sight Under the loupe the opacity is resolved into very minute dots, and it exhibits a peculiar dull yellowish lustre.

The *arcus senilis* consists of very minute drops of fat which lie in the lamellæ of the cornea as far back as Descemet's membrane (Fig. 261). In addition to these there are

FIG. 261.—ARCUS SENILIS. (After Takayasa.) Magnified 75×1.

Bowman's membrane, *B*, is rendered cloudy by a deposit of extremely fine fat granules. This is still more the case with the lamellæ of the cornea, *C*, where the granules are larger and lie inside of the lamellæ, not in the interspaces between them, which in the drawing are left light. In the most superficial lamellæ the deposit of fat granules stops where Bowman's membrane ends and the conjunctiva of the limbus, *L*, begins. The deeper we go, the further toward the periphery do the granules extend, finally reaching even as far as the sclera, *S*.

often flakes of hyalin, the size of which varies from that of the finest dust to pretty large concretions. Sometimes also there is found a deposit of lime granules. The cause of the arcus senilis is supposed to be a senile atrophy of the limbus conjunctivæ with destruction of a part of the vascular loops contained in it. A pathological change which occurs very rarely in the arcus senilis is:

Senile Marginal Atrophy[12].—This is a very indolent process occurring in old persons. The arcus senilis becomes steadily broader, while the cornea in the area of the arcus gets thinner, so as to form a gutter-shaped depression, which ultimately, yielding to the intra-ocular pressure, becomes ectatic. The peripheral margin of the furrow is sloping, the central margin forms a steep wall. Along this wall some of the corneal lamellæ are cut off short, others become fibrillated and are continuous with the lax connective tissue which covers the floor of the furrow. The lamellæ of both the floor and wall of the furrow contain droplets of fat. Bowman's membrane ceases at some distance from the central wall. Thickenings and fissures are found in Descemet's membrane, but not till after the furrowed cornea has begun to bulge.

[11] From γέρων, an old man, and τόξον, bow.
[12] Synonym: peripheral furrowing keratitis (Schmidt-Rumpler), dystrophie marginale symétrique.

A similar, perhaps not identical condition, associated with slight inflammatory symptoms, occurs at times in younger people.

Opacities resembling the arcus senilis are found in conjunction with affections of the limbus—e.g., with the growths of spring catarrh, with small neoplasms, and sometimes with a large pinguecula.

597. Pannus Degenerativus (Baas).—This condition, also called lamellar deposition, represents an intermediate stage between inflammation and dystrophy, since it is attended by the formation of new tissue. It attacks eyes which are blind or at any rate badly diseased, especially those affected with glaucoma or irido-cyclitis. In such eyes the cornea is already dull, opaque and may be vesiculated. The chief additional evidence that pannus degenerativus produces is the formation of large, quite resistant, tremulous, blebs (so-called *keratitis bullosa*), the walls of which consist not of epithelium but of a layer of connective tissue (Fig. 262). These blebs last several days

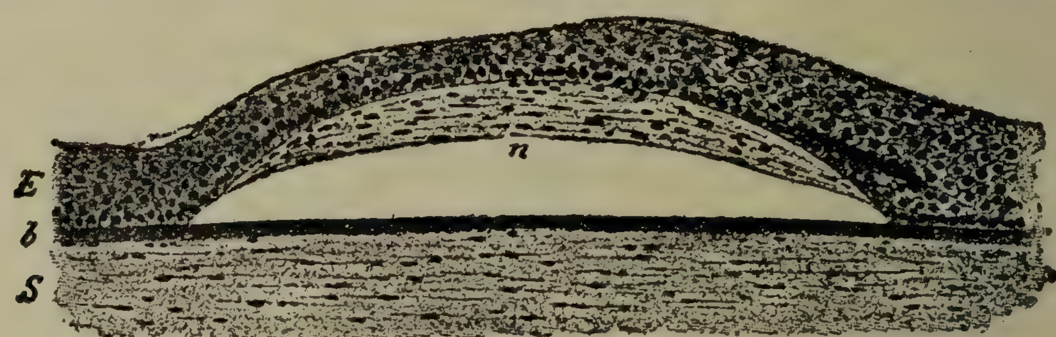


Fig. 262.—Keratitis Bullosa. Magnified 92 X1.

The figure is taken from an eye rendered blind by irido-cyclitis. Bowman's membrane, *b*, covering the stroma of the cornea, *S*, is stained dark because it is filled with lime granules corresponding to the zonular opacity of the cornea which is present (§ 598). The epithelium, *E*, is detached over the area of half a millimetre. At the highest point of the bulla it is thin, and on its posterior surface lies the new formed connective tissue, *n*.

before they rupture and in the meantime are accompanied by violent inflammatory symptoms. They show a great tendency to recur, always in the same spot, and each recurrence is marked by renewed symptoms of irritation.

Pannus degenerativus begins with the formation of small gaps in Bowman's membrane. These were first described by Wintersteiner as ruptures of the membrane, but probably represent the places when the corneal nerves go through. They become filled with a fibrous connective tissue, containing numerous nuclei. This tissue, overflowing the margin of the gap, pushes the epithelium before it, and spreads further and further between the epithelium and Bowman's membrane. At first this tissue occurs in isolated patches and because it is derived from the non-vascular stroma of the cornea is itself non-vascular; later the patches may become confluent and be vascularized from the limbus. Liquid collects between Bowman's membrane and this connective-tissue layer, so that the latter with the epithelium is lifted up to form the wall of the bleb; Bowman's membrane itself remaining in place.

Sclerosis of the Cornea (Meller) is but a later development of pannus degenerativus. The deposit is of considerable amount, has few nuclei, and is of a lamellar structure. The cornea is opaque white so as to look like the sclera, while in ordinary pannus degenerativus there is only a moderate opacity. Sclerosis is not associated with glaucoma and irido-cyclitis, but occurs after repeated operations. It appears to be related to dystrophia epithelialis.

The treatment of degenerative pannus is confined to the alleviation of the inflammatory symptoms which are associated with the formation of the blebs. Iridectomy, preventing a recurrent increase of tension, may prevent

also a recurrence of the blebs. We may also remove the blebs and either paint the base with tincture of iodine or cauterize it. If they keep returning in spite of this we may have to enucleate the eye in order to give the patient ease.

598. Zonular Opacity[13].—This is the most frequent of the pathological type of dystrophies. It forms a gray stripe from 3 to 5 mm. broad, which passes straight across the cornea, a little below its centre. It develops with extreme slowness, occupying years in its progress, the first parts to appear being the two terminal points of the opaque stripe—that is, the portions of the opacity lying nearest the outer and inner margins of the cornea. These points are always separated from the margin of the cornea by a narrow, transparent zone. Starting from them the opacity gradually pushes its way toward the middle line, where the two parts of it unite, and thus close in the opaque zone which covers the lower half of the cornea. This zone is, accordingly, broadest and most opaque at its two extremities, these being its oldest parts (Fig. 263). On examining it pretty closely, especially with a magnifying glass, we ascertain that the opacity, which has a sharply defined outline on all sides, is composed of minute white or gray dots which lie quite superficially in the epithelium or directly beneath it; hence we usually find the surface of the cornea over the opacity roughened like shagreen or covered with minute prominences. Often in the midst of the opacity we see rounded or irregular areas which are quite clear (as shown in Fig. 263), or the opacity is traversed by fissures and chinks; and portions of the opacity may actually drop off altogether.

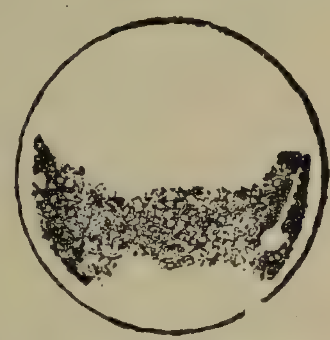


Fig. 263—Zonular Opacity of the Cornea.

Zonular opacity of the cornea generally develops in eyes which have nearly or quite lost their sight in consequence of some intra-ocular affection (irido-cyclitis, glaucoma), and in this case it is practically of little significance. It is only very rarely (and then only in elderly people) that we encounter it in eyes which are otherwise perfectly sound, so that here the corneal opacity itself is the sole cause of the disturbance of vision (senile zonular opacity).

The anatomical changes which underlie zonular opacity of the cornea consist in the deposition of lime in the form of very minute granules in Bowman's membrane (*b*, Fig. 262; *h*, Fig. 264), which thus becomes opaque, white, rigid, and brittle. From examination with the slit lamp it seems likely that the process of calcification is preceded by a degenerative condition of the walls of the interlamellar canals in the subjacent cornea (Koeppe). In the spots where the calcification is far advanced we ordinarily find just as in degenerative pannus, new-formed connective tissue (*g*, Fig. 264) on Bowman's membrane, between the latter and the epithelium. Owing to the presence of this tissue the surface of the epithelium, *e*, becomes irregular, a thing which explains the granular character of the corneal surface over the opacity. Acting in the opposite direction the connective tissue pushes Bowman's membrane backward, causing breaks in it and displacement of the broken portions. In advanced cases minute granules of lime also make their appearance in the lamellæ of the cornea lying beneath Bowman's membrane.

Zonular opacity of the cornea depends upon a disturbance of nutrition, caused by a lessened ability on the part of the cornea to withstand external injurious influences. The

[13] Synonyms: Ribbon shaped opacity of the cornea, calcareous band of the cornea.

position and extent of the opacity correspond to the palpebral zone of the cornea—i.e., that part of it which lies exposed in the palpebral fissure even when the latter is but slightly opened. Since this form of opacity affects corneæ which have generally been made insensitive already and often opaque, too, by some antecedent disease, it must be assumed that it occurs because these corneæ are unable any longer to withstand properly the external injurious influences to which they are subjected in the region of the palpebral fissure. If such influences exert their effect for a very long time, even healthy corneæ may react to them by the production of a zonular opacity. Thus it has been found in hat makers, into whose eyes particles of hair were constantly flying from the hare skins that they were engaged in cutting (Topolanski), in a physician who had blown calomel into his eyes every day for twelve years (Fuchs), and in persons continuously exposed to mercurial vapor (Clarke). According to Leber, one of the injurious influences that affect the palpebral region of the cornea is evaporation: the nutrient fluid of the cornea

Fig. 264.—Zonular Opacity of the Cornea in an Atrophic Eyeball. Magnified 68×1.

Bowman's membrane, *h*, stands out because of its dark coloration which it owes to minute granules of lime deposited in it. At the left end of the drawing the deposit is slight, but increases to the right to such an extent that the individual granules can no longer be distinguished. At the same time the membrane becomes thinner and on its posterior surface uneven. Where the calcification of the membrane is the greatest, a layer of compact connective tissue, *g*, is found lying upon the latter. At the point where this connective tissue is most pronounced it pushes Bowman's membrane backward against the parenchyma of the cornea, *H*, and has caused a rupture of the brittle membrane. Through the point of rupture, connective tissue goes down into the deeper parts. The epithelium, *e*, is normal only at the spot where the calcification of Bowman's membrane is as yet slight; further along it is raised by the connective tissue and is of unequal thickness. Cf. Fig. 262.

being supposed to be richer in lime salts in cases of zonular opacity, and these salts being precipitated under the influences of evaporation.

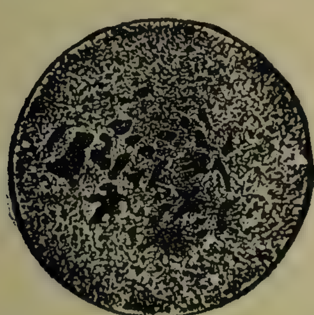
The zonular opacity being superficially placed can readily be removed by scraping off the epithelium and the cloudy corneal layers immediately subjacent (abrasio corneæ). There is, of course, no object in doing this except in the cases where, as in the senile form, we have to do with an eye that but for the opacity would be serviceable for vision. In such cases it may happen that portions of the opacity at its centre detach of themselves, so that there is a spontaneous restoration of sight.

Internal Secondary Zonular Opacity.—Under this name Koeppe describes a condition resembling in appearance and character the ordinary zonular opacity, but seated in the deepest layers of the cornea. It occurs in cases of long-continued chronic iridocyclitis. New-formed anastomotic vessels occupy the deepest layers of the cornea. The endothelium remains intact.

599. Nodular Opacity of the Cornea (Groenouw). This dystrophy always affects both eyes and occurs mainly in males. In the cornea are gray spots of rounded or irregular shape. The largest occupy the area of the pupil and sometimes coalesce to form quite large irregular figures (Figs. 265 and 266). The smaller spots lie in the periphery of the cornea and are sometimes arranged in the form of an irregular circle. The spots are quite superficial, and the cornea shows elevations corresponding to them. The unevenness of the corneal surface thus produced contributes not a little to the impairment of the sight. Between the spots the cornea shows a faint uniform cloudiness. The spots are produced by the deposition of substances insoluble in the body fluids (an acidophile, often laminated substance beneath the epithelium and a basophile granular substance in the cornea itself). Groenouw found hyalin in the cornea; Chevallereau, crystalline agglomerations of sodium urate.

In the allied **Lattice-shaped Opacity** of the cornea there is present together with the spots a network of minute lines in the cornea (Haab, Dimmer). These lines, Byers regards as representing corrugations of Bowman's membrane due to slight chronic hypotony. He calls the condition anterior corrugation of the cornea to distinguish it from posterior corrugation or striate opacity (see § 589). In its treatment he advises exercise and open-air life to raise the intra-ocular tension. Fehr regards this disease and nodular opacity as varieties of the same condition. Both forms of dystrophy usually begin in youth and last for life, slight inflammatory symptoms making their appearance now and then, while the spots themselves very gradually increase. They frequently occur in several members of the same family.

600. Dystrophia Epithelialis Corneæ.—In this there is a diffuse opacity of the cornea which is most intense in the pupillary area, and which without any sharp line of demarcation fades off into the transparent margin of the cornea. The greatest change is found in the epithelium, which is coarsely roughened and looks swollen, and in advanced cases also regularly shows vesicular elevations. These last, because they are transparent, appear when seen against the pupil as black spots, standing out upon the opacity. This proves that the opacity is situated for the most part in the epithelium; but if we scrape the latter away, we find in the uppermost layers of the corneal parenchyma a faint gray



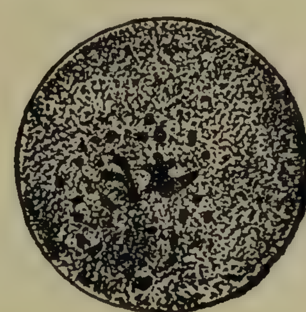


FIG. 265. NODULAR OPACITY OF THE CORNEA. FIG. 266.

opacity which can be resolved by means of a strong glass into extremely minute gray dots. The diffuse appearance of the opacity, the marked alteration of the epithelium and the simultaneously present insensitiveness of the cornea, correspond to the picture of a marked glaucomatous opacity of the cornea. However, all other evidences of glaucoma are wanting, except in the rare cases in which a secondary increase of tension is superadded to the change in the cornea. The disease attacks elderly people, and sometimes only one, sometimes both eyes. It begins with slight symptoms of irritation, or without any irritation at all, so that in the latter case the disturbance of vision is the only thing that calls the patient's attention to his trouble. The opacity steadily increases in the course of years, so that ultimately even counting the fingers is scarcely possible. The cause of the disease and also an effective treatment of it are unknown. In one case of the translator's, arsenic seemed to help.

601. Other Forms of Dystrophy.—Other conditions of degeneration of the corneal stroma include the deposition of *urates* (Uhthoff), *fatty* infiltration (Tertsch), *calcification* of the stroma without involvement of Bowman's membrane (Dystrophia calcarea of Axenfeld), *hyaline* degeneration (Dystrophia hyaliniformis lamellosa of Koeppe), and some other forms whose nature is uncertain.

For degenerative processes occurring in corneal opacities see § 608.

602. Conditions Allied to Dystrophy.—Clinically resembling dystrophies, but differing in that they either remain stationary or actually disappear, if the cause is removed, are the changes produced in the cornea by the continuous action of various substances. Such changes include *lime* and *lead* incrustation (see § 607); *argyrosis* (§ 453), which, however, affects the cornea only in particularly bad cases, causing then a grayish opacity; and the opacity from *nitronaphthalin* occurring in those who are engaged in the manufacture or application of aniline dyes, and who are exposed for years to the emanations from these substances. In this case there develops gradually a pretty deep, smoky-gray or brownish opacity which occupies chiefly that portion of the cornea which lies in the palpebral fissure; the surface of the cornea over the opaque parts is coarsely roughened. Inflammatory symptoms are absent. The opacity is situated in the epithelium and the

most superficial layers of the cornea itself and, provided work in the manufactory is given up, disappear after rather a long time—sometimes after over a year—has elapsed. Impregnation of the cornea with iron (*siderosis corneæ*) occurs either when a fragment of iron in the interior of the eye has produced a general siderosis or when a superficially impacted piece of iron after removal has left a rust ring on the surface of the cornea. The slit lamp shows that in either case the brown coloration is due, not to the deposition of iron particles, but to a uniform tinging of the interlamellar spaces of the stroma (Koeppe).

603. Pigmentation of the Cornea.—Apart from the pigmentation just described (§ 602) pigmentation of the most posterior layers of the cornea occasionally develops when the anterior chamber has been obliterated for some time and meanwhile pigment has got into the cornea *from the apposed iris.* Again, by a way analogous to that in which pigmented precipitates are formed, iris pigment may be cast on the posterior surface of the cornea in irido-cyclitis and when operations are performed in the anterior chamber.

A dark pigmentation of the cornea extending from the limbus, may occur in *Addison's disease* (Meesmann).

A peculiar sort of opacity and coloration (*blood-staining*) develops when the anterior chamber remains for some time filled with blood. Then the cornea, because it takes up the coloring matter of the blood, assumes an intense reddish, brownish, or brownish-green color, and at the same time becomes so opaque that the parts beneath can no longer be distinguished through it. It gradually clears again, beginning at the edge, but only in rare cases becomes once more perfectly transparent, and then only after months or years have elapsed. In that stage in which the central brown portion is surrounded by a narrow marginal zone which has regained its transparency, it looks as if a brown-colored crystalline lens had prolapsed into the anterior chamber and was lying behind the cornea (Vossius, Treacher Collins, etc.). With the slit lamp the staining appears to affect mainly the interlamellar spaces, in part also the lamellæ themselves, and along with the diffuse staining, granules and larger golden-yellow particles may be seen (Koeppe). In most cases the microscope shows in the lamellæ of the cornea highly refractive granules looking like large cocci or short rods, and giving the same color reactions as the erythrocytes. In some cases granules of hæmosiderin are found (cf. § 608).

For the congenital melanosis corneæ see § 621.

604. Pitting of the Cornea.—Allied to Gaule's furrows in trigeminus paralysis (§ 560), are the pits (dimples) of the cornea described by Fuchs. They are flat, dish-shaped depressions with distinct but rounded borders which appear at the margin of the cornea, generally on the temporal side. The depressions are usually elliptical with the long axis parallel with the margin of the cornea. They are from 1 to 2 mm. broad, 2.5 to 3.5 mm. long, and at most 0.5 mm. deep. Sometimes they are smaller and may be circular in shape. The floor shows a light cloudiness having a sort of silvery lustre and looks xerotic. The sensitiveness of the cornea in the area of the depression is somewhat diminished. The depression lasts for a few hours only, at most till the following day, and disappears without leaving the slightest trace. Usually these depressions are consequent upon some swelling of the adjoining parts (conjunctiva or episcleral tissue) irrespective of whether this swelling is produced by inflammation, hæmorrhage or simply a subconjunctival injection. By making a series of subconjunctival injections we may produce them experimentally. They are evidently due to loss of water and are probably dependent on injury of one of the nerves which enter the cornea at its margin. They have no clinical significance and require no treatment.

XII. Opacities of the Cornea

605. Inflammatory Opacities.—Opacity of the cornea is a constant accompaniment of every inflammation of the latter. This recent inflammatory opacity is of a changeable nature, increasing or diminishing according to the course of the inflammation. From this variety we must distinguish those opacities which represent either the residua of an inflamma-

tion that has run its course, or the results of an injury. Such opacities are stable in character, are unattended by inflammatory symptoms, and are covered by a smooth epithelium. These permanent opacities, of which alone we shall treat here, we call opacities of the cornea in the narrower sense of the word. They are by far the most frequent cause of poor sight, and hence have a particular claim upon the interest of the physician.

They must be distinguished from the **Non-inflammatory Opacities**, which are always transient. These include the *traumatic striate opacity* (see § 589) and the *pressure opacity* which is produced by elevation of the intra-ocular tension. It is diffuse, smoky opacity, which is most marked in the centre of the cornea and gradually diminishes toward its margin. That it is not of inflammatory nature is proved by the fact that, after the disappearance of the rise in tension, it very soon—often in less than an hour—vanishes completely, which would not be possible if it depended upon an inflammatory infiltration of the cornea. In fact, the pressure opacity is simply an œdema of the cornea, which is situated mainly in the epithelium, and readily subsides (see Fig.375).

Permanent opacities of the cornea are generally the result of an ulcerative keratitis. In this case the tissue of the cornea, which has been destroyed by suppuration, is replaced by cicatricial tissue, and the opacities thereby produced are corneal cicatrices in the true sense of the word. In this category also are to be counted most opacities which remain after injuries. The cornea may also have opacities remaining after a non-suppurative keratitis, either because its tissue has been so altered by the deposition of a former exudate that it does not regain its physiological transparency even after the exudate has vanished, or because new tissue is formed, or because of degenerative changes. Opacities that are situated in the epithelium only are comparatively rare, occurring, for instance, in those cases in which the epithelium as a result of constant mechanical irritation—in trichiasis—becomes thickened and hence opaque.

606. Appearance.—The appearance of opacities of the cornea varies according to their *age*. An opacity whether due to inflammation or injury tends to clear as time goes on, this tendency being the more marked, the younger the subject (cf. § 534). The appearance also varies with the intensity of the opacity. Faint opacities appear as translucent bluish-white spots with outlines altogether hazy—*maculæ* or *nubeculæ corneæ*. Some opacities are so faint as to be quite invisible, even by oblique illumination, and yet, on account of their diffuseness, may cause considerable reduction in sight. These can be seen with the ophthalmoscope, if we examine them by the direct method, using a + 20 D lens. Dense opacities are grayish-white or pure white (*leucomata*), and are usually pretty sharply outlined; moreover, in the beginning they are traversed by vessels which afterward become fewer or disappear altogether. The cornea then looks like ground glass, permeated with striæ, which represent the remains of former vascularization (Butler). (Cf. § 608.) The surface of the opacity generally lies on a level with the adjacent healthy cornea, especially, if the opacities are small, although elevation or depression of the surface of the cornea at the site of the scar is also observed. Elevation of the surface is commonly the result of an *ectasis* of the scar. More rarely it is caused by excessive devel-

opment of cicatricial tissue, or by thickening of the epithelium upon the surface of the scar. Depression of the surface of the cornea at the site of the scar, occurs most frequently in the case of small scars from the incomplete filling up of the ulcer with cicatricial tissue (*facet of the cornea*). In the case of larger scars that have developed in consequence of extensive perforation of the cornea, or more rarely as a result of a severe non-purulent keratitis, a flattening of the entire cornea may follow from the retraction of the cicatricial tissue (*applanatio corneæ*). This is especially likely to occur if a plastic irido-cyclitis had accompanied the inflammation of the cornea; for, on account of this irido-cyclitis, extensive membranous exudates are deposited in the interior of the eye, which by their contraction diminish the intra-ocular pressure. and so favor flattening of the cornea.

607. Varieties of Inflammatory Opacities.—From the form and position of corneal opacities we may often gather an impression as to the variety of keratitis to which they owe their *origin*. Thus:

(*a*) Maculæ of the cornea originate from small corneal ulcers. They most frequently develop in childhood as a consequence of phlyctenular conjunctivitis, and in that case are often distinguished by being situated on the margin of the cornea. Quite characteristic opacities are the elongated ones that are left by a vascular fasciculus. These after they have lasted a long time clear up in the portion that adjoins the margin of the cornea.

(*b*) Opacities which are faint and diffused, but which are nevertheless spread over the greater part of the cornea, are mostly the result of pannus or of parenchymatous keratitis. Opacities resulting from pannus are situated superficially, while those due to parenchymatous keratitis are situated in the depth of the cornea, and when examined with the magnifying glass disclose, even years after the inflammation has ceased, the presence of deep-seated vessels (Hirschberg).

(*c*) Extensive, tendinous-looking opacities, without incarceration of the iris, in which chalky-white dots are often visible, are observed after particularly severe cases of parenchymatous keratitis. Similar white dots occur sometimes in the opacities due to pannus (§ 469); also in those resulting from corrosion by lime, in this case depending upon imbedded calcareous particles (lime incrustation). Finally, scars with incrustation of lead, produced by dropping lead water into an eye in which the corneal epithelium has been broken by ulceration or otherwise, are also distinguished by a sharply circumscribed, extremely white opacity.

(*d*) Marginal, crescentic or arcuate opacities are the consequence of catarrhal ulcers or of keratitis marginalis profunda; they should not be confounded with an arcus senilis.

(*e*) Marginal scars with incarceration of the iris form after perforating ulcers in phlyctenular conjunctivitis. They are round, often consisting of a thinner, dark centre (the incarcerated iris) surrounded by a white cicatricial ring (Fig. 268). They lie so far peripherally as often to extend into the limbus, and on account of this peripheral situation are associated with a particularly marked displacement of the pupil (Fig. 239).

(*f*) Large, dense scars with inclusion of the iris, which often occupy the whole cornea except a narrow rim about the margin, are most frequently produced by an ulcus serpens or by gonorrhœal conjunctivitis. The same sort of extensive cicatrices also occur after keratomalacia, diphtheria, and burns; in the last two cases scars upon the conjunctiva are never wanting, and conduce to the correct diagnosis.

(*g*) Sharply defined punctate or striate scars are the result of traumatism, whether effected by accident or by design (operation).

(*h*) Dense, white scars, which occupy the lowermost part of the cornea and terminate above in an almost horizontal border, are caused by keratitis e lagophthalmo. Sometimes we see men in whom such scars are present in both eyes. In this case the

scars are usually the consequence of some severe disease, in which a condition of stupor and a resulting imperfect closure of the lids were present and lasted for some time.

(*i*) Opacities in the lowermost part of the cornea having the shape of a triangle with its apex directed upward are the result of a parenchymatous keratitis which, contrary to rule, has become localized in the lower half of the cornea, or they are due to the deposition of an exudate upon the posterior corneal surface.

(*j*) Small, bluish-white opacities which are situated at the margin of the cornea and project into the transparent part of it under the form of obtuse-angled triangles, are the residua of a sclerosing keratitis.

608. Metamorphoses of Corneal Opacities.—1. *Formation of Clear Striæ.*—It often happens that faint scars dating from childhood no longer appear in adult life under the guise of a continuous opacity, but are traversed by clear striæ, interlacing in all directions and thus dividing the opacity into small separate areas; in other cases the transparent striæ follow the branching of vessels which had been present in the scar when it was still recent (Fig. 267). This peculiar aspect of an opacity always indicates that the latter has lasted a very long time. The explanation of it probably is that in the interstitial growth of the cornea, new-formed transparent fibres develop between the old opaque ones.

2. *Deposition of Hæmosiderin.*—In maculæ we not infrequently find a quite diffuse yellow or yellowish-brown coloration, usually surrounding an opaque yellowish brown streak or spot. This coloration is regarded as being due to a deposition of hæmosiderin, but how the hæmosiderin gets into the scar has not been satisfactorily explained. Salzmann has seen this form of degeneration in scars which were from one and a half to two years old and certainly contained no blood-vessels. A like tinging has been found in keratoconus (§ 613) and also as a symptom of a nervous disease which bears a great resemblance to multiple sclerosis. In this disease the coloration starts from the margin of the cornea (Kayser, Fleischer, Salus). Recently Stähli has found in the lower part of the normal cornea of elderly people minute brown lines, which, may consist of hæmosiderin.

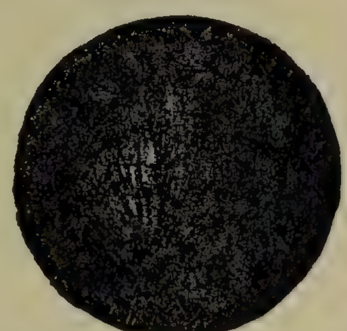


Fig. 267.—Scar of the Cornea with Striate Clearing.

3. *Hyalin Degeneration.*—In old dense scars or staphylomata of the cornea, citron or golden yellow spots often occur which are caused by deposition of hyalin. These spots appear under the form of very refractive amorphous flakes of golden yellow color in the scar tissue itself and in the epithelium over the scar. In addition, calcification is often present, either in the remains of Bowman's membrane, or in the scar tissue itself or in the flakes of hyalin. One might confound these spots with a purulent infiltration, but the latter is never of such a pure and intense yellow color. One consequence of this degeneration is a spontaneous necrosis affecting large portions of the scar tissue which are then exfoliated by a demarcating inflammatory process (*sequestrating cicatricial keratitis*). This process goes on slowly and insidiously, and leaves a loss of substance (*atheromatous ulcer*) which differs from other ulcers in that it is always covered or filled with epithelium (§ 534). Occasionally it happens that pathogenic germs effect an entrance and produce a purulent inflammation in the neighborhood of the sequestrum, an inflammation which if there is an incarceration of the iris, is likely to produce an endophthalmitis.

609. Complications and Sequelæ.—With many scars, *incarceration of the iris* occurs. This is a proof that there has been an antecedent perforation of the cornea; hence such scars are always very opaque. It is important to determine in any special case whether a scar of the cornea is or is not connected with the iris, since an incarceration of the iris may entail serious consequences. We recognize the presence of such an anterior synechia by the displacement of the pupil toward the site of the incarceration, and furthermore by the unequal depth of the anterior chamber, which is always shal-

lower near the place where the iris is adherent. In many cases, also, the dark color of the cicatrix gives evidence of the incarcerated iris, the pigment of which shows through the cicatricial tissue (Fig. 268).

The union between iris and cicatrix is often confined to quite a small spot, so small, sometimes, that only a minute filament rises from the iris and passes over to the scar in the cornea. At other times, on the contrary, broad adhesions exist, and there may even be an incarceration of the entire pupillary margin of the iris in the cicatrix (Figs. 274 and 275).

In rare cases an anterior synechia exists *without a preliminary perforation* of the cornea. This may occur in any condition in which the iris either by traction from in front (contracting exudates) or pressure from behind, is kept in contact with the cornea for some time. It is found for example, in keratitis pustuliformis profunda, in long-standing abolition of the anterior chamber, and in protrusion of the iris from seclusio pupillæ. The iris in such cases becomes agglutinated to the cornea in spots, and if it

Fig. 268.

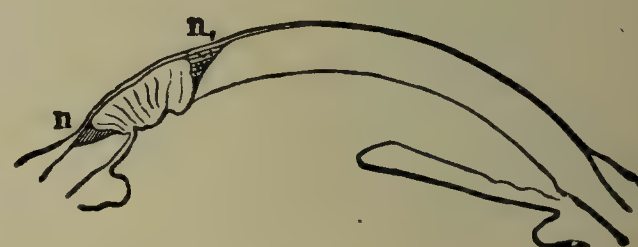


Fig. 269.

Fig. 268.—Scar of the Cornea with Anterior Synechia. Produced by an ulcer in the course of a phlyctenular conjunctivitis. The dark central portion of the scar is surrounded by a white ring. The pupil is displaced toward the scar.

Fig. 269.—Section Through a Scar with Incarceration of the Iris. The iris runs from the ciliary body to the scar, and in this part of its course is greatly thinned and is adherent to the posterior surface of the cornea, so that the sinus of the chamber is obliterated, a condition which may subsequently lead to increase of tension. In the scar itself the iris is thick and puckered. On the posterior surface it is covered with the retinal pigment layer, on its anterior merely with epithelium, so that if the latter were removed, the tissue of the iris would be directly exposed. Scar tissue, n n_1, is found only between the iris and the edge of what was formerly the perforation opening. It fills up the space between the two, which is triangular in cross section. It is this scar tissue that corresponds to the white ring which in Fig. 268 surrounds the dark incarcerated iris.

afterward, either spontaneously or as a result of an iridectomy, returns to its normal position, these agglutinated parts of it remain attached to the cornea. We then either find the iris extensively adherent to the cornea, or one or two tags arise from it, whose apices are inserted into the posterior corneal surface.

Anterior synechiæ in many cases entail no bad results, but often they give rise to two conditions, which, unless averted in time, inevitably lead to total blindness. One of these is *secondary glaucoma.* The conditions under which this arises may be seen from the following schematic statement:

Scars without incarceration of the iris	Flat: no increase of tension.	
	Ectatic (keratectasia): increase of tension frequent.	
Scars with incarceration of the iris	Flat	Pupillary margin partly free: no increase of tension.
		Pupillary margin totally incarcerated: increase of tension always occurs.
	Ectatic (staphyloma): increase of tension always occurs.	

In the two cases last given, increase of tension is absent if there is a counteracting influence afforded by other changes which act to diminish the tension (e.g., fistula of the cornea, shrinking exudates in the vitreous).

Blindness may also result from a *secondary endophthalmitis* (late infection, §§ 97, 756). This is the more likely to develop if the cicatricial layer over the incarcerated iris is thin and the scar is markedly ectatic, because then the protective cover of epithelium is more readily injured. According to some, secondary infection may also occur without incarceration of the iris. Thus if the scar is associated with a defect of Descemet's membrane even a superficial purulent infiltration in the tissue over this defect may give rise to a peculiarly rapid destructive inflammation of the eye.

610. Disturbance of Vision Produced by Opacities of the Cornea.—Every opacity which falls wholly or in part within the pupillary region of the cornea results in disturbance of vision; for the sum of the incident rays at the site of the opacity, instead of being all permitted to pass through the cornea, is divided into two parts; one part is absorbed by the scar or is reflected off from it; the other part penetrates through it into the eye. The relation between these two parts depends upon the density of the opacity; the denser it is, the more numerous are the reflected, the less numerous the transmitted, rays. Hence the cicatrix does harm by cutting off light. To be sure, this factor becomes a matter of serious consideration only in very dense opacities since we are able to see with very much less light than we usually get. The real cause of the disturbance of vision produced by corneal opacities is not, therefore, the cutting off of light, but rather the *scattering* (*diffusion*) of light (see § 86). For, rays passing through a turbid medium, such as a cloudy cornea, are not regularly refracted, but are scattered in all directions, just as if they emanated from the turbid stratum itself. Thus, when a physicist requires a uniform illumination, he makes the light from a luminous body pass through a ground glass plate or through oiled paper, substances which may then be considered to act as self-luminous bodies themselves. The diffusion of the rays is the more complete the denser the opacity.

With respect to the density and the extent of the opacity, the following cases are possible: 1. A *dense* opacity occupies the whole pupillary region of the cornea. Then all the light that issues through the scar is diffused; no image at all of external objects is formed upon the retina, and therefore there cannot be qualitative but only quantitative vision. 2. A *faint* opacity covers the whole pupillary area. In this case the diffusion of light is not complete. A part of the rays is refracted, although not quite regularly; another part is diffused. Hence there are retinal images formed, which are, however, indistinct; and, besides, there is much diffused light. 3. *Only a part* of the pupillary region is taken up by the opacity, while the remaining part is normally transparent. Then distinct retinal images are produced by means of the latter clear portion, but at the same time much diffused light is thrown into the interior of the eye by means of the clouded portion. Hence in this case, also, vision is disturbed, and that by the dazzling which the diffuse light causes.

An additional cause of disturbance of vision is the irregular curvature of the corneal surface, which is so frequently present at the site of opacity. There is thus produced an *irregular astigmatism* (see § 207). If the opacity of the cornea is attended with a flattening of the surface, as in facets of the cornea, this spot refracts less strongly and is hypermetropic; if the cornea is bulged forward at the site of the opacity, excessive refraction, and hence

myopia, are produced. In ectatic cicatrices of the cornea the abnormality of curvature is not confined to the scar, but extends to the neighboring transparent portion of the cornea also, so that, as a rule, no portion of the cornea retains its normal curvature. In consequence of the irregular astigmatism, objects appear indistinct, distorted, and often also double or multiple.

The disturbance of vision produced by an opacity of the cornea often entails still other *indirect results.* Among these are strabismus, nystagmus, and myopia. The last named is in many cases only apparent, the patient seeming near-sighted only because he brings minute objects unusually close to his eye, in order to make their retinal images as large as possible, and so in a measure compensate for their indistinctness. Nevertheless, elongation of the axis of the eye—i.e., true myopia—may also ultimately develop in these cases (cf. §§ 200, 209).

611. Treatment.—The chief task that this has to attend to is to improve the sight. The means employed for this purpose are as follows:

(*a*) *Clearing up of the Opacity.*—In the case of every opacity of recent date we must first try to clear it up as much as possible by the application of irritants (see § 546)

As Pyle has pointed out, it is particularly important to do this in the case of young children—first, because we are much more likely to succeed in clearing up opacities in them than in adults (see § 534); second, because the indirect results of the opacities, such as myopia (see above), are particularly apt to develop and advance in childhood; and third, because if such indirect results have already begun, it is much easier to check them at the outset than later.

For clearing up old opacities, especially those produced by parenchymatous keratitis, *electricity* has done good service in some cases. The positive pole of a constant-current battery is placed on the temple or the neck, while the negative pole is applied to the previously cocainized cornea. The negative pole consists of a solid cylinder of silver 7 mm. in diameter. This is surrounded by an insulating envelope of caoutchouc, the only portion exposed being the surface at its end, which is concave so as to fit the surface of the cornea. Contact between the electrode and the cornea is effected by a drop of mercury, which readily adheres to the concave surface of the silver. The current intensity employed is 0.2 to 0.5 milliampères (Alleman).

In older opacities, which cannot be cleared up any further by medicinal means, the next step apparently would be to render the cornea transparent once more by excising the opaque layers. Such attempts, however, have resulted unsuccessfully, for the loss of substance resulting from the excision of the opacity heals again with the formation of opaque cicatricial tissue. Removal of opacities to restore transparency is indicated only when they are situated in the epithelium, since losses of epithelium are made good by normal transparent epithelium. Cases in which removal of the epithelium —*abrasio corneæ*—is indicated, are those in which the epithelium has been thickened by mechanical irritation, as in trichiasis; also in those in which lead, lime, or grains of powder are imbedded in the epithelium, and finally in some cases of zonular opacity of the cornea. We may also remove fistulous or protruding scars, not with the idea of restoring the transparency, but in order to replace a yielding scar tissue by a piece of sound, firm cornea (§ 945).

(b) *The optical aids* that may be employed for improving the sight are glasses and the stenopæic aperture. The object of the latter is to bring nothing but the transparent part of the cornea into use for vision, and to exclude the portion bearing the opacity, by which means the dazzling due to diffusion is prevented. Glasses are often of advantage when the opacity is complicated with changes in the curvature of the cornea and in this case contact glasses may be tried (§ 174).

(c) Displacement of the pupil by an *iridectomy* is generally the only means of restoring sight in the case of dense opacities which entirely conceal the pupil. Iridectomy is also indicated when it is not a question of producing an optical improvement, but of checking the injurious consequences that many scars cause, such as increase of tension and late infection. (For the indications and the method of performing this operation, see §§ 947 et seq.

(d) *Tattooing of the cornea* (§ 946) is done to relieve the disfigurement caused by large, white opacities and also to render opaque a faint, hazy, irregular scar which occupies a portion of the pupillary area and causes dazzling and irregular astigmatism.

XIII. Ectasiæ of the Cornea

612. Ectasiæ in General.—Any ectasia or protrusion of the eyeball, whether localized or general, implies either increased intra-ocular pressure or diminished resistance of the ocular coats. The former is the more freuent cause so that most ectasiæ are due either to primary or secondary glaucoma. Diminished resistance is most often due to diffuse or localized inflammation, causing thinning or perforation of the coats of the eye. Very often both causes are combined, as in most cases of inflammatory ectasia, in which the inflammation thins the coats of the eye and makes them unduly yielding and at the same time supplies the conditions that lead to increase of tension.

If the ectasia is caused by increased tension, it usually develops at a time when the vision is already nearly or quite abolished. In primary ectasiæ the cornea may be clear and the vision fair, although the latter is always considerably affected by the change of refraction—usually a considerable degree of myopia and astigmatism—produced by the change of shape of the cornea.

A considerable ectasia produces a marked deformity. The protrusion may actually simulate an exophthalmus, and interferes with the movements of the lids so that the ectasia becomes irritated and inflamed from deficient moistening. In extreme cases the lids may not close over it at all, and the protruding portion becomes xerotic or ulcerated.

Ectasiæ of the cornea are called *inflammatory* or *non-inflammatory* according as they are or are not dependent on inflammation of the cornea. Non-inflammatory ectasiæ comprise keratoconus, which develops without increase of tension, and keratolobus, which is due to increase of tension and is really a form of glaucoma (§ 836). Inflammatory ectasiæ comprise keratectasia and staphyloma, the former being due to non-perforating, the latter to perforating keratitis.

1. Keratoconus

613. Symptoms and Course.—In keratoconus, the central part of the cornea very gradually and without inflammatory symptoms begins to bulge forward in the form of a cone. At first the cornea is perfectly transparent and its peripheral portions keep their normal curvature. Accordingly the bulging forward of the centre of the cornea, as long as it has not advanced too far, is recognized only by the diminution in size which the corneal reflex presents in the central part of the cornea. In the subsequent course of the disease the bulging of the centre constantly increases, and the peripheral parts of the cornea are also involved in the conical projection, so that we can perceive the conical shape of the cornea at a glance, especially when we look at the eye from the side (Fig. 270). Finally, the apex of the cone becomes opaque and its surface uneven. The sensitivity of the cornea at the apex of the cornea is often diminished.

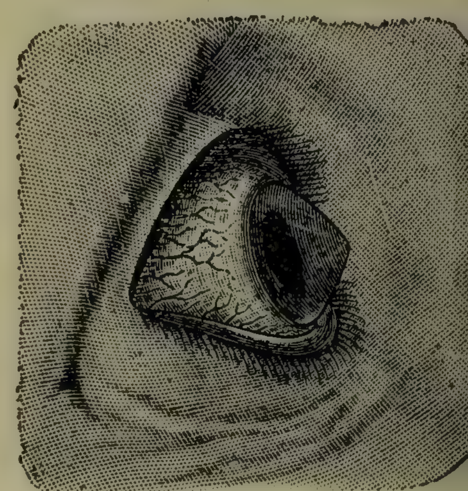


FIG. 270.—KERATOCONUS. (After Elschnig.)

A keratoconus of slight degree may be *readily overlooked*, since the cornea is perfectly transparent. The *diagnosis*, however, can be made even in the earlier stages by examining the corneal reflex, particularly if we use Placido's keratoscope (§225) for the purpose (see Fig. 109.) The latter instrument at the same time shows most clearly that the apex of the cone is not ordinarly situated in the centre of the cornea, but somewhere below it. Again, if we illuminate the pupil with the ophthalmoscope, we see in the red field peculiar shadows which shift suddenly with the slighest movement of the mirror.

In keratoconus there is a marked and sudden *thinning of the cornea* within the area of the conical protrusion. In advanced cases this thinning can be demonstrated by the ease with which the apex of the cone can be dimpled by means of a sound. Anatomical examination has shown that the cornea may be reduced in its centre to a third of its normal thickness (Wagner, Hulke). In less marked cases the thinning can be demonstrated by the behavior of the posterior corneal reflex (§ 389). Near the periphery of the cornea, this reflex and also its distance from the anterior reflex are normal, but at some point nearer the centre of the cornea the posterior reflex suddenly becomes deformed and separates abruptly from the anterior. It is evident that at this point the posterior surface of the cornea suddenly becomes much more concave, so as to make the cornea much thinner.

Owing to the considerable degree of stretching of the cornea, lacerations are produced in Descemet's membrane like those occurring in buphthalmus (see § 836). The *opacities* which are found at the apex of the keratoconus are partly the result of this laceration and unevenness of Descemet's membrane, partly the result of mechanical injuries to which the superficial layers of the cornea are exposed at the point of greatest prominence. In particularly bad cases, ulceration at the apex of the cone may be produced in this way.

Besides opacities which are lasting and which steadily increase, there are sometimes discovered *opacities that are present for a time only.* These form very minute parallel and generally vertical gray lines at the apex of the cone, and probably represent wrinkles on the posterior surface of the cornea. Other temporary opacities are produced by ruptures of Descemet's membrane, which admitting aqueous, cause the cornea to swell and protrude still more (*acute keratoconus*) and become opaque. Subsequently the endothelium regenerates, and then the cornea resumes its former aspect except that a faint whitish line remains at the site of the rupture.

In many cases of keratoconus a yellowish or greenish ring, surrounding the apex of the cone is found (Fleischer). According to some this ring consists of hæmosiderin, according to others of pigment entering the eye from without, but its true source is not yet certainly known.

In very rare cases a *pulsation* in the apex of the cone has been demonstrated, objectively with the ophthalmometer, subjectively by the change in the shape of the diffusion images. This has also been observed in a cornea in which there was no keratoconus (Schneider).

In high degrees of keratoconus, in which the apex is already opaque, the distinction from a keratectasia following a central ulcer of the cornea is often very difficult. We must then take into consideration the condition of the other eye. In keratoconus we almost always find the second eye diseased as well, although not ordinarily to the same extent; while a central keratectasia could only by a rare accident be present at the same time in both eyes.

The chief *symptom* is the disturbance in sight due to the refractive changes (astigmatism and myopia) produced by the bulging. The astigmatism is often of very high degree (6–12 D or more), often shows considerable changes from time to time and, owing to the irregularity of the curvature, is only partially corrigible by glasses. The myopia may also reach a considerable degree and, as it is due to changes confined to the anterior segment of the eyeball, is generally unattended by the fundus lesions characteristic of high myopia in general (§ 745). In the later course of the disease the vision is still further reduced by the opacities developing at the apex of the cone.

Keratoconus is a rare disease, which, as a rule, affects both eyes. It is occasionally congenital but generally begins between the twelfth and the twentieth year, develops very gradually in the course of years to the height above described, and ultimately, sooner or later, comes to a standstill. A subsidence of the ectasis is not observed; but rupture of the cornea is not observed either. Nor does increase in tension, which so frequently develops in inflammatory ectasiæ of the cornea, occur.

The *cause* of the protrusion lies in a progressive thinning of the central portion of the cornea, which consequently gives way before the intra-ocular pressure, i.e., the tension is relatively too high (see § 394). How this thinning is produced is unknown.

Keratoconus occurs much oftener in women than men, and especially in delicate, anæmic women. In women who become repeatedly pregnant, a distinct advance of the keratoconus has been observed with each pregnancy. By some the condition is attributed to disturbance of the endocrine organs.

When we see a keratoconus at the outset we can never foretell to what *extent* it will develop, since in some instances it remains stationary after having attained only a moderate pitch, at other times it reaches the very highest point, and moreover ordinarily acts in a different sort of way in the two eyes of the same patient.

Treatment can point to but slight results in this disease. If we are dealing with cases of moderate intensity, and particularly if after being under observation for a long time they prove to be stationary, it is best to content ourselves with an optical correction of the refractive errors. Usually a satisfactory improvement in the sight can be secured by concave spherical glasses combined with cylinders. In selected cases marked improvement of sight is secured by the use of discs with stenopæic holes or slits, the size

and shape of these being determined by careful experiment (Mackay, Hen sen, Snellen, Fox). Also the hydrodiascope or contact glasses (§ 174) ma be tried. When the protrusion has become so great that serviceable visio cannot be obtained even with glasses, operation is indicated (see § 944).

2. *Keratectasia*

614. By keratectasia we understand a protrusion of the cornea, pro duced by a non-perforating keratitis. The protrusion, therefore, in thi case consists of corneal tissue, in contradistinction to staphylomata, i which it is formed of the tissue of the iris. From ectasiæ of non-inflam matory origin—i.e., keratoconus and keratoglobus—keratectasia is dis tinguished by the fact that the bulging portion of the cornea, in consequenc of the inflammation, is opaque.

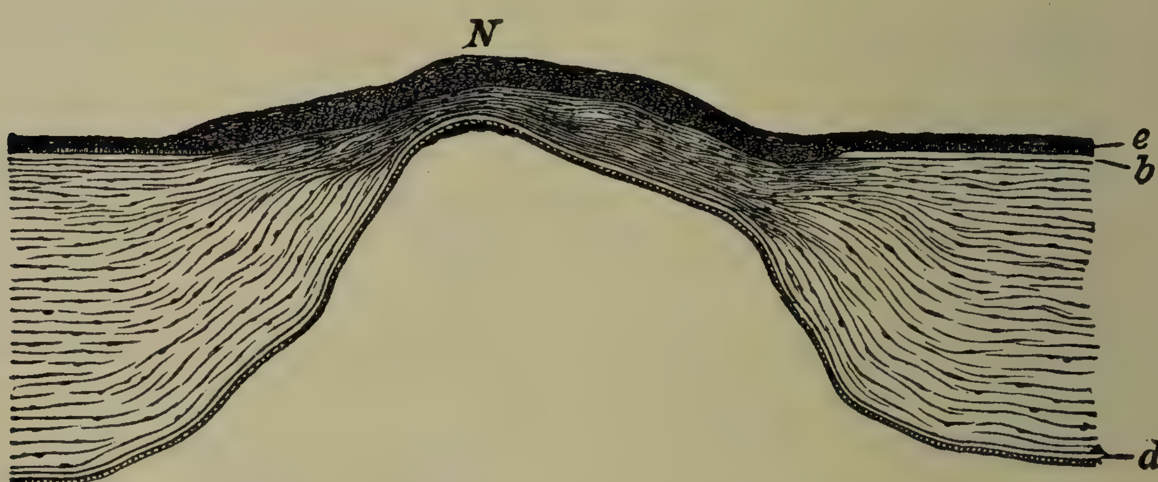


FIG. 271.—KERATECTASIA PRODUCED BY AN ULCER. Magnified 25 ×1.

The thinned and protruding cicatrix is distinguished by its denser texture from the adjacent normal cornea. The epithelium, *e*, over it is thickened, while Bowman's membrane, *b*, is wanting. On the other hand, Descemet's membrane, *d*, with its epithelium, is everywhere present—a proof that the ulcer has not perforated.

Inflammation produces protrusion of the cornea by *thinning* in those cases in which an ulcer of the cornea has destroyed the superficial lamellæ of the latter to such an extent that the posterior lamellæ are no longer able by themselves to offer resistance to the intra-ocular pressure (*keratectasia ex ulcere*, Fig. 271). If all the layers as far as the membrane of Descemet have been destroyed, the latter alone protrudes in the form of a transparent vesicle—keratocele (§ 532). An obliquely set marginal ectasia or a total protrusion of the cornea may be produced by catarrhal ulcers (§ 537) or a senile marginal atrophy (§ 596).

Ectasis of the cornea may also take place because of a *softening* that results from inflammation. Under this head belong the *keratectasia e panno*, which develops when a thick pannus penetrates pretty deeply into the cornea proper, also the keratectasia following parenchymatous keratitis. In these cases there is a uniform protrusion of the whole cornea.

In keratectasia the protruding portions of the cornea have very little power of regaining their transparency. Accordingly, the prognosis for vision in pannus, keratitis parenchymatosa, etc., must be regarded as essentially more unfavorable as soon as protrusion of the cornea shows itself. And in considering the prognosis we must take into account, besides the opacity

of the bulging cornea, its alteration in curvature and the resulting alteration of refraction of the eye. Sometimes keratectasia is followed by increase of tension.

Treatment is powerless against a fully developed keratectasia; it is attended with success only when there is a question of combating a protrusion that is in the process of development. The means suited for this latter purpose are repeated punctures of the cornea with the subsequent application of a pressure bandage, and iridectomy. Very small ectasiæ, as, for example, small keratoceles, we may perforate with a cautery point and then induce the formation of a flat cicatrix by the long-continued application of a pressure bandage.

3. *Staphyloma of the Cornea*

615. Character.—A staphyloma is an ectasia produced by a perforating keratitis. It is a protuberant scar which is not as in keratectasia composed of altered corneal tissue but is derived from a prolapsed iris which replaces the cornea. A staphyloma is denoted as partial or total, according as it occupies part or the whole of the area of the cornea.

A *partial staphyloma* rises as a white prominence, usually in the form of a cone (staphyloma partiale conicum); a spherical protuberance (staphyloma partiale sphæricum) is less common. A partial staphyloma usually extends in one direction as far as the margin of the cornea, while in the other direction there is a portion of the cornea of varying extent which is still left, and which, moreover, is generally transparent, so that the iris can be recognized behind it. The iris is drawn forward to the staphyloma, so that the pupil is displaced toward the latter and often partly concealed by it. Indeed, the pupil may be closed up altogether if the whole pupillary margin of the iris is incorporated in the staphyloma. In *total staphyloma* there is found in place of the cornea an opaque, protuberant cicatrix, the base of which is encircled by the margin of the sclera or by the very outermost rim of the cornea, which may still be preserved. In one series of cases the protuberant cornea has the form of a cone (staphyloma totale conicum). In conical staphyloma the protuberance starting from the margin of the sclera slopes gradually up to its apex (Fig. 276). In other cases, however, the protuberance is hemispherical (staphyloma totale sphæricum), and its walls, rising abruptly from the sclera or even overhanging it, are sharply demarcated from the latter (Fig. 273). The spherical is more frequent than the conical form in total staphyloma. Many spherical staphylomata, above all, those of recent date, have such a very thin wall that the layer of black pigment (Fig. 273, *i*) on its posterior surface is seen through it, shining with a bluish lustre. Such staphylomata accordingly form a slate-colored or bluish-black hemisphere, which in form and color has a certain resemblance to a blue grape, whence the name staphyloma (σταφυλή, a bunch of grapes). Afterward thickening of the wall of the staphyloma occurs. If this takes place first under the form of separate, stout bands, by which the surface of the staphyloma is constricted in spots somewhat after the fashion of a blackberry, what is called staphyloma racemosum is produced. Old staphylo-

mata have for the most part a thick white wall, in which usually one or two dark spots may be observed, resulting either from a deposition of pigment or from localized thinning (Fig. 273, *a*). Staphylomata are generally traversed by one or two pretty large vessels originating from the conjunctiva. On account of the opaqueness of the staphyloma, nothing is to be seen of the deeper parts of the eye. The iris is all taken up into the staphyloma—that is, all of it except its extreme periphery, which is so closely applied to the posterior surface of what remains of the marginal portion of the cornea that there is no longer any anterior chamber.

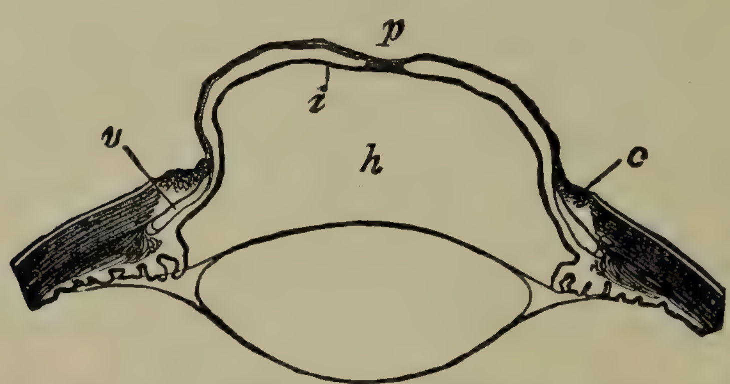


FIG. 272.—TOTAL PROLAPSE OF THE IRIS.

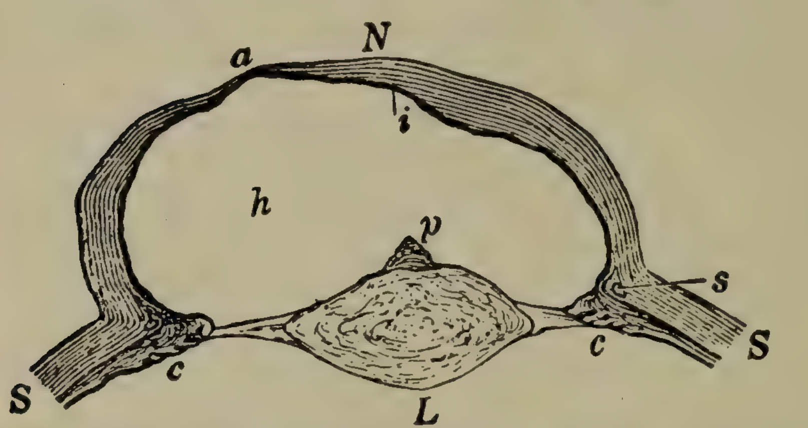


FIG. 273.—TOTAL SPHERICAL STAPHYLOMA OF THE CORNEA WITH CONSECUTIVE ELEVATION OF TENSION, ORIGINATING FROM THE TOTAL PROLAPSE OF THE IRIS REPRESENTED IN FIG. 272.

Out of the iris, *i* (Fig. 272), which has been thinned out in consequence of its protrusion, a thick cicatrix, *N*, has grown, in which a thin spot, *a*, corresponds to what was once the pupil (*p*, Fig. 272), while on the posterior surface of the cicatrix the retinal pigment of the iris remains as a black coating, *i*. The staphyloma is apparently directly continuous with the sclera, *S*, from which it separated only by Schlemm's canal, *s*. For, on account of the elevation of tension, the periphery of the iris has been pressed against the cornea so that the anterior chamber (*v*, Fig. 272) has disappeared, and the iris and the marginal portions of the cornea are fused into a single mass which has been incorporated in the staphyloma. As a comparison of the two figures shows, the corneo-scleral margin has become distended. Because of this, and also because of the shrinking of the lens, *L* the zonula has become tightly stretched, and has drawn the atrophic ciliary processes, *c*, inward. The lens is cataractous, shrunken, and has at its anterior pole a pyramidal cataract, *p*.

616. Anatomy and Pathogenesis.—The way in which a staphyloma develops in the case of a total prolapse of the iris is as follows. A prolapsed iris (Fig. 272, *i*) becomes more or less inflamed and is covered with a fibrinous exudate. The latter exudate closes the pupil (*p*), glues the iris to the margin of the ulcer and thus shuts off the space behind the iris—the dilated posterior chamber (*h*)—completely from the remains of the anterior chamber (*v*). The aqueous accumulates in the posterior chamber, distends the latter more and more, so that the prolapse protrudes still more, pushes the portion of the iris which remains in the eyeball against the corneo-scleral margin, and thus shuts off completely the passages of out-flow. There is thus produced a peculiar variety of seclusio pupillæ. At the outset the increase in volume of the interior of the eye is compensated for by the stretching of the wall of the staphyloma, but later when this wall has become solidified by cicatrization the increase in tension becomes manifest, being evidenced by a perceptible hardness and by pain. By the cicatrization the prolapsed portion of the iris is transformed into tough scar tissue.

The wall of a staphyloma consists of a dense, tough cicatricial tissue, which is traversed by a few vessels, and often contains pigment imbedded in it. The thickness of the wall differs greatly; it varies from the thickness of a sheet of paper to a thickness three times as great as that of the normal cornea, and more. Very thick staphylomata are often as hard as cartilage, forming what are called cicatricial fibromata or keloids. Thick and thin spots frequently occur in the wall of the same staphyloma (Figs. 273 and 276). The anterior surface of the staphyloma is covered by a thick, irregular layer of laminated epithelium sometimes containing epithelial pearls. On this there is sometimes a rather delicate

vascular layer resembling a pannus. The posterior surface is frequently uneven, on account of the inequality in thickness of the wall. It is covered by a coating of black pigment (Fig. 273, *i*, and Fig. 276), which is nothing but the retinal pigment layer of the iris. As, however, this has to be distributed over such a large surface, it is rarefied, so that the epithelial coating shows numerous gaps, and in the centre of the staphyloma, corresponding to what was once the pupil, it is often entirely wanting. Why Bowman's membrane should be wanting on the anterior surface of the staphyloma, and Descemet's membrane on its posterior surface, is self-evident since the staphyloma is not cicatrized cornea, but is iris, and represents a spot at which the cornea has been destroyed. It is only on the sloping sides and the edges of the staphyloma, which are formed of the remains of the cornea, that both these membranes can still be demonstrated. This is more the case in a conical than in a spherical staphyloma. A spherical staphyloma, in fact, is produced in cases where the sides of the perforation shelve off abruptly, so that the

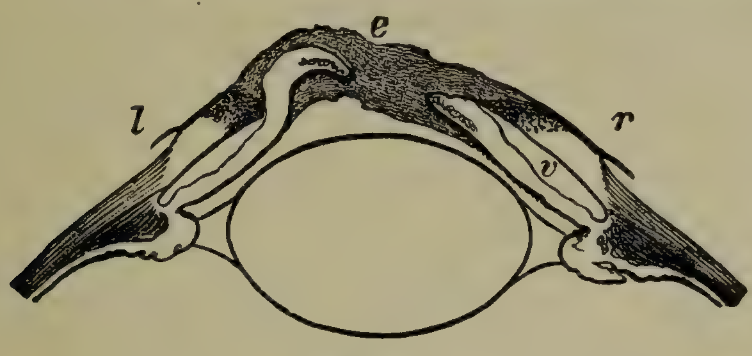


FIG. 274.

FIG. 275.

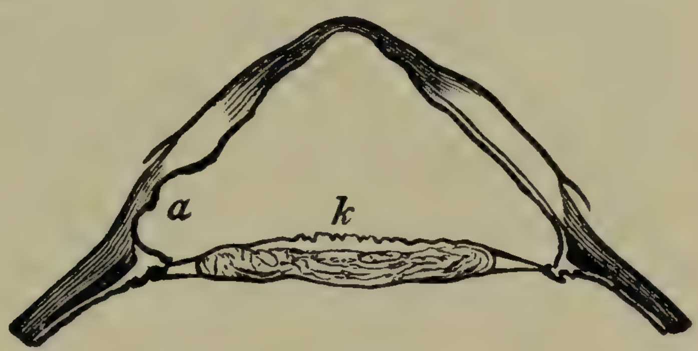


FIG. 276.

FIGS. 274–276.—DEVELOPMENT OF A TOTAL CONICAL STAPHYLOMA. Magnified 3×1.

FIG. 274.—A large ulcer has destroyed the central portions of the cornea. Into the aperture left by the perforation projects the iris; on the right side, *r*, presenting only by its pupillary border, while on the left side, *l*, it forms a real prolapse. A gray mass, *e*, composed of coagulated fibrin, pus corpuscles, and detritus, fills the perforation and covers the exposed iris. The anterior chamber, *v*, is very shallow, and nevertheless the posterior chamber has become shallower as well because the lens is pushed forward. Moreover, the latter, owing to the relaxation of the zonula, has an increased curvature.

FIG. 275.—In place of the prolapsed iris a somewhat protuberant scar has developed, which is thinner than the cornea, and in which is included the entire pupillary margin of the iris. In consequence of this seclusio pupillæ the whole iris has been pushed forward right up against the cornea, so that the anterior chamber has disappeared and the posterior chamber has become correspondingly deeper. The lens has re-acquired its normal shape, and shows beginning opacification of its cortical layers and wrinkling of the anterior capsule in the region, *k*, formerly occupied by the pupil.

FIG. 276.—The increase of tension that has set in as a result of the seclusio pupillæ has produced a uniform protrusion of the marginal portions of the cornea, which now form the sloping sides of the conical protuberance, and which are not separated by any sharp line of demarcation from the central cicatrix. On the right side of the drawing the iris can still be made out, although fast adherent to the cornea and greatly atrophied. On the left side nothing is left of the iris but the retinal pigment layer coating the posterior wall of the staphyloma. The wall of the staphyloma shows various protuberances caused by the constant increase in tension. The largest of these, *a*, situated directly in front of the ciliary body, represents a beginning intercalary staphyloma. The posterior chamber is very deep, the ciliary body, owing to atrophy, is flattened out, and the lens is shrunken, flat like a cake, and presents a capsular thickening, *k*.

cornea even in the immediate vicinity of the opening has its normal thickness. In this case, when the prolapsed iris is driven out into the opening, the adjoining corneal tissue takes little or no part in it. The sides of the prolapse rise at right angles to the adjoining corneal surface, and thus a spherical staphyloma is formed. A staphyloma of the sort readily develops when, as in Fig. 272, only a narrow marginal rim of cornea is left. This rim then is afterward carried forward under the influence of the intra-ocular pressure. while the sclera, being firm, offers resistance, so that now there is a furrow formed at the corneo-scleral junction (Fig. 273). The latter is particularly apt to develop such a

furrow, since even under normal conditions it forms a re-entrant angle. A conical staphyloma, on the other hand, is the result of a less extensive perforation (Fig. 274), in which the portions of cornea forming the walls of the aperture taper down toward the latter, and on account of their thinness are driven forward at the same time with the prolapsed iris. The fully developed ectasis, therefore, consists only in its central part of old prolapsed iris, its lateral slopes being in large part formed of the tapering, protruded cornea (Fig. 276). It is thus really a combination of a staphyloma and a keratectasia.

In staphyloma the *iris* persists as an independent structure only in cases in which at least a part of the pupil has remained free, so that the anterior and posterior chambers can communicate through it. If, on the other hand, the entire pupillary border has been incorporated in the cicatrix (seclusio pupillæ), increase of tension sets in, and as the iris is consequently pushed forward right up to the cornea (Fig. 275), the shallow anterior chamber, which was present at the outset (Figs. 272 and 274, *v*), disappears. Then the iris becomes more and more intimately adherent to the cornea, and becomes constantly thinner and thinner as a result of atrophy, so that at last scarcely anything of it but the pigment layer remains to cover the posterior surface of the staphyloma (Fig. 276). In such cases, even when the marginal portions of the cornea are still somewhat transparent, an iridectomy has become technically impossible.

As the anterior chamber becomes shallower the posterior becomes deeper; in total staphylomata, the whole large space between the posterior surface of the staphyloma and the lens is to be looked upon as the posterior chamber (Figs. 273 and 276).

The *ciliary body* suffers mainly on account of the increase in tension, which causes it to atrophy, especially if an ectasis of the sclera develops in the ciliary region (staphyloma ciliare). Furthermore, the ciliary processes are sometimes very strongly pulled upon by the fibres of the zonula, and are thus elongated (*c*, Fig. 273).

The *lens* very frequently suffers changes in the case of staphylomata. In total staphylomata it is often entirely wanting, because it has been discharged from the eye through the pupil at the time when a large perforation existed. If the lens is still present it frequently shows alterations of position, being tilted in consequence of the unequal bulging of the staphyloma. Sometimes we find it partially adherent to the staphyloma, or it vibrates with the movements of the eye, because of the atrophy of the stretched zonula of Zinn. These alterations in the lens favor the development of an increase in tension; for this reason, after performing incision or ablation of the staphyloma, we remove the lens from the eye. Very frequently the lens is rendered opaque either in toto or only at its anterior pole (anterior polar cataract, *p*. Fig. 273). In some few cases we may find the lens greatly diminished in size, or even shrunken into a mere membrane (Fig. 276).

The *deeper parts* also suffer from the increase in tension. Excavation of the optic nerve, atrophy of the retina and chorioid, and fluidity of the vitreous thus develop.

617. Etiology.—The protrusion which constitutes a staphyloma may be primary or secondary in its development.

(*a*) The protrusion is called *primary* if it occurs at the same time with the perforation of the cornea, the iris becoming prolapsed and bulging forward because it is too weak to sustain even the normal intra-ocular pressure. The cicatrization which follows, and which in favorable cases produces flattening of the prolapse, can not in unfavorable cases do away with the protrusion. On the contrary, the prolapsed iris remains protruded at the same time that it is gradually converted into cicatricial tissue; it becomes consolidated while still in a position of protrusion (thus from the prolapse of iris in Fig. 272 is formed the staphyloma represented in Fig. 273). A total or partial staphyloma develops according as a total or partial prolapse of the iris has existed. The causes which oppose the conversion of a prolapse of the iris into a flat scar and which favor the formation of a staphyloma are chiefly two: The first is large size of the perforation. In very

small perforations there is no development whatever of staphyloma; and the larger the perforation, the more likely is it that a staphyloma will develop. The second cause is improper behavior on the part of the patient. In this regard the chief factors to be considered are, in adults, great physical exertion; in children, crying, and also squeezing together of the lids; and, in both, great straining at stool. The temporary increase of tension thus induced distends the newly formed and yielding cicatricial tissue; but as the latter has no elasticity, it does not return to its former dimensions after the elevation of tension has disappeared, but remains permanently protruded. In that case, however, a permanent protrusion leads sooner or later to permanent increase of tension which now does not have its cause in external influences but in the obliteration of the sinus of the chamber produced by the incarceration of the iris.

(*b*) We speak of *a secondary protrusion* when a prolapse of the iris first heals with the formation of a flat scar, which latter afterwards bulges out again. The cause of this is frequently supplied by the same injurious influences that have been enumerated above—e.g., by the too early resumption of work by a patient with a recently cicatrized corneal ulcer. The recent cicatrix is still too yielding to offer a proper resistance to the repeated though transitory elevations of intra-ocular pressure, and so becomes gradually distended. But in any case a protrusion is sure to occur whenever the inclusion of the iris is of such a kind that by it there is produced a blocking of the channels of outflow and consequently a permanent increase of intra-ocular tension. This is very often the case in partial inclusion of the iris and very regularly so in inclusion of the whole pupillary margin (seclusio pupillæ, Figs. 274–276).

In individual cases a primary and secondary protrusion cannot always be sharply differentiated. A very frequent case is that in which the iris, being extensively prolapsed, is protruded from the start; the intra-ocular pressure in this case being at first normal and afterwards gradually rising, so that the protrusion then increases still more.

618. Consequences of Staphyloma.—The *sight* is always diminished. In total staphyloma it is reduced to the mere ability to distinguish between light and darkness. In partial staphyloma the degree of sight depends upon the character of the part of the cornea that is still preserved, and also upon the position of the pupil. Even in the most favorable case, in which a part of the pupil happens still to lie behind perfectly transparent cornea, the sight is considerably reduced by the irregular curvature which is present not only at the site of the staphyloma itself, but to a less degree over the whole cornea. Large staphylomata produce a very conspicuous disfigurement. They also cause trouble by giving rise through mechanical irritation to catarrhal conditions of the conjunctiva, with increased secretion, lacrimation, etc. Closure of the lids is rendered difficult in the case of large staphylomata by the great size of the protrusion; the apex of the latter, being but incompletely covered by the lids, becomes dry (xerotic), or becomes the site of ulcers (atheromatous ulcers). Sometimes the lids are forced so much apart by the staphyloma that ectropion develops.

Staphyloma of the cornea is almost invariably accompanied by *elevation of tension.* This sets in at different stages in the development of the protrusion, and may be partly the result, partly the cause of the latter. Besides causing the increase of intra-ocular pressure that is perceptible to the touch, this elevation of tension finds its chief expression in a diminution of sight, which finally ends in complete blindness. It sometimes also causes pain.

As a rule, the increase of tension resulting from staphyloma develops quite gradually. But sometimes often without any apparent cause, protrusion of the cicatrix and increase of tension take place simultaneously and in a sudden fashion, with severe pain and marked congestive symptoms.

As soon as increase of tension has set in, it gives rise to further changes both in the staphyloma and also in the whole eyeball. Thin-walled staphylomata are made to protrude farther and farther by the heightened pressure, and thus suffer an increasing attenuation of their wall, until the latter, from almost any trifling cause *ruptures* at some particularly yielding spot. Opening of the staphyloma may also occur from perforation of one of those ulcers which so frequently develop at the apex of the staphyloma, either as a result of mechanical injury or because of the inadequate nutrition of the scar tissue. In either case when perforation takes place, the aqueous humor is discharged in great abundance; and the staphyloma collapses, becomes smaller and remains so for some time. But perforation of the staphyloma may also be followed by profuse intra-ocular hæmorrhage, or by severe iridocyclitis or panophthalmitis. Then the eyeball undergoes atrophy, and by this means a sort of spontaneous cure of the staphyloma takes place.

The scleral portion of the eyeball, too, when the elevation of tension has lasted a pretty long time, gives way and becomes distended, especially in young people in whom the sclera is more extensible. In this case we observe both total and partial *ectasia of the sclera.* In the former (§ 633), the sclera becomes uniformly distended, the entire eyeball grows larger, and the sclera becomes so thin as to look bluish owing to the way in which the chorioidal pigment shines through it. In the second case (§ 631), the sclera in the vicinity of the cornea bulges forward under the form of a circumscribed swelling, which appears dark from the pigment shining through it; intercalary and ciliary staphylomata (see § 631) are developed. Very frequently general and partial ectasiæ of the sclera are found simultaneously in the same eye, which may thus grow to an enormous size.

619. Treatment.—Stress is chiefly to be laid upon *prophylaxis.* The physician who has to treat an eye with prolapse of the iris must make every endeavor to secure the production of a flat cicatrix. He should not suffer a staphyloma to develop before his very eyes. In this connection, what has been said in regard to prolapse of the iris (§ 545) may be consulted. When we have succeeded in effecting the formation of a flat cicatrix we must, while the latter is still recent, take measures to keep it from bulging out again. With this end in view we should refrain from discharging the patient too soon from treatment, and particularly we must advise him to abstain for a long time from all severe physicial exertion. It is often advisable before dis-

charging the patient, to perform an iridectomy, whenever possible, as by this means the subsequent development of an ectasis is most effectually counteracted.

If we have to do with a staphyloma which has already developed, our treatment must have a different object in view, according as the case in hand is a total or a partial staphyloma. With the former the sight is irreparably lost, since there is no transparent cornea left; we must hence confine ourselves to the relief of the symptoms and of the disfigurement produced by the staphyloma. With partial staphylomata, our first aim is to improve whatever sight may be left, or at least to preserve it from injury (as would be produced by increase of tension). For the methods employed to cure staphyloma, which are all of an operative character, see §§ 942, 943.

It cannot be denied that, in spite of all we can do, the cicatrix in many cases keeps constantly bulging anew, the increase in tension constantly returns and thus the eye slowly but inevitably goes blind.

XIV. Other Anomalies of the Cornea

620.—Tumors of the Cornea.—Tumors that develop primarily in the cornea are among the greatest of rarities. Isolated instances of primary papilloma, fibroma, dermoid, myxoma, sarcoma, and epithelioma of the cornea have been published. The carcinomata and sarcomata which not infrequently are observed upon the cornea do not originate there, but in the adjacent conjunctiva, and in fact generally in the limbus. These tumors have been already considered under diseases of the conjunctiva; so also has been the dermoid, a congenital form of tumor, situated partly upon the cornea, partly in the conjunctiva.

621. Congenital Anomalies.—These include anomalies of—

1. *Size.* Megalocornea, or the condition in which the cornea is excessively large (over 13 mm. in diameter) occurs in hydrophthalmus (§ 836), but also sometimes when the eye as a whole is not enlarged; microcornea (cornea less than 9 mm. in diameter) is one of the evidences of an abnormally small eye (microphthalmus).

2. *Shape.* Instead of being horizontally oval the cornea may be circular or vertically oval. The latter form is associated with inverse astigmatism. In eyes with typical congenital coloboma of the iris, the cornea often has the shape of an egg standing on end. It has a like shape, however, in eyes with no coloboma and especially in those affected with parenchymatous keratitis (Fuchs).

3. *Curvature.* High astigmatism and occasionally keratoconus occur as congenital conditions.

4. *Transparency.* Congenital opacities of the cornea comprise *embryotoxon* an opacity of unknown causation resembling the arcus senilis, but lying near the centre of the cornea and less regular in shape. Other opacities are the results of *fetal diseases* (parenchymatous keratitis or gonorrhœal conjunctivitis, which have produced scars or staphylomata) or *birth injuries* (injuries from the forceps). *Melanosis corneæ* is a congenital pigmentation of the cornea. The pigment may either be seated in the epithelium, being continued into the cornea from the pigment that is not very infrequently present in the limbus; or in the endothelium, in which it forms rather large spots in the centre of the cornea, which, like the pigment remnants found on the lens (see § 736), are regarded as representing the remains of a pupillary membrane.

CHAPTER XXIII

DISEASES OF THE SCLERA

I. Inflammation of the Sclera

622. Inflammation of the sclera (scleritis) is one of rarer affections o the eye. It usually attacks the anterior segment of the sclera, lyin between the equator of the eyeball and the margin of the cornea. It some times affects only the superficial layers of the sclera (superficial scleritis episcleritis), sometimes the deep layers also (deep scleritis or sclerit proper).

623. Episcleritis.—Episcleritis (superficial scleritis) is a focal inflam mation, a circumscribed inflammatory nodule forming in the sclera. A the affected spot the sclera, owing to the deposition of exudate, bulges ou in the form of a boss, so that a prominence which is sometimes flat, some times more acute, and which may reach or surpass the size of a lentil, i found here (Fig. 277). This is traversed by vessels which, because deepl situated (episcleral), are violet in color; and it is immovably attached to th sclera, while the conjunctiva, though, to be sure, injected, can be move about freely. The nodule feels hard, and is sometimes very sensitive to th touch. Except at the site of the nodule the eye may be perfectly free from injection. The subjective disturbances vary greatly; frequently the disease is associated with but slight discomfort, while in other cases very violent pain is present, which for a long time deprives the patient of sleep.

In the subsequent *course* of the disease disintegration and ulceration of the nodules never occur; on the contrary, they always disappear by resorption. After the inflammation has remained at its acme for some weeks, the nodule gradually flattens, becomes paler, and at length disappears completely, after lasting altogether from four to eight weeks. Sometimes it leaves no trace behind it; more frequently, however, at its site there is left a slate-colored patch, and there the sclera appears somewhat depressed and is closely adherent to the conjunctiva (cicatrix in the sclera). Otherwise the eye suffers no after-injury from the inflammation.

Episcleritis in the well marked cases is a very characteristic and easily recognizable disease. In light and abortive cases the diagnosis is sometimes difficult. For instance, a scleritic nodule which is small and situated particularly close to the margin of the cornea might be taken for phlyctenule. The following characters may be regarded as distinctive. The scleritic nodule never actually lies in the limbus, and besides is not situated in the conjunctiva, but beneath it, so that the latter can be moved about over it. Lastly, the subsequent course will soon clear up the diagnosis, as the phyctenule is converted by superficial disintegration into a conjunctival ulcer—a thing which never occurs with a scleritic nodule.

Scleritis is peculiarly prone to recur. The disease may, to be sure, stop with one or two attacks, or years may intervene between the attacks; but

in other cases, scarcely has the first nodule disappeared—nay, even the first one may not have disappeared—when a second one appears upon another portion of the sclera. Sometimes the disease does not cease until nodules have developed, one after another, in the entire circumcorneal space, and at length a zone of gray discoloration is visible entirely surrounding the cornea. By this time the disease has exhausted itself, since a new nodule does not generally develop in the spot where another was situated before. But, in the meantime, several years may have elapsed, during which the patient with but brief interruptions is annoyed by attacks of inflammation. Moreover, the disease very frequently attacks both eyes. The *prognosis* of episcleritis is hence unfavorable in respect to the duration of the disease; while with regard to the final outcome it must be stated as favorable, because the usefulness of the eye for vision suffers no impairment, even if the process lasts a long time.

Fig. 277.—Episcleritis.

The recent focus is on the outer side of the sclera; from the upper and outer side of the eye dilated conjunctival vessels run down to it. To the outer side and below, and somewhat closer to the cornea, lies a grayish spot at the point where half a year before an episcleritic eminence had been situated.

Episcleritis occurs, as a rule, only in adults and especially in elderly people. In some cases it appears to be connected with rheumatic or gouty affections; in others its origin is obscure. *Treatment* avails but little against it. We are able to ameliorate the symptoms and accelerate somewhat the subsidence of the nodules, without having it in our power to prevent the recurrences. Sodium salicylate or aspirin and sometimes also colchicine are given internally; furthermore, diaphoresis, derivative remedies in the shape of mildly purgative mineral waters, iodide of potassium, etc., may be recommended. As regards local remedies, we may try to produce more rapid subsidence of the nodule by massaging it. Fat, either without adaddition or under the form of the yellow-precipitate ointment, is introduced into the conjunctival sac, and the nodule, which can be felt through the lid, is then rubbed and squeezed by pressure made through the lid with the fingers. If there is violent pain, we may employ hot, moist compresses, dionin, atropine, and local blood-letting (six to ten leeches upon the temple) in addition to the massage: the latter procedure, indeed, in these cases frequently can not be performed because the nodule is too painful. In this event the application of the constant current to the nodule by means of a small electrode (Reuss) has been recommended. Orthoform ointment (10 per cent) or acoine oil (1 per cent) may also be tried.

624. Episcleritis Periodica Fugax.—This disease, also called *subjunctivitis* (Graefe), is an inflammation of the vascular episcleral tissue, distinguished by its transient character and by its tendency to recur. The recurrences often take place with a considerable regularity at intervals of some weeks or months, and may keep on being repeated for years. The separate attacks affect sometimes one, sometimes both eyes. The eye attacked shows marked redness and œdematous swelling of the episcleral tissue and of the over-lying conjunctiva. Often the inflammation is partial in the sense that it is confined to one portion of the anterior segment of the eyeball, or begins in one quadrant of it and travels from this to another. In severe cases, contraction of the pupil and spasm

of the ciliary muscle (temporary myopia) are present. The pain is sometimes sligl sometimes pretty violent. Ordinarily the inflammation runs its course and the eye b comes normal again in a few days. The disease is, therefore, free from danger, but owi to its frequent recurrences is very troublesome. On the whole, it is a rare affection, a commonly attacks persons in middle life, sometimes without known cause, sometimes the result of a rheumatic or gouty tendency. Quinine, sodium salicylate, and treatme directed against a uratic diathesis prove advantageous; but many cases defy all treatme

625. Deep Scleritis.—In this form, too, a swelling of the sclera exis which may appear under the form of separate prominences, but which mo frequently is not so sharply circumscribed. In the latter case the scle shows an extensive bluish-red injection, sometimes covering the whole ci cumcorneal region, and a more uniform non-nodular swelling. Later th part of the sclera often takes on a peculiar pale-violet color and a tran parent look, that make it resemble fine porcelain. But the deep is di tinguished from the superficial form most of all by the course of the inflan mation and by its being communicated to other parts of the eye.

The inflammation in the sclera in this case as in the superficial form leads not to disintegration of the inflammatory products, but to their di appearance by resorption with the formation of a residual dark-colored cica trix. In the meantime, however, the sclera has been undergoing conside able attenuation at the site of the cicatrix, so that it is no longer able t offer resistance to the intra-ocular pressure, even though this does not ex ceed its normal amount. Hence *ectasis* of the diseased spot occurs. In som cases this ectasis affects the entire circumcorneal zone of the sclera, whic has been colored gray by the scleritis and becomes constantly more and mor dilated. Thus the cornea, together with the adjacent portions of the sclera is projected forward, so that the eyeball is elongated in a sagittal directio and becomes pear-shaped. In other cases a circumscribed protrusion of th thinned-out spots, raising them above the level of the healthy sclera, i produced, so that there are seen rising about the cornea a number of humps which, because of their thin walls, show the dark pigment shining through As these belong to the region of the ciliary body, they are called ciliar staphylomata (see § 631).

Complications affecting other portions of the eye are likewise a charac teristic feature of the deep form of scleritis. In the cornea, infiltrates deeply situated, develop, which do not break down into pus but become absorbed again, leaving a permanent opacity (sclerosing keratitis, see § 582) In the iris we find the signs of iritis, mainly under the guise of posterio synechiæ and even of occlusio pupillæ, but never of hypopyon. In the chorioid, the inflammation affects chiefly its most anterior portion, and causes injury to vision chiefly through accompanying opacities in the vitreous. So in this form of scleritis almost all parts of the eye suffer; and for this reason it is much more dangerous than the superficial form.

The superficial and the deep variety of scleritis are not by any means sharply dis- tinguished from each other, but show many transition forms. We cannot in fact, see in the living eye how far the inflammation penetrates into the sclera. When we make the assumption that in the deep form the inflammation does extend more deeply, we have no direct proof of it; we can only infer this indirectly from the subsequent thinning of the

sclera and from the spread of the inflammation to the subjacent uvea. In fact, many authors assume that the real starting point of the inflammation in this form lies in the uvea and they call it, therefore, sclero-chorioiditis or uveo-scleritis. (Cf. remarks on Uveitis Anterior, § 576).

In parenchymatous keratitis we sometimes find a coincident, slight but widely diffused, circumcorneal scleritis. Isolated nodules are sometimes also observed in the sclera

Fig. 278.—Deep Scleritis. Section through the Region of the Ciliary Body, Taken from the Eyeball of a Twenty-four-year-old Girl. Magnified 24 × 1.

The conjunctiva, *B*, which in the enucleation had been cut off close to the limbus is pretty strongly infiltrated. In the subjacent sclera, *S*, are numerous narrow files of round cells, which show a special disposition to accompany the blood-vessels. The scleritic nodule, that was visible on clinical examination, corresponds to a large, irregularly shaped mass of infiltration, occupying almost the entire thickness of the sclera. The posterior section of this mass is the part that presents the most marked accumulation of cells; in the anterior section the cells are most densely placed near the margin that is directed toward the cornea—looking as if the infiltrate were making an attempt to push its way into the latter. The anterior layers of the cornea, *H*, are occupied by quite a large infiltrate, *i* (sclerosing keratitis). In its posterior layers can be made out files of cells, which accompany the deep-seated, new-formed vessels, and which show a tendency to pass up from the deep to the middle layers of the cornea. The ciliary body, *C*, and the iris, *I*, are atrophic. In the former the ciliary processes in particular are greatly diminished in size. At the anterior border of the ciliary muscle can be seen the cross section, *c* of the circulus arteriosus iridis major. The iris also is thinned, and chiefly at its peripheral portion, which over the space, *a b*, is closely applied to the posterior surface of the cornea, and has become adherent to it. Descemet's membrane, *d*, and the lumen of Schlemm's canal are consequently no longer to be made out in this situation. This peripheral adhesion of the iris is in consonance with the increase of tension that existed in the living eye.

as a result of syphilis, but a typical recurrent scleritis does not ordinarily arise from this cause.

In many cases of diffuse deep scleritis there develop in the inflamed zone hard whitish nodules of the size of a pin's head, which lie beneath the conjunctiva and all about the same distance from the margin of the cornea. They might be taken for tuberculous nodules, which, however, they are not. In fact, they disappear again without undergoing disintegration. Real tuberculous nodules do occur. See below.

Morbid Anatomy.—In deep scleritis the sclera is permeated in its middle and dee layers with uninuclear leucocytes (Fig. 278). In particularly severe cases in whic clinically the whole tissue appears to contain a gelatinous infiltration, the sclera at th diseased spot is transformed throughout its entire thickness into a tissue which contain an excessive abundance of cells and in which necrosis has set in here and there. Some times even whole lamellæ of the sclera have become necrotic en masse and form a se questrum. Sometimes in the cellular tissue there are scattered nodules containing gian cells—this without there being any tuberculosis present. In exceptional cases, however there are real tuberculous nodules.

The more intensely the sclera is diseased, the surer we are to find inflammator changes not only in the cornea, but also in the uvea and especially in the chorioid.

626. Deep scleritis almost always affects both eyes, and, as treatment is unable to arrest it, is prolonged over a course of years. It leads to the formation of dense corneal opacities, to seclusio pupillæ with its baneful consequences, to opacities of the lens and vitreous, to myopia of high degree due to the elongation of the axis of the eye, and finally to elevation of tension due to the ectasiæ in the sclera. Hence the disease always ends by producing great impairment or even complete loss of sight.

The deep form, in opposition to the superficial variety, affects mainly young people (but not children). It is often found in conjunction with the signs of scrofula, tuberculosis, or hereditary syphilis. In women, who are more frequently attacked than men by this disease, disturbances of menstruation appear to furnish the exciting cause.

Treatment has very little power over deep scleritis. It has first of all to combat by dietetic and medicinal remedies any constitutional affection that may lie at the root of the disease, and for this purpose the preparations of iodine (iodide of potassium, iodide of iron, iodureted mineral waters), are used, or in case of the disturbances of menstruation, the preparations of iron, and in tuberculous cases injections of tuberculin. As regards the eye itself, the inflammation of the cornea and iris must be treated in the appropriate way. In the subsequent course of the disease iridectomy is frequently required, either for optical reasons, to place the pupil behind that part of the cornea that is still transparent, or to prevent an elevation of tension excited by the seclusio pupillæ or by the ectasiæ of the sclera. Iridectomy, however, should, if possible, not be performed until after the subsidence of all inflammatory symptoms.

627. Scleritis Posterior.—In exceptional cases scleritis is situated in the equatorial region or in the posterior segment of the eyeball. Such a *scleritis posterior* is hard to diagnosticate, because the inflammatory swelling of the sclera, being in a place which it is impossible to get at, can neither be seen nor felt. The symptoms are pain and disturbance of vision. The ophthalmoscope shows the cause of the latter to be a gray cloudiness and swelling or even a detachment of the retina over the scleritic focus. In severe cases there are even some exophthalmus and limitation of the mobility of the eyeball. As the inflammation in the sclera abates, the changes in the retina and the associated disturbance of sight likewise disappear.

628. Ulcers of the Sclera.—Ulcers of the sclera are extremely rare, and even when present almost always develop by extension from neighboring structures (cornea or uvea). They are produced by injuries attended with infection or by the disintegration of syphilitic, tuberculous, or leprous nodules or malignant neoplasms.

II. Injuries of the Sclera

629. Injuries may affect the sclera alone with the over-lying conjunctiva or, as is almost invariably the case in ruptures and perforating wounds of the sclera, may extend to the deeper parts as well. Injuries of the latter class, which are always of a serious character, will be considered under injuries of the eyeball (§§ 853 et seq).

Injuries *confined to the sclera* itself produce no direct interference with function and rarely any serious consequences. Non-perforating and uncomplicated perforating wounds of the sclera have little tendency to gape, rarely become infected, and heal by primary union. Repair is effected mainly by the adjoining vascular episcleral tissue or in the case of perforating wounds by the uvea. The scar formed by any ordinary wound is quite inconspicuous; the overlying conjunctiva is attached to it, so that in this spot it cannot be displaced by movements of the lids. Deeper and especially perforating injuries may cause scars which are conspicuous because tinged with uveal pigment, or because either protruding or indrawn.

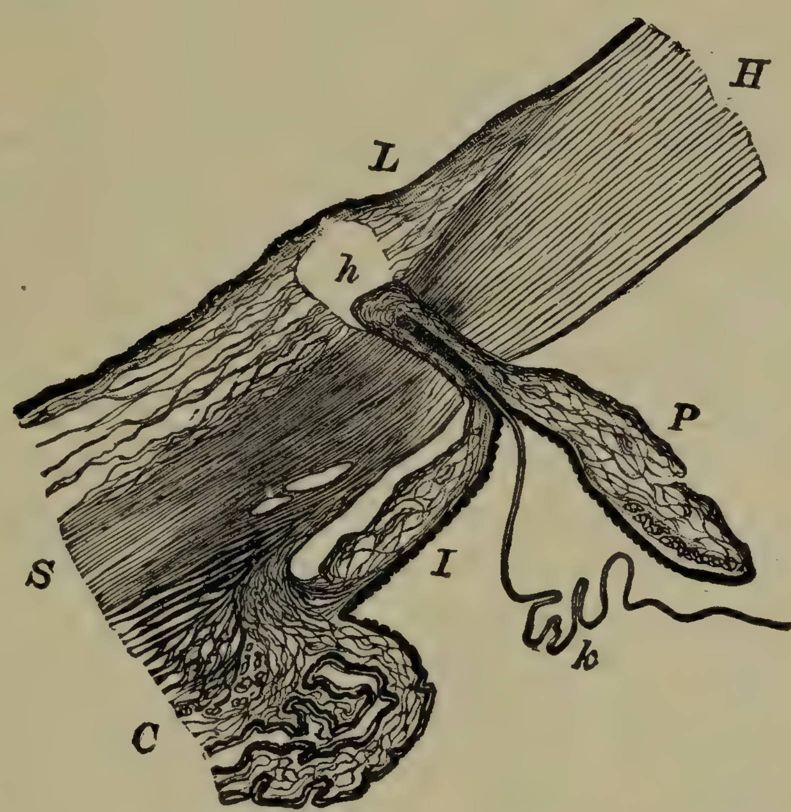


Fig. 279.—Inclusion of the Iris with Cystoid Cicatrization after the Extraction of a Senile Cataract by Von Graefe's Peripheral Linear Section. Magnified 13×1.

The section by which the extraction was made cut, in two the line of junction between the sclera, *S*, and the cornea, *H*, so that by its anterior half it lies in the sclera by its posterior half in the cornea. On account of the interposition of the iris, *I*, the lips of the wound have not united; in fact, the tract of the wound extends under the form of a cavity, *h*, even into the tissue of the conjunctiva of the limbus, *L*, so that the wound is closed only by a very thin layer of conjunctiva which, on account of the cavity beneath it, looks like a vesicle. The iris by being jammed into the wound is folded upon itself, the point of flexion corresponding to the exterior surface of the sclera. Peripherally from the site of its incarceration the iris being drawn tense runs in a straight line to the ciliary body, *C*; consequently, the anterior chamber is here reduced to a narrow slit, although the sinus of the chamber remains pervious. The pupillary portion of the iris extends centrally from the scar into the anterior chamber, where it lies free. On its anterior surface may be seen the entrance of a crypt, near its posterior surface the cross section of the sphincter pupillæ. Along with the iris, the much-puckered capsule, *k*, of the lens is, also, drawn up to the cicatrix, and has become adherent to it.

Scleral wounds that are near the corneal margin often close incompletely, the sclera gaping more or less, although the wound is covered by conjunctiva. This is usually caused by an intrusion of the iris or lens capsule into the wound, which keeps the lips of the latter from uniting. Through the gap thus formed, aqueous constantly exudes beneath the conjunctiva, which thus either takes on a gelatinous appearance near the scar or protrudes as a circumscribed cystoid swelling (*cystoid cicatrization;* see Fig. 279 and § 508).

Wounds of the posterior segment of the sclera are so covered by the orbital tissues that they require no *treatment*, but rest in bed. The quieter the eye is kept, the better the wound heals. Wounds of the anterior seg-

ment, since they communicate with the conjunctival sac, must be kept from infection by the use of detergent solutions, instillations of argyrol, or the application of a 1-per-cent collargol ointment. If such wounds gape, a scleral suture (see § 917) must be applied under rigid aseptic precautions. Such a suture prevents prolapse of the contents of the eyeball and effects a smoother and more rapid union.

III. Ectasiæ of the Sclera

630. Nature of Ectasiæ.—Ectasia of the sclera is the result of a disproportion between the intra-ocular pressure and the resistance of the sclera. Either the tension of the eye is pathologically heightened or the tenacity of the sclera is diminished. Often both causes are combined. Scleral ectasiæ develop slowly, and the disproportion between the tension of the eye and the resistance of the sclera must persist for a pretty long time before it can make the sclera become ectatic.

In the young in whom the sclera is more yielding it is possible for it to give way in all parts before an undue intra-ocular pressure. The ectasia is then *total.* In the great majority of cases, even when the pressure is excessive, and nearly if not quite always when the pressure is normal, ectasis takes place only at certain spots, which are either naturally more yielding or have become so through disease or because of a congenital defect. In that case the ectasia is *partial* and may be located either in the anterior, equatorial, or posterior segment of the eyeball (*anterior, equatorial,* and *posterior* ectasiæ).

The spots which are naturally weak and therefore give way readily to *excessive ocular pressure* are those where vessels or nerves perforate the sclera (lamina cribrosa, equatorial region where the venæ vorticosæ emerge, circumcorneal region where the anterior ciliary vessels are transmitted). The excessive intra-ocular pressure (glaucoma) may be primary or secondary. Primary glaucoma causes congestion in the venæ vorticosæ and hence distention in the equatorial region (equatorial staphylomata). In children it causes total ectasia. On the other hand, circumcorneal ectasiæ (anterior staphylomata) are ordinarily due to secondary glaucoma occasioned by seclusio pupillæ or ectatic scars of the cornea (see § 848). Both primary and secondary glaucoma cause recession of the lamina cribrosa—a condition, however, which is not usually classed as an ectasis of the sclera but as an excavation of the optic nerve (see § 827).

A much less common cause of ectasia is *diminished resistance of the sclera,* which makes it yield to even the normal intra-ocular pressure. It occurs in the deep form of scleritis, which leads to anterior scleral ectasiæ (§ 625); it also occurs when tumors (malignant new growths, gummy or tuberculous nodules) develop in or beneath the sclera. Injuries of the sclera also diminish its tenacity, and hence the scars after penetrating wounds (and especially after ruptures) of the sclera very frequently become ectatic. Scleral ectasiæ arising in this way lead subsequently to elevation of the intra-ocular pressure, which then, however, must be regarded not as

the cause, but as the result of ectasis, even though, just as in the case of ectasiæ of the cornea, it does contribute to make the latter larger still.

Posterior partial ectasiæ, other than the glaucomatous recession of the lamina cribrosa mentioned above, are likewise referred to a lack of resistance of the sclera at the posterior pole of the eye.

The sclera wherever it is ectatic is thinned so as to be readily dimpled with the point of a sound, and because of this thinning the uveal pigment shows through the wall and imparts to it a dark slate-gray or bluish-black, or in the case of total ectasiæ simply a bluish color. By means of focal illumination, light can often be made to pass through the ectatic sclera, so as to show the coating of pigment on its inner surface (cf. § 615).

631. Anterior and Equatorial Ectasiæ.—*Anterior ectasiæ* (anterior staphylomata) occupy the portion of the sclera adjoining the cornea (Figs. 280 and 281). They appear in the beginning under the form of small, dark spots, which afterward become larger and bulge out. When several lie close together they become confluent, forming a large swelling which surrounds the cornea like a ring. This swelling at various points is constricted in a radial direction by the stronger, less distended fibres of the sclera, so that in a small way it resembles the large intestine with its sacculations. The limbus, under the form of a somewhat depressed gray line, marks the boundary between the ectasia and the cornea. When the latter is also opaque and ectatic, the sharp line of demarcation between scleral and corneal ectasis is often lost, and both ectasiæ unite to form a single protuberance occupying the anterior segment of the eyeball. It often happens that an anterior scleral staphyloma exists, or, at all events, has its chief development, on one side only. Then the base of the cornea at this side is pushed forward, so that the entire cornea gets to lie obliquely. If, for instance, the scleral staphyloma occurs on the inner side, the cornea looks outward instead of straight forward (Fig. 281, *h*).

Equatorial ectasiæ (equatorial staphylomata) are dark prominences in the region of the equator of the eyeball. They can be seen only when the eye is turned far toward the side opposite the staphyloma. They occur at either one or more spots upon the equator, but never surround the entire eyeball like a ring, as is often the case with anterior staphylomata.

Morbid Anatomy.—In anatomical structure a scleral staphyloma is essentially different from staphyloma of the cornea. While the latter consists of cicatricial tissue which replaces the cornea that has been destroyed, a scleral staphyloma is formed of the sclera itself, which has not disappeared at the site of the ectasis, but is simply thinned, so that often it is no thicker than a sheet of paper. Often the thinning is not uniform, and commences suddenly, owing to the abrupt disappearance of the inner layers of the sclera at the margin of the ectasis. The sclera then in the spot where it bulges looks as if it had been gnawed into from the inner side, and thus deprived of its innermost layers (Fig. 276, *a*). The uvea is always solidly adherent to the inner surface of the ectasia, and is here so atrophic that scarcely anything is left of it but its pigment layer, which forms the dark coating of this inner surface.

An anterior scleral staphyloma may be of two kinds—*ciliary* or *intercalary staphyloma*. The former (Fig. 280) belongs to that part of the sclera the inner surface of which is coated by the ciliary body; the latter (Figs. 281, 282), develops in that narrow strip of the sclera which is situated in front of the ciliary body, between it and the margin of the

cornea and which lies in front of the root of the iris and in the region of the anterio chamber (Figs. 20 and 24). But although it is just in this portion that an intercalar staphyloma develops, the iris does not lie behind the latter but in front of it, just as in th case of a ciliary staphyloma. This comes to pass in the following way: The formatio of the ectasia is preceded by increase of tension, which causes the most peripheral portion of the iris to be pressed forward and to become united with the sclera (see § 839 and Figs 373 and 374). Hence that part of the iris lying free in the anterior chamber is given of from the sclera at a point farther forward than usual. Looked at with the naked eye, i seems as if the insertion of the iris had been pushed forward, up to the sclero-corneal junc- tion or beyond it. Now an intercalary staphyloma develops precisely in that region o the sclera which is united with the periphery of the iris—i.e., at l in Fig. 282, where the beginning of such an ectasia may be made out from the fact that just in front of the poin where the iris is given off the innermost lamellæ of the sclera have separated and the iri

Fig. 280.

Fig. 281.

Fig. 280.—Staphyloma Ciliare. (After Pagenstecher.) The eye is bisected horizontally. Surrounding the cornea there is an ectasia, c, of the sclera, which attains its greatest breadth at the temporal side, t; and on the nasal side, n, is narrower and less prominent, for which reason the cornea appears tilted toward the nasal side. The inner surface of the ectasia is coated with the elongated ciliary processes; the iris is invisible because it is pressed against the posterior surface of the cornea, which hence looks pigmented in black. The retina and chorioid have been to some extent separated from their bed by the dissection; in the retina groups of punctate hæmorrhages, b, are observable. The head of the optic nerve, O, shows a deep excavation due to pressure.

Fig. 281.—Staphyloma Intercalare. The eyeball is horizontally bisected and is depicted of somewhat more than the natural size. The ectasia, s, of the sclera is interposed on the nasal side, n, between the ciliary body, c, and the cornea, h, so that the latter is displaced and tilted toward the temporal side. The inner surface of the ectasia is covered with pigment, representing the remains of the root of the iris which has become adherent to the thinned sclera; this pigment in consequence of being spread over so large a surface, shows numerous gaps. Toward the outer side the ectasia constantly diminishes in breadth, so that, at the spot where the temporal wall of the eyeball, t, has been cut through nothing but a very narrow interspace is observable between the ciliary body and the iris, a condition due to that agglutination of the root of the iris to the sclera which characterizes an increase of tension. In the bisected optic nerve, i, no excavation is present; and, in the retina can be seen the fovea centralis, f, and the expansion of the retinal vessels.

has been pushed into the gap. Hence, too, later on and even when it has grown to be so very large, the ectasia always lies between the real origin of the iris at the anterior border of the ciliary body (Fig. 282, a) and its apparent origin at the spot where the portion of the iris that is yet free commences (Fig. 282, b). The inner surface of an intercalary staphyloma is coated with a layer of pigment, which is nothing but the completely atrophic root of the iris that has become united to the sclera.

In the living subject an intercalary may be distinguished from a ciliary staphyloma by the fact that in the former the anterior ciliary vessels are seen emerging at the posterior border of the ectasia, in ciliary staphyloma at its anterior border; furthermore, a thin ciliary staphyloma usually transmits light, and so admits of our recognizing the elongated ciliary processes as black striæ on its inner surface (c, Fig. 280).

Course and Treatment.—In anterior and equatorial staphylomata the sight is at length completely destroyed through rise of tension. Sometimes the ectasis comes to a stop, in other cases it keeps growing greater and

greater. The eyeball projects far beyond the palpebral fissure, can be covered but incompletely by the lids, and is extremely disfiguring. Conjunctival catarrh, lacrimation, and blepharospasm develop as a result of the mechanical irritation, and not infrequently the lower lid is pushed so far out by the enlarged eyeball as to be everted (ectropion). Finally, some slight injury suffices to cause rupture of the staphyloma at a particularly thinned-out spot. The greater part of the liquefied vitreous is expelled, and in consequence a violent hæmorrhage may take place, and the eye undergo destruction with the symptoms of panophthalmitis.

Anterior and equatorial ectases usually entail further changes in the interior of the eye. In consequence of the enlargement of the ring formed by the ciliary body, the iris

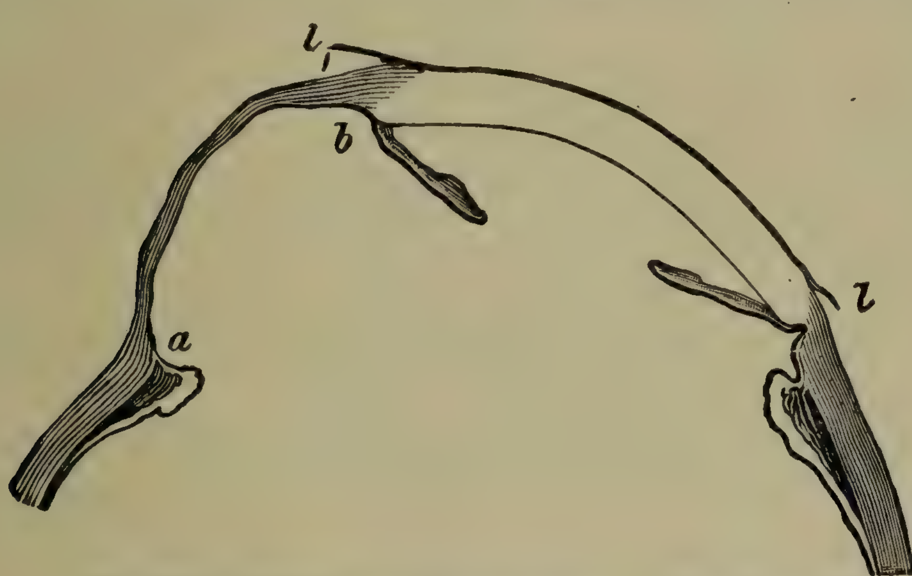


Fig. 282.—Intercalary Staphyloma. Magnified 4 × 1.

The figure represents a vertical section through the anterior half of the ectatic eyeball, which presents a great resemblance to the eye shown in Fig. 281, except that the most marked ectasia in the present case is situated above the cornea. The limits of the cornea are marked by the limbus *l* and *l*₁. At *l* may be seen how the root of the iris is applied to the sclera, and the beginning of a process of thinning in the sclera can be made out, while on the other side of the eye there is a fully developed intercalary staphyloma, which extends from *a* to *b*, and which in the living eye formed a dark translucent prominence. In the region of the staphyloma the sclera is reduced to half its normal thickness, and its inner surface is covered with a thin pigment coating representing the remains of the iris. The iris is adherent to the sclera from the ciliary body, *a*, to the anterior border, *b*, of the ectasia. The ciliary processes owing to atrophy, are flatter than normal.

becomes stretched and atrophic, and may even in places be separated from its insertion (spontaneous iridodialysis). The same is true of the zonule of Zinn, which, through atrophy, gets to be so deficient that the lens becomes tremulous or even undergoes luxation. The ciliary body, chorioid, retina, and optic nerve become atrophic; the latter generally presents a deep excavation due to the increase of tension (Fig. 280, *O*).

Wherever feasible *iridectomy* or some other operation to reduce the intra-ocular tension is indicated. This puts a stop to the further enlargement of the scleral ectasiæ (and in especially favorable cases even causes diminution in the size of an ectasia already existing), and likewise preserves the sight, so far as it still exists, from total destruction. If, as indeed is generally the case, iridectomy is on technical grounds no longer practicable there is nothing left to do but *enucleation,* in case the eye distresses the patient by its size, its painfulness, or the disfigurement it causes.

632. Posterior Scleral Ectasiae.—These can be recognized only with the ophthalmoscope, which shows that the portions of the fundus included in the ectasis are more myopic (less hyperopic) than the rest, because they are further back (§ 411). The posterior ectasiæ include—

1. *Recession of the lamina cribrosa* (excavation of the optic nerve) in glaucoma. The recession may amount to one or two millimetres, the amount being precisely measurable with the ophthalmoscope (§ 411). This condition is associated with characteristic ophthalmoscopic appearances (§ 827) and with the evidences of glaucoma.

2. *Staphyloma posticum Scarpæ.* This is a more or less hyperbolic protrusion wit thinning of the sclera, occupying all or part of the portion of the eyeball behind th equator. Its apex is in or near the fovea centralis, the degree of protrusion diminishin gradually from this point to the margin of the ectasis. It is the cause of the ordinar form of axial myopia and in its higher degree is associated with characteristic ophthal moscopic changes (§ 745). It tends to increase during the growing period and some times all through life and really represents an exaggeration of an ordinary developmenta process which is either excessive in degree or, because the sclera remains unusuall yielding, is continued during adult life instead of stopping at the usual time (§§ 191,199) It is not associated with increase of tension.

3. *Congenital ectases.* These include (*a*) ectasiæ at the nasal or lower side of th disc, which are evidenced ophthalmoscopically by a nasal or inferior crescent. Suc ectasiæ are not associated with myopia or at least not with myopia of any amount. (*b* Protrusions of the sclera accompanying colobomata of the chorioid (§ 769). The *sclera protrusion of Ammon* is such an ectasis associated with inferior coloboma. The con genital ectases are not progressive and not associated with increase of tension.

633. Total Ectasia.—Total ectasia can develop only in youth, when the sclera i still everywhere yielding. Even then the distention is rarely uniform. Usually th anterior segment of the eye is distended more than the posterior. It occurs most fre quently in conjunction with staphyloma of the cornea or with anterior scleral staphy oma. By a combination of these two kinds of ectasia a very extraordinary enlarge ment of the eyeball sometimes develops.

Much more rarely another, pure form of scleral ectasia is observed, in which the eye simply shows a uniform enlargement in all its dimensions (buphthalmus). For this condition which is really an infantile glaucoma, see § 836.

634. Other Morbid Conditions.—Tumors sometimes pass over from other parts of the eye to the sclera. Primary tumors of the sclera are very rare; they include fibromata, osteomata, sarcomata, and cysts.

Congenital anomalies include—

1. Anomalies of *form*—congenital posterior ectasiæ (§ 632) and congenital buphthalmus (§ 836). *Coloboma* of the sclera appearing as a fissure at the sclero-corneal junction with a tumor-like protrusion of the chorioid has been described (Harrington). The tumor was reduced and the gap covered with conjunctiva.

2. Anomalies of *pigmentation.*—*Melanosis scleræ* is one of the symptoms of melanosis bulbi and depends on the presence of chromatophores in the episcleral tissue. It is characterized by the presence of quite large, violet-gray or slate-colored spots found especially at the points of exit of the anterior ciliary vessels.

Sometimes the sclera is uniformly thinned (Buchanan) so that over its whole visible surface it appears markedly bluish instead of white. Such *blue sclerotics* are significant of a condition, often hereditary, which is associated with fragility of the bones and deafness.

CHAPTER XXIV

DISEASES OF THE INTERIOR REFRACTING MEDIA

I. Morbid Conditions in the Anterior and Posterior Chambers

635. The anterior chamber may show alterations in depth or contents. Changes in depth are caused by any condition, displacing either iris or cornea forward or back. The chamber is excessively *deep* in ectasiæ of the cornea (keratoconus, hydrophthalmus) or in recession of the iris due either to its retraction (total posterior synechia), to underdevelopment of the ciliary body (myopia), to excessive accumulation of aqueous (chronic iritis), or to atrophy, dislocation, or absence of the lens. It is abnormally *shallow* or actually *absent* in flattening of the cornea and in advancement of the iris.

The latter condition may be due to anterior synechiæ, seclusio pupillæ, swelling of the lens (incipient cataract, traumatic cataract), and forward displacement of the lens caused by diminution in pressure in front (perforation of the cornea) or increase of pressure behind (endophthalmitis, subretinal tumors, certain forms of glaucoma). The conditions causing changes of depth may affect part of the chamber only, in which case the latter will be of *unequal depth.*

Mixed with its normal *contents* (aqueous) the anterior chamber may contain pus (hypopyon; see §§ 554 and 676) or other inflammatory exudates, blood, and occasionally other matters.

Bleeding into the anterior chamber occurs in inflammation of the iris, injuries (especially iridodialysis), and sometimes spontaneously. The blood poured out first coagulates, the coagula being attached to the iris and often concealing the injury itself. In a day or two the coagula, owing to disintegration of the fibrin, liquefy, and the blood corpuscles are set free. There is thus formed a red mass (*hyphæma*[1]) at the bottom of the anterior chamber, which is obviously liquid since its upper border is level and remains so when the head is tipped to one side. Blood in the aqueous whether in the form of coagula or hyphæma is usually soon absorbed. In otherwise healthy eyes small quantities of blood may often disappear completely within twenty-four hours. The process of resorption lasts longer when there is much blood in the anterior chamber, and especially, when the eye is diseased in other ways as well and has not normal metabolism. Blood lying in the pupillary area often remains long after the blood on the iris has been absorbed. The longer the blood remains in the anterior chamber, the darker does its color become. So, in cases in which a hæmorrhage into the anterior chamber has been repeated after the lapse of some time, we see a hyphæma which is composed of two strata of different color; the lower dark stratum represents the first hæmorrhage, the upper bright one belongs to the recently extravasated blood. Very old extravasations of blood sometimes acquire a brown or dirty-green color, and the cornea, too, may take on a similar coloration (see § 603). The iris, too, when it borders on the hyphæma and sometimes after the hyphæma has been absorbed, looks greenish, the coloration here being imparted by the very thin layer of blood covering the iris.

Occasionally after effusion of blood into the vitreous and perichorioidal space, the hæmoglobin of the blood very suddenly dissolves and permeates the whole eye so that

[1] From ὑπό beneath, and αἷμα, blood.

the aqueous is tinged a ruby red but is transparent so that the iris is seen clea through it.

If the blood remains a long time in the anterior chamber it may—especially if th is a coexistent inflammation—serve as the substratum for the formation of new tiss In this way the good result of operations, such as iridectomy and iridotomy, designed the restoration of a clear pupil, is often rendered of no effect, as the blood extravasat during the operation covers up the opening that has been made and subsequently caus its re-occlusion by means of a membrane.

Among other substances found in the anterior chamber is *pigment*, usually under t form of deposits on the walls of the chamber (§ 690), and observed once under t form of a mass on its floor (Salzmann). Exceedingly rare findings are *cholester* filling the whole chamber with glittering crystals like bronze dust (Hebron) and *f* which because of its lightness collects in the upper part of the chamber (Leber).

Parasites found very rarely in the anterior chamber are the cysticercus and t larvæ of flies. The cysticercus forms vesicles which either lie free in the anter chamber or are attached to the anteror surface of the iris.

Cysts of Anterior and Posterior Chambers.—The posterior surface of the anter chamber formed by the iris is not lined with epithelium. Sometimes however it acqui an epithelial lining which shuts off the channels of outflow and then the anterior chamb is converted wholly or in part into a cyst-like structure (*anterior chamber cyst*). H this is possible is clear to us when we remember that in the process of healing in wounds the cornea the epithelium on the surface usually gows rapidly down into the deeper pa (epithelial indipping, Fig. 260). Sometimes it happens that the epithelium exten beyond the inner orifice of the wound and into the interior of the eye. In that case grows on along the walls of the anterior chamber and covers both the posterior surface the cornea and the anterior surface of the iris (Fig. 324, *D* and *b*). Such a formation an epithelial lining for the anterior chamber cannot be diagnosticated clinically, since t epithelium is transparent; but it is very destructive to the eye as it leads to increase tension because the epithelial lining hinders filtration through the sinus of the chamb

By the intrusion of an anterior chamber cyst into the substance of the iris an i cyst (§ 731), may be formed.

In extremely rare cases an anterior chamber cyst protrudes exteriorly at the an of the chamber forming a *scleral cyst.* In this case the wound canal which is lined w epithelium and is in communication with the anterior chamber becomes distended the accumulation of aqueous in it. Thus there is formed in the sclera at the corneal ma gin a rounded, very thin-walled cyst, which juts out beneath the conjunctiva pushi the latter before it. This is distinguished from a conjunctival cyst by being readi separable from the conjunctiva and by its intimate attachment to the eyeball (Sal mann).

Cyst-like structures (*pseudocysts*) may be produced by the fact that portions of t anterior chamber become sacculated by adhesions of the iris to the cornea, and portions the posterior chamber by adhesions between the iris and the lens—such sacculated po tions afterward dilating from accumulation of the liquid contained in them.

II. Opacities of the Lens (Cataract)

A. General Considerations.

636. Nature and Site of Cataract.—Opacities of the lens, called cat ract [2] may be situated in the lens itself or in the capsule. Accordingly, w distinguish between lenticular and capsular cataracts; by the combinatio of the two is produced capsulo-lenticular cataract.

Historical.—Cataract was already well known to the ancient Greek and Roma physicians. On account of the gray appearance of the pupil, they denoted it by the nam of glaucoma, which word in the course of time has changed its meaning. The ancien

[2] Waterfall, from καταῤῥήγνυμι, I pour down.

also knew the operation for cataract, which they performed by depressing the opaque lens into the vitreous by means of a needle (depressio cataractæ). Nevertheless, they had an erroneous conception of the nature of the disease, in that they located the opacity not in but in front of the lens. This error originated from the views that they held with regard to the function of the lens. This body, bright as crystal, the most obvious thing when the eyeball is opened, was considered by the ancients to be the true seat of vision, the percipient organ, such as now we know the retina to be. According to this view, the loss of the lens would necessarily entail complete blindness; but since the ancients knew that in the operation for cataract the opacity is removed from the pupil, and nevertheless the sight is not lost, but, on the contrary, is restored, they could not consistently regard the opacity as located in the lens. They thought the opacity which they depressed into the vitreous was situated in front of the lens. They believed that it originated from the pouring out of an opaque liquid between the iris and lens, and hence they called cataract hypochyma (ὑπό, beneath, and χέω, I pour) or suffusio, suffusion. Since it was imagined that the opaque liquid fell down from above in front of the lens, the name cataracta (cataract), which still is usually employed, came into use in the middle ages.

Our knowledge of the true nature of cataract dates from the beginning of the eighteenth century. Even before this, one or two savants, like Mariotte and Boerhaave, recognized the real situation of the opacity, without their doctrines', however, obtaining general acceptance. In 1705, Brisseau, a French surgeon, performed depression of cataract in a cadaver and then opened the eye, when he found that the opacity which he had depressed into the vitreous was the lens. He laid his observations and conclusions before the French Academy, but obtained no credence. The academy confuted him by holding up the doctrines of Galen in regard to cataract. It was not till three years later when new proofs had been brought, that the Academy recognized the new doctrine, which soon found general acceptance.

637. Objective Signs.—The objective symptoms of lenticular opacity vary according to its extent and its intensity—partial opacities often requiring for their recognition oblique or axial illumination and, if the opacities lie far in the periphery, artificial dilatation of the pupil as well. By oblique illumination the lenticular opacities appear under the form of gray or white spots or striæ. These often exhibit shapes which have a connection with the structure of the lens—e.g., the shape of sectors or radii. By oblique illumination also it can be determined at what depth the opacities are situated in the lens. Opacities of the anterior capsule are distinguished by their brilliant white hue, sharp outline, and very superficial situation; sometimes they form a distinct prominence upon the anterior surface of the lens. With axial illumination the lenticular opacities do not appear white, but dark, like black dots or striæ, which stand out in contrast with the red hue of the pupil (see § 398). Commencing, slight opacities of the lens can be recognized only by means of the ophthalmoscope (axial illumination with a faint light or the examination by the direct method with a +15 or 20D lens in the ophthalmoscope). Far advanced opacity of the lens can be recognized at a glance with the naked eye by the change of color of the pupil, which is white or a gray of varying degrees of brightness.

The *site* of a partial opacity is determined by the parallax (page 380) and of a total opacity by the shadow cast by the edge of the iris (see § 649).

In former times when focal illumination and the ophthalmoscope were unknown, it was quite possible for pupillary membranes, which made the pupil appear gray or white, to be regarded as opacity of the lens, and they were therefore called cataracta spuria. We

shall not fall into this mistake if we observe the connection which a pupillary membra almost always has with the margin of the pupil, and which is particularly marked when call in the aid of atropine.

638. Subjective Symptoms.—The main subjective symptom cataract is an *impairment of sight*, the degree of which depends upon t situation and the nature of the opacity. Patches of cloudiness that a small, sharply circumscribed, and at the same time as opaque as possible as, for example, anterior polar cataract—cause little or no impairment the sight. Larger opacities disturb the sight to a considerable degr Furthermore the diminution in visual acuity is greater when the opacity diffuse, less when it is sharply circumscribed, so that quite clear interspac are found between very opaque spots. This case is the same as with window-pane, through which nothing can be distinguished when it is ur formly covered with watery vapor; although, if a wire screen is placed front of the otherwise clear pane, we can still see pretty well through it. Tl interference with vision is also greater when the opacity is situated in tl central portions of the lens than when it occupies the periphery. In tl latter case, in fact, the sight may be perfectly normal; this being particular the case as long as the opacities continue to lie completely behind the iri Upon the situation of the opacities furthermore depends the sort of illum nation that will be required in order for the patient to see the best. With central opacity the sight is better when the pupil is dilated, because the sti transparent, peripheral portions of the lens are then used for seeing. Pe sons thus affected, therefore, see better when the illumination is reduced, in the evening twilight; they have *nyctalopia*. In bright daylight the complain of being dazzled, and shade their eyes with the hand. In such case the vision can also be improved by the artificial dilatation of the pup by means of atropine. The reverse occurs when the opacities occupy th periphery of the lens. Then vision is better when the pupil is contracte so that the opacities are covered by the iris. Such patients try to get bright light, and see better by day than night—*hemeralopia*.

Other symptoms present in the early stages of cataract are the presenc of a spot before the sight, getting in front of objects looked at, and polyopia If the spot before the sight is motile (*musca volitans*), it must be due to a accompanying opacity of the vitreous, such as, indeed, is frequently presen in incipient cataract. A spot before the eye due to an opacity in the len itself moves only with the eye, but a lenticular opacity rarely manifest itself in this way, being evidenced only by a general obscuration of vision *Polyopia monocularis* is the condition in which to the patients looking witl one eye an object appears double or multiple (§ 171). In cataract its caus is found in the optical irregularities which develop in the lens as it grow opaque (irregular lenticular astigmatism), so that the lens throws upon the retina not one but several images of the same object. These symptom (muscae and polyopia) often bring the patient to a physician at a time wher as yet no considerable diminution of the sight exists.

An opacity causes more disturbance of vision when it is situated at the posterio than at the anterior pole of the lens; for the nodal point of the eye—i.e., the point througl

which all rays must pass that enter the eye without undergoing refraction (principal rays) —lies close to the posterior pole of the lens (§ 161).

Myopia often develops in the beginning of senile cataract. In this case we are dealing with elderly people, who formerly saw well at a distance and used convex glasses for reading; and who then began to notice that they could read fine print again without glasses, and are perhaps very much pleased at this so-called "second sight." That, as an off-set to this, they do not see as well at a distance as formerly, often escapes their notice. Examination of the eye with glasses shows that it has become myopic, so that the near point has got back again to the reading distance. This myopia is ascribable to an increase in the density of the lens that takes place while the cataract is beginning to form, an increase by which the refractive power of the lens is heightened (see also § 648).

The *color sense* and the *visual field* are little or not at all affected in cataract even when the sight has become very poor. Hence the presence of a marked impairment of color vision, a restriction of the field, or a definite scotoma is an indication that the percipient parts (retina, nerve, or central connections) are abnormal. Tests to determine these points should always be made in cataract. In incomplete cataract, when there is still vision enough, these tests are made in the ordinary way.

If the lens is yellowish, as occurs in senile cataract and also in non-cataractous lenses in old people, color perception is somewhat affected, being, in fact, like that of a normal person by lamp light. Yellow appears nearly white, blue is poorly seen.

As the opacity increases, the sight becomes more and more reduced, the muscæ volitantes and the polyopia disappear, and the patient grows blind. But even when he has lost the ability to distinguish objects (qualitative vision), he has still always left him the perception of light. If this is deficient or entirely wanting there must be a complication affecting the retina or the optic nerve, in which case an operation for cataract would have little or no result.

When the cataract is *total*, the tests, if the eye is otherwise normal should show the following—

1. There should be good *light perceptions*, i.e., a candle flame set in a dark room at a distance of sex metres from the patient should be recognized instantly, (see § 185).

2. The *projection* tested with a light, whose situation is varied, should be normal (see § 265).

3. The *color sense* should be normal. This may be tested by placing colored glasses before the eye in a good light (Dimmer).

4. The light *reaction of the pupil* should be normal. In cataract this reaction is well marked even when the illumination comes from the side, since the light entering the eye, being widely diffused by the opacity, affects the pupillomotor zone of the retina, even when not directed at the latter.

639. **Morbid Anatomy.**—The anatomical changes forming the basis of lenticular opacity affect the lens fibres and the capsular epithelium, but not the capsule itself which never becomes opaque.

In the *capsular epithelium* we find destruction of the epithelium cells here and there and side by side with this, what may be looked upon as a regenerative process, namely,

a growth of epithelial cells. This latter process, however, takes place irregularly, so th close by the gaps in the epithelial layer, spots are present in which several rows of cells l on top of each other (Fig. 284 e_1). Moreover, the epithelium often grows backward pa the equator, so that the posterior capsule, too, gets to have an irregular epithelial coatir on its anterior surface. At the anterior capsule these cells by their proliferation form a *anterior capsular cataract,* which therefore lies between the capsule and the lens (Fi 283). The cells elongate into fibres forming a sort of fibrous tissue which looks like co nective tissue, but still is not true connective tissue since it has originated from epitl elium. By the interposition of this tissue between the capsule and the lens a distin elevation is formed upon the anterior surface of the lens. The opacities of the *posteri capsule* lie, as a rule, upon its posterior surface.

The opacity in the *lens* itself begins sometimes directly beneath the capsule, som times deeper in, at the border line between the nucleus and cortex. Here by sep

FIG. 283.—ANTERIOR CAPSULAR CATARACT. Magnified 40 × 1.

The capsular cataract forms a projection upon the anterior surface of the lens, covered by th capsule, *k*, which is unchanged and simply thrown into folds. The capsular epithelium *e*, loses its regu larity at the border of the cataract, its cells being increased in number and separated by the catarac from the capsule, so as to form for a short distance the posterior boundary of the cataract. The catarac consists of a fibrous tissue, with cells lying in the spindle-shaped gaps between the fibres. Succeedin the cataract posteriorly, is liquor Morgagni, *M*, which is coagulated into a pulverulent mass, separatin the capsule from the cataractous layers of the lenticular cortex (which are not represented in the drawing

aration of the lens fibres there are formed clefts filled with liquid. (These clefts hav been attributed to irregular shrinking of the lens; see § 655). The lens fibres themselves which bound the clefts, are at first still normal and hence transparent. (The fluid whic collects between them may at first be transparent, too, and nevertheless the spots loo cloudy, because the fluid in the clefts has a different refractivity from that of the len substance itself. So, too, a white opaque foam is produced when we mix transparen air with equally transparent water by agitation.) Afterward the lens fibres themselve become cloudy. They look at first as if sprinkled with fine dust, owing to the accumu lation in their interior of a fatty substance in extremely minute drops. At the same tim that the lens fibres get cloudy, their calibre becomes uneven, because they swell u in spots (*q*, Fig. 284). In this way bodies that are large, vesicular, and frequently nucle ated (vesicular cells; *b*, Fig. 284) are produced. Finally, the lens fibres break down com pletely, so that from the lens tissue is formed a pultaceous mass consisting of drops o fat, the so-called spheres of Morgagni (a coagulation product, Fig. 284, *M*), remain of lens fibres, and an albuminous liquor (Fig. 284, *z*). As the lens fibres break down the connection between them and the capsule, which is a very intimate one in the normal lens, is loosened, and a liquid (the liquor Morgagni) collects in open spaces between the lens and capsule (at *v* in Fig. 284, appearing under the form of separate vacuoles, but at *r*, Fig. 308, accumulated in greater amount and separating the capsule from the cortex). By this process the removal of the lens from the capsule, as is done in a cata- ract operation, is facilitated.

The *nucleus* of the lens is transformed by the sclerosis of the lens fibres into such a resistant mass that generally it remains unchanged in the midst of the disintegrating cortex (*k*, Fig. 308). Hence the nucleus of a cataractous lens is usually not essentially different from the nucleus of a healthy lens of the same period of life (Becker). But, if there is no hard nucleus present yet, the disintegration of the lens is complete (Fig. 309).

The *subsequent changes* in the opaque and disintegrated lens consist in the first place

in a gradual resorption of the pultaceous lens masses. In this way lenticular opacities may clear up again; not, to be sure, in the proper sense of the word, by the opaque lens fibres' becoming once more transparent, but by the disappearance of the opaque parts. The sclerosed nucleus resists resorption as it does disintegration. Cholesterin is not infrequently excreted in the cortex in tabular crystals, which are sometimes large enough to be visible to the naked eye as glistening points. Lime salts may be deposited in the pultaceous lens masses.

640. Replacement of Lens by Inflammatory Products.—The lens being an epithelial structure, is not subject to inflammation. But when the lens capsule is opened, exudate (pus) and afterward cicatricial tissue may enter the capsular sac. The opening in the capsule that affords entry of this sort may be produced by traumatism or by the eroding action of pus in the neighborhood. Pus entering in this way dissolves the lens substance, and more rapidly even than does the aqueous; and granulation tissue acts in the same way. The nucleus withstands this action; but when the cortex is

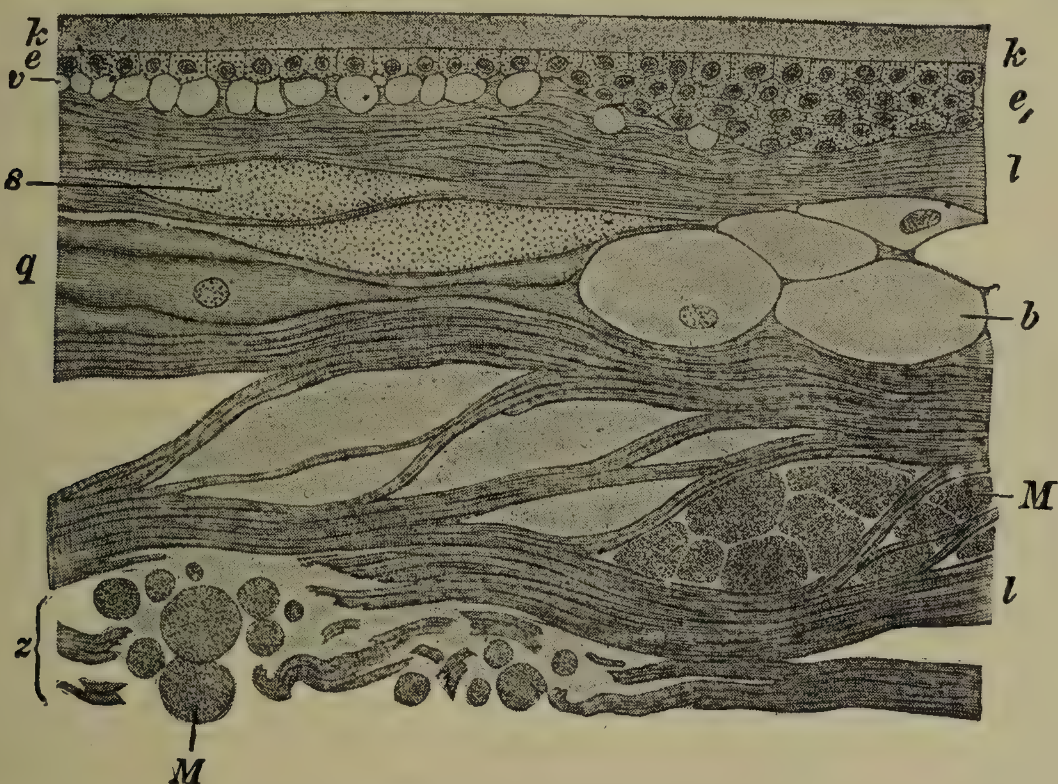


FIG. 284.—CATARACTA CAPSULO-LENTICULARIS. Enlarged 170 × 1.

k, anterior capsule of the lens; *e*, epithelium, occurring at e_1 in several layers because of proliferation; *l*, normal lens fibres; *v*, light-colored vacuoles (drops of liquor Morgagni) between *l* and the epithelium. The fissures originating through the separation of the lens fibres are filled with a granular mass (coagulated fluid), *s*, which in places forms the spheres of Morgagni, *M*. The lens fibres themselves are swollen up (*q*), or transformed into vesicular cells (*b*), or entirely disintegrated (*z*).

entirely replaced by cicatricial tissue, separating the nucleus from the capsule, the nucleus acts like a foreign body. Typical giant cells then appear on its surface and gradually eat their way into its substance (Salzmann).

Cicatricial tissue in the capsular sac may in time be converted into bone, forming an osseous mass surrounded by the capsule (so-called *ossification* of the lens).

B. *Clinical Forms of Cataract*

641. Every opacity begins at first at some special spot in the lens (partial cataract). It may remain permanently limited to this spot (partial stationary cataract), or it may gradually spread over the whole lens and lead to total cataract (progressive cataract).

(A) PARTIAL STATIONARY CATARACT

642. (1) *Cataracta Polaris Anterior.*—A small white dot is seen at the anterior pole of the lens. Anatomical examination has proved that it repre-

sents an opaque tissue lying beneath the anterior capsule and between it and the lens—that is, that we are dealing with a capsular cataract (Fig. 283). Hence this form of opacity is also called anterior central capsular cataract.

Anterior polar cataract may be either congenital or acquired. Congenital anterior polar cataract is bilateral, and consists of a minute faint dot upon the anterior pole of the lens. It is caused by some interference with the development of the lens, the precise nature of which has not been determined. The acquired form develops in early childhood and is caused by a perforating ulcer of the cornea, especially one due to ophthalmia neonatorum. When such an ulcer perforates and the aqueous escapes, the lens pushes forward so that its umbo comes into direct contact with the suppurating cornea. From the latter, toxic substances diffuse through the capsule of the lens and destroy its epithelium. Afterward a reactive proliferation of the adjoining epithelium, which has been preserved, takes place together with the formation of a new tissue beneath the capsule, this tissue appearing under the form of a white spot.

Acquired anterior polar cataract is bigger and more densely white than is the congenital form. Sometimes the layer of opaque tissue that lies beneath the capsule and corresponds to the cataract is so extensive as to produce a distinct visible conical protrusion of the anterior pole of the lens. This is called a *pyramidal* cataract (Fig. 273, *p*).

The *ulcer* which causes the cataract usually occupies the centre of the cornea. Sometimes, however, it is peripherally situated. That, nevertheless, the capsular opacity always occupies the anterior pole, is accounted for by the fact that in the area of the pupil—which is very narrow as long as the inflammation lasts—the lens capsule is directly exposed to the deleterious action, while in the rest of its extent it is protected by the iris. In rare cases deeply penetrating ulcers may cause an anterior polar cataract even when there is no perforation.

When by the perforation of an ulcer the lens becomes applied to the cornea, the two become glued together by a mass of exudation, which may afterward become organized and be drawn out into a long filament when the anterior chamber is reconstituted. Generally the filament ends by rupturing, but exceptionally it may persist all through life and connect the corneal cicatrix with the anterior pole of the lens.

Corneal opacities acquired in very early childhood often clear up to an extraordinary degree, so that in anterior polar cataract it is not always a dense cicatrix, but often only a slight cloudiness of the cornea, that is found. If this latter is overlooked, the method of development of the cataract might become a matter of doubt.

Anterior polar cataract, through subsequent contraction of the newly formed tissue, sometimes causes a wrinkling of the adjacent portions of the anterior capsule, visible upon examination with a magnifying glass. Beneath the capsular opacity disintegration of the adjacent lens fibres sometimes takes place. In such a case opacity of the entire lens (total cataract) may afterward ensue.

Anterior polar cataracts of small area may exist without giving rise to any essential disturbance of vision, this generally being caused more by the opacity of the cornea than by that of the capsule. *Treatment,* therefore, is generally not required except in those rare cases in which the cataract is so large as to occupy almost the whole of the pupil when the latter is contracted. Then an iridectomy would be indicated.

643. (2) *Cataracta Polaris Posterior.*—This consists of a small white dot at the posterior pole of the lens (Fig. 289), which, on account of its deep location, is generally to be discovered only with the ophthalmoscope. By

axial illumination it appears as a black dot which coincides with the corneal reflex no matter how the eye is moved. The dot usually represents the remains of the hyaloid artery (see § 58 and Fig. 55) deposited on the posterior capsule. It is hence also called posterior central capsular cataract. It is then sometimes associated with persistence of the hyaloid artery or with lenticonus. In other cases a posterior polar cataract is due to opacity seated in the lens itself. The interference with vision is inconsiderable when the cataract is small. Treatment, none.

644. (3) *Circumscribed Opacities of Various Kinds in the Lens itself.*—In this category belongs *central cataract*, a small spherical opacity directly in the centre of the lens. The *cataracta fusiformis*, or spindle-shaped cataract, consists of an opaque line which runs in the axis of the lens from the anterior to the posterior pole, and presents a spindle-shaped swelling at a point corresponding to the centre of the lens. In *cataracta punctata*, extremely minute white dots are found, either distributed uniformly through the whole lens or united in a group in the anterior cortical layer.

With the dots may be found striæ or little stellate figures. The smaller dots look bluish or even sky-blue (*cataracta cœrulea;* cf. § 649), the larger, which are rounded or oval, are dull gray. The spots can be seen only by oblique illumination. By axial illumination, being perfectly transparent, they are invisible. The disease is said to occur in connection with Mongolian idiocy and then to develop between the 8th and 17th year; in other cases probably it is congenital.

Allied to cataracta punctata is the *cataracta coronaria* of Vogt, which is said to be very frequent, developing usually about the age of puberty in the form of rounded or oblong opacities arranged like a crown in front of and behind what is later the nucleus, but so far to the periphery that they can be seen only with the dilated pupil. It has been regarded as constituting the first stages of a process which, if progressive, produces senile cataract.

Numerous other forms of circumscribed stationary lenticular opacities are known, all of which, however, occur so rarely that they do not need to be minutely described here. All the opacities above mentioned are sharply circumscribed, and are sometimes of very regular and graceful shape; they are mostly found in both eyes. They are often congenital and inherited, although the same forms of cataract are not always met with in the different members of the same family. Eyes affected with cataracts of this sort not infrequently present other congenital malformations too, or are found in individuals whose whole development, mental or physical, is imperfect. Most of these opacities in themselves cause little impairment of the sight, which, however, is often defective for other reasons.

645. (4) *Zonular (Perinuclear, or Lamellar) Cataract.*—This is the most frequent form of cataract in children. After dilating the pupil, we see a gray, discoid opacity in the lens, surrounded by a perfectly transparent marginal portion (Fig. 285). The diameter of the opaque disc is sometimes larger, sometimes smaller, and so, too, consequently, is the width of the peripheral, transparent zone (*P*). With the ophthalmoscope, the cataract appears as a dark, sharply defined disc surrounded by the red and illumined periphery of the pupil. The darkness of the disc is greater near the edge than in the centre. Along its margin, small opaque dentations called

riders, are not infrequently found projecting from the margin of the cataract into the transparent periphery, like the spokes on the steering-wheel of a steamship (in Fig. 285 represented only in the upper half of the cataract).

Zonular cataract does not always present a uniformly gray disc, but frequently exhibits a *complicated structure.* Densely opaque dots or graceful figures are often noticed in the anterior or posterior opaque layers, or markedly opaque sectors are contrasted with the less opaque matter in their vicinity. Again, while the riders correspond to partial opacities of a neighboring layer, the latter may also be opaque through its entire extent so as to surround the inner opaque layer like a cloak, while at the same time separated from it by a thin transparent stratum. Thus there originate double or even triple cataracts.

Zonular cataract is caused by an opacity of the layers lying between the nucleus and cortex (Fig. 286, *s*), while these two themselves are transparent (Jäger). The riders on

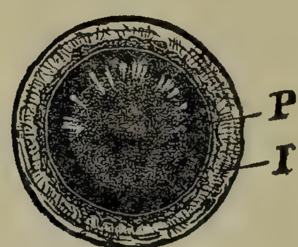


FIG. 285.

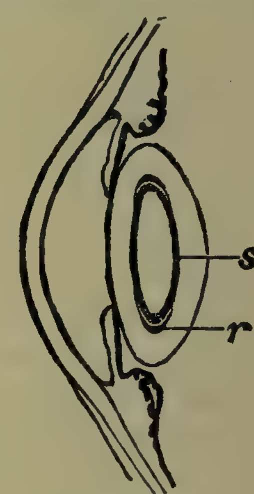


FIG. 286.

FIG. 285.—LAMELLAR CATARACT SEEN BY REFLECTED LIGHT. Magnified 3×2. The iris, *I*, has retracted under atropine. The opacity forming the lamellar cataract is denser at the margin than at the centre. The riders are depicted in the upper half, but are left out below to show how a lamellar cataract looks without them. Between the margin of the opacity and the margin of the pupil, *P*, is a black interspace corresponding to the transparent periphery of the lens.

FIG. 286.—LAMELLAR CATARACT IN CROSS SECTION. SCHEMATIC. Magnified 2×1. The layers *s*, placed between nucleus and cortex, are opaque, but the adjacent layer is so only in the equatorial region, *r*, so that here riders are formed.

the periphery of a zonular cataract signify that opacities are present in a second layer outside of the first, and that, moreover, they are present at first only at isolated spots along the equator of this first layer (*r*, Fig. 286). These partial opacities embrace the equator of the inner opacity in front and behind; they ride upon it, as it were, when ce the name riders. The microscope shows that within the opaque layer numerous small gaps, or vacuoles, filled with liquid are present between the lens fibres; while within the nucleus itself only one or two such vacuoles are to be found. In addition, larger fissures occur, surrounding the nucleus like a shell (Fig. 287).

Zonular cataract almost always affects both eyes. It is either congenital or, if not, originates in earliest childhood and is often hereditary. It is found, above all, in children who have suffered from convulsions (Arlt). At the same time there are found peculiar changes in the teeth (Horner), which, together with the cataract itself, are regarded as due to tetany (Peters, and others), although formerly both were attributed to rickets.

The teeth, especially the incisor teeth, in the slight cases, display horizontal rows of small depressions or horizontal furrows in the enamel (Fig. 288). When these defects are still more strongly marked, the tooth gets to have a sort of terraced shape, and is tapered down toward the incisor surface. Sometimes the teeth are so abortive in development that they are represented by small, cubical or irregular stumps. In the more

serious cases the enamel coating is absent on the incisor surface or is even absent altogether; the dentin lies bare, and upon its rough surface a thick layer of yellow tartar is deposited. Owing to this deficiency in the enamel coating, the teeth rapidly become carious and break off, so that it is not unusual to find, especially in peasants, nothing of the incisor teeth except the broken-off stumps. The lens, which in its development has much in common with the teeth, is also supposed to undergo some disorder during a period of its development, while later, after the disappearance of the disorder, normal transparent layers of lens substance are again deposited.

Zonular cataract is stationary as a rule, although there are cases in which it gradually develops into a total opacity.

The degree of interference with vision, due to the cataract, does not depend upon the diameter of the opacity, for zonular cataracts of even small diameter are always large enough to occupy the whole pupillary area of the lens, so that the transparent peripheral zone is always entirely concealed behind the iris when the pupil is not dilated. Hence, as far as vision is concerned, the denseness of the opacity is the only thing that has to be considered. As this varies greatly, all gradations are found between almost normal sight and considerable impairment of vision.

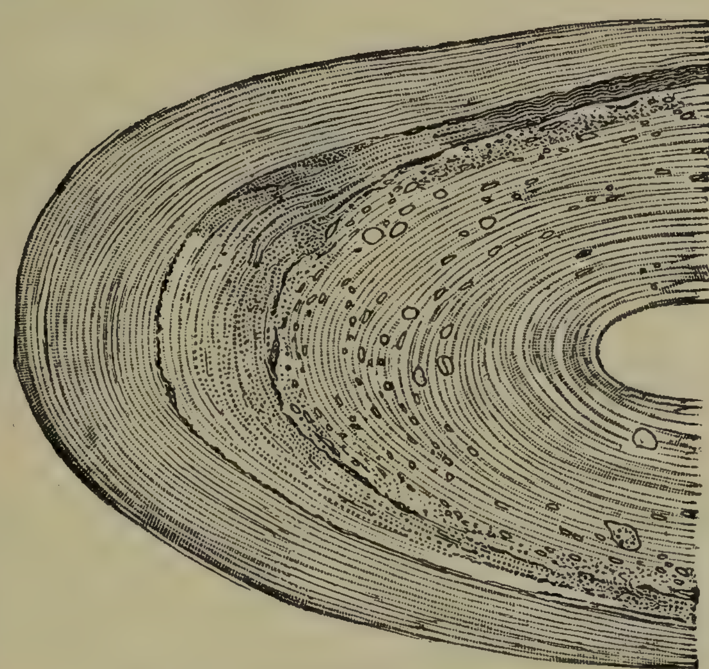


Fig. 287.—Cataract. Magnified 12×1.

The specimen was from a man, fifty-seven years of age, who had suffered since childhood from zonular cataract of both eyes. The diameter of the opacity, measured in the extracted lens, amounted to 6 mm. In the specimen the capsule and also the most superficial layers of the cortex are wanting, as they were left behind in the eye when the extraction was made. The layers lying between the nucleus and the cortex show numerous small cavities (droplets), which, in conformity with the fibrillary structure of the lens, are elongated, and are arranged concentrically. In some places they have coalesced so as to form larger cavities. Such droplets are found in the nucleus also, but in smaller number while the cortex is free from them. Besides the zone of vacuoles, which corresponds to the perinuclear opacity, two slender clefts are visible, forming arcuate lines, one of which is still within the zone of vacuoles, while the other runs along the peripheral border of the latter.

Children with zonular cataract, as with other partial opacities of the refracting media, very often become myopic.

Treatment consists in the correction of the myopia by proper glasses. If the opacity is so considerable as to cause marked impairment of sight we may remove the lens, either by discission, in young subjects, or by extraction, in older patients.

Optical iridectomy done to uncover the still transparent peripheral portion of the lens is usually unsatisfactory, because the central opacity remains, and the iridectomy itself causes disfigurement and gives rise to dazzling.

646. (5) *Cataracta Corticalis Anterior et Posterior.*—In this there is found in the anterior or posterior cortical layer of the lens a stellate or rosette-shaped figure, the centre of which corresponds to the pole of the lens, while its rays are directed radially toward the periphery (Fig. 290). Anterior cortical cataract is much rarer than the posterior variety; sometimes both are found together. The two forms of cataract occur generally

in those eyes which suffer from affections of the deep parts, like chorioidi retinitis pigmentosa, fluidity of the vitreous, etc.; the disturbance in nutrition of the lens, thus produced, induces the formation of an opac in it. The interference with vision is usually considerable, since it is caus not only by the opacity of the lens, but also by the involvement of fundus. Anterior and posterior cortical cataracts remain stationary

FIG. 288. TEETH WITH HYPOPLASIA OF THE ENAMEL FROM A MAN WITH ZONULAR CATARACT.
The teeth are yellow with rough surface. In most of them two furrows, separated by a protub ance, run around the crown. The protuberance represents an enamel of normal thickness, whil the course of the furrow the enamel is very thin. So, too, the little pits which are to be seen most d tinctly on the right upper canine tooth, indicate spots where the enamel is thinned.

many years and then at length pass into total opacity of the lens. The accordingly constitute a transition between the stationary and the pr gressive forms of cataract. When they have induced total cataract, the afford a bad prognosis for the operation on account of their being compl cated with a lesion of the fundus.

Posterior cortical and *posterior polar* cataracts are frequently confounded. Bo occupy the same site and hence both appear to move with the corneal reflex as the eye moved. Accordingly, a drawing is presented of the two kinds of cataract side by side show the difference. Polar cataract is a round dot (Fig. 289), and represents a tiss deposited upon the posterior surface of the posterior capsule of the lens, i.e., really outsi

FIG. 289.—POSTERIOR POLAR CATARACT. Magnified 2 × 1.

FIG 290.—POSTERIOR CORTICAL CATARACT. Magnified 2 × 1.

of the lens system. It hence shows no indication whatever of a radial structure, whic is the very feature that is characteristic of a posterior cortical cataract. The latter, i fact, is not only much larger than a posterior polar cataract, but also has always, in co formity with the radiating arrangement of the lens fibres at its posterior pole, the shape a star or a rosette, with coarse or fine radial striation (Fig. 290).

Cataracta corticalis anterior and posterior are sometimes observed after *injury the lens*, and that both when the lens capsule is opened and also in simple contusion of th lens without opening of the capsule. The stellate opacity in the cortex develops in th days immediately succeeding the injury and may either rapidly pass over into complet opacity of the lens, or may remain stationary, or may even disappear. The rapid devel opment of these opacities, as well as the fact that they can disappear, indicates that the consist in some change of the lens fibres which can abate again—perhaps being a swellin of the fibres without disintegration.

(B) PROGRESSIVE CATARACT

647. Course.—Progressive cataracts begin as partial opacities, which steadily extend until at length they occupy the entire lens. This is true, with the qualification that the portion of the lens already sclerosed—that is, the nucleus—ordinarily remains exempt from opacity. Hence, opacity of the lens in all its parts occurs only in young persons whose lens as yet has no hard nucleus; in older people the nucleus generally remains transparent. The time required for a lenticular opacity to involve all the parts that are capable of becoming opaque at all, varies very greatly. There are cases in which a transparent lens becomes completely opaque within a few hours, while other cataracts take many years to become total.

We distinguish in the course of a progressive cataract four stages, which are best marked in the most frequent form, senile cataract. To this form, therefore, the following description mainly applies:

648. (1) *Cataracta Incipiens.*—Opacities occur in the lens, between which are found spots that are still transparent. The shape of the opacity is most frequently that of sectors (so-called spokes) the base of which look toward the margin, and their apex toward the poles of the lens.

One of the precursors of cataract is the *unequal refractivity* that ophthalmoscopic examination frequently shows in the separate portions of the lens. In that case, when the pupil is illuminated with the ophthalmoscope, spots are seen here and there which first shine more vividly red than the rest, then upon a slight turn of the mirror appear as dark shadows in the red of the pupil. These are comparable to the striæ that are found in defective glass or in a block of ice.

Often the nucleus of the lens is distinguished by its specially great reflective power, so that, without being really opaque, it becomes visible upon ophthalmoscopic examination as a dark-red globular body, in the centre of the brightly illuminated pupil. In conjunction with the marked difference in refractivity between the cortex and nucleus that exists in these cases, a fairly high degree of shortsightedness (*lenticular myopia*) develops.[3] Such eyes show even to the unaided view a specially strong senile reflex; the pupil looks so gray that one might think himself justified in assuming the existence of a commencing cataract. The diagnosis of cataract, however, should be made only when the ophthalmoscope actually shows circumscribed opacities in the lens.

Such opacities appear most frequently under the following forms:

1. Opaque *sectors* (radii) which look grayish-white by reflected light, black by transmitted light, and whose apices converge toward the pole of the lens (Fig. 291). They correspond to the natural grouping of the lens fibres into sectors. Sometimes they are broad and triangular, sometimes narrow, and are occasionally represented by nothing but slender radiating lines. That form of cataract in which the lens is traversed by numerous, very slender radiating lines is found chiefly in myopic eyes. The clouding of the sectors begins in their periphery, where they are visible with the ophthalmoscope long before their apices project into the region of the pupil and impair vision. As a rule, they appear first in the lower portions of the lens.

2. A *diffuse, smoke-like cloudiness* occupies the central portion of the lens. It belongs to those layers which directly inclose the nucleus. This sort of opacity disurbs the sight much earlier and to a much greater extent than do the opaque radii, because, in the first place, it is found in the pupillary area from the start; and, secondly, because it is diffuse and does not leave any places that are quite transparent.

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[3] To the alteration in the nucleus, producing this increased refractivity of the lens as a whole, the name nuclear sclerosis is applied by some, although the term properly denotes any progressive hardening of the nucleus. The full amount of lenticular myopia produced by nuclear sclerosis (in this restricted sense) amounts according to some to 3 or 4 D.

3. A disc-shaped *opacity* which is situated *in the posterior layers of the cortex*, bu which, in contradistinction to the typical posterior cortical cataract (Fig. 290), present an irregular and ill-defined contour and a cobweb-like structure (Fig. 292). This sort o opacity, too, causes from the outset very great disturbance of the sight.

4. We find, as an extremely frequent occurrence, in the eyes of old people, a ring like opacity which lies near the equator of the lens, and which, on account of its resem blance to the arcus senilis of the cornea, was named by Ammon the *arcus senilis* (siv *gerontoxon*) *lentis*. This opacity is composed of two parallel opaque rings, one of whicl lies in front of the equator of the lens, and the other behind it. It does not impair vision since it lies wholly behind the iris, and, moreover, it shows little tendency to spread.

Frequently, in commencing senile cataract, several or even all of the above-men tioned forms of opacity are met with.

5. While ordinarily the *opacity of the anterior capsule* is not associated with a catarac until the latter becomes hypermature, it exceptionally happens that such an opacity precedes the lenticular opacity proper and so represents the beginning of the senile cata-

Fig. 291.

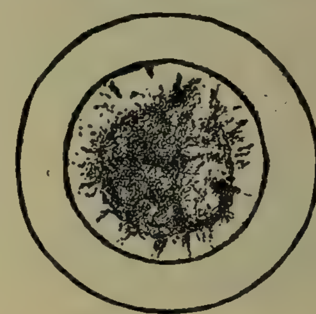

Fig. 292.

Fig. 291.—Incipient Cataract under the form of opaque sectors, which look black when seen by transmitted light with the ophthalmoscope.

Fig. 292.—Incipient Cataract under the form of an irregular disc which is more markedly opaque at its edges, and which is situated in the posterior layers of the cortex.

ract. A densely white dot forms in the centre of the pupil. This gradually enlarges by outgrowth taking place at its edge. Then the lens fibres directly adjacent become opaque, and finally the entire lens opacifies. The whole process runs an extremely slow course and takes a number of years before it produces complete opacity.

6. In young people, cataract often begins in the form of *irregular, macular* or *cloud-like* opacities.

649. (2) *Cataracta Intumescens.*—In proportion as the lens becomes more opaque, it contains more and more water, and hence swells up. The distended lens has a bluish-white color and a marked silky lustre of the surface, and shows very clearly the stellate markings of the lens. The increase in volume of the lens is recognized by the increasing shallowness of the anterior chamber. As long as the opacity has not reached to the anterior capsule, the iris throws a shadow upon the lens. To see this, a light is held near the eye and to one side of it. Then a black shadow is seen at that side of the pupillary margin that is turned toward the light (Fig. 293). This arises from the fact that the opaque layer of the lens upon which the iris casts its shadow lies some distance behind the iris. This opaque layer acts like a screen which receives the shadow of the iris. An observer, looking at the eye from in front, sees then that portion of the shadow which does not lie behind the iris itself (*a b*, Fig. 294). This portion of the shadow becomes narrower the nearer the opacity approaches the iris, and finally disappears altogether when the opacity reaches the anterior capsule.

A shadow is also cast by the edge of the iris in shrunken (hypermature) cataracts, being due in this case to the interposition of transparent aqueous between the iris and the

opacity (cf. § 651). This shadow must not be confounded with that which is found in immature cataract. Nor should the black rim of pigment on the margin of the pupil seen in every case of cataract, be regarded as the shadow cast by the iris. It is easily distinguished by its appearance, and also by the fact that it is visible not only at the side toward the light, but all round the iris.

During the stage of intumescence the opacity of the lens becomes total. As soon as this has taken place the lens begins gradually to lose water, so that it returns once more to its former normal volume. The lens then enters upon the stage of maturity (3) or is *ripe.*

The intumescence of the maturing cataract is caused by the swelling of the cortex. It is, therefore, more distinctly pronounced the softer the cataract is, since then there is much cortex; on the other hand, it is altogether wanting in the dark, hard cataracts which consist of scarcely anything but nucleus (cataracta nigra, see § 653). For the same reason

Fig. 293.

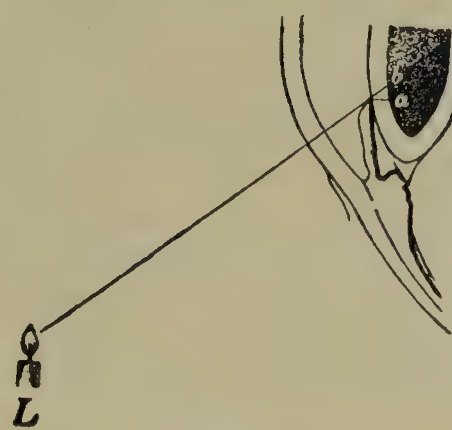

Fig. 294.

Fig. 293.—Shadow of the Iris Seen from in Front. The crescentic shadow appears at that side of the pupillary margin that is turned toward the source of light, *L*.

Fig. 294.—Shadow of the Iris upon the Lens in Schematic Cross Section. The inner layers of the lens are supposed to be opaque, the peripheral ones transparent. The source of light, *L*, throws upon the surface of the opacity a shadow from the iris, whose central border is at *b*. An observer looking from a point straight in front of the eye, sees a portion of this shadow, of the width *a b*, running alongside of the pupillary margin of the iris.

too, the ordinary phenomena of hypermaturity do not occur in the latter; instead of undergoing further disintegration, the horny lens remains unchanged, or, at most, thickening of the capsule is added.

The *progress* of a cataract is sometimes rapid, sometimes slow, the latter especially in senile cataract which not infrequently remains almost unchanged for years. Hence, if we find in an elderly patient the first stages of a cataract which as yet produces no interference with vision worth mentioning, it is best not to frighten the patient by communicating his condition to him, as he perhaps may enjoy sufficiently good vision for several years to come. For our own security we may communicate the discovery to some near relative of the patient's. Sometimes the lenticular opacity progresses by fits and starts—a cataract which has remained unchanged for quite a long time becoming almost completely mature within a few months, or even weeks.

For these reasons it is for the most part impossible to answer with precision the patient's question as to when the cataract will become ripe. The following diagnostic points may serve for an approximate determination: The lenticular opacity develops the more rapidly the younger the person is. Light-colored cataracts become matured more rapidly than dark ones, and those with broad radii more rapidly than those with slender radii. A cataracta nigra can never become matured in the ordinary sense of the word, since it is not a cataract proper but a sclerosis of the entire lens which may be said to have been converted in toto into a nucleus, and hence always preserves a certain degree of transparency. For the laity a criterion of the ripeness of a cataract—i.e., of its readiness for operation—is the fact that the eye is no longer in a condition to count fingers. This does not hold good for dark cataracts (see § 653) which generally do not

become so opaque that the patients cannot perceive large objects. Nevertheless, thes can be operated upon with good results, since the lens has been transformed into a hard horny, translucent mass which can readily be shelled out cleanly from its capsule Ordinary cataracts, too, are now operated on before maturity.

The rapidity of ripening is also influenced by the etiology of the cataract. Certai cataracts, such as diabetic, traumatic, and glaucomatous cataracts, furthermore com plicated cataracts, particularly those resulting from detachment of the retina, are dis tinguished by their rapid rate of progress. The time required for ripening can be mos readily determined, at least in the case of senile cataracts, if the other eye already con tains a ripe cataract and the time that this has taken to develop is known, since pre sumably the rate of advance of the cataract is the same in both eyes. But even thi guide may fail, for it happens not so infrequently that cataract advances at a very dif ferent rate in the two eyes.

Instead of progressing, the striæ and other opacities of an incipient cataract some times though rarely, undergo spontaneous absorption.

Patients with progressive cataract ought to be re-examined from time to time to determine their vision and *refraction.* For often in them the sight deteriorates not so much from the progressive opacification itself as from the changes in lenticular density and corresponding changes in refraction that the opacification produces (cf. §§ 638, 648). In such a case often a change of glasses will not only enable the patient to see better, but will give him the comfort of knowing that his cataract is not advancing as fast as he supposed.

Opacities of the lens, especially when examined with a loupe and focal illumination conjointly, often present quite clearly the appearance of being formed of minute drops. Sometimes these are of bluish color (cataracta cærulea cf. § 644). Such opacities are most frequently found in cataracts of young persons and in complicated cataracts.

650. (3) *Cataracta Matura.* (Stage of Maturity).—The anterior chamber is once more of normal depth, and the iris no longer casts a shadow—a proof that the opacity of the lens has become total. The lens has lost its bluish-white, iridescent look, and has a dull-gray or brownish color; the radial markings of the stellate figure of the lens are still for the most part recognizable. A mature cataract separates readily from its connection with the capsule. This is partly because the disintegration of the lens fibres has proceeded right up to the capsule, partly because the lens, formerly enlarged, has diminished again in volume, and thus the connection between the surface of the lens and the capsule is loosened. The lens then lies in its capsule like a ripe fruit in its rind (Arlt); and thus it has become ripe for operation, i.e., can be removed from its capsule without any portions of the lens remaining behind.

651. (4) *Cataracta Hypermatura.*—The further metamorphosis of a mature cataract consists in the complete disintegration of the opaque lenticular mass. This becomes converted into a pultaceous substance, which no longer shows any trace of the original structure of the lens, its formation out of sectors, etc. Hence, in a hypermature cataract we either see no markings at all, or nothing but irregular spots—no radii nor sectors. The consistence possessed by a hypermature cararact depends upon whether the gradual loss of water, which began after the intumescence of the cataract and reduced the latter to its normal volume, keeps on or not. If the loss of water continues, the pultaceous mass which is produced by the disintegration of the lens fibres becomes more and more inspissated. It dries up along with the nucleus of the lens into a flat, cake-like mass; the anterior

chamber consequently becomes deeper and deeper. This is the ordinary form of hypermature senile cataract.

If the loss of water ceases after the lens becomes entirely opaque, the lenticular mass grows more fluid in proportion as it keeps on breaking up into smaller and smaller parts. If this process goes on in a young person, in whom there is no hard nucleus in the lens, the latter becomes liquefied through and through, so that the lens consists of a milky fluid (*cataracta fluida* sive *lactea*). If this metamorphosis affects an old lens, the nucleus, which has failed to become opaque, also escapes disintegration, and sinks under the form of a heavy compact mass to the bottom of the liquefied cortex. The cataract has then a homogeneous white appearance, corresponding to the milky cortex, and in its lower portion presents a brownish shading, which is bounded above by a semicircular line, and which represents the upper half of the dark nucleus. As the latter alters its position with the movements of the head, the brownish shadow can also be seen to change its place, but if the patient lies on his back the nucleus sinks backward and the pupil then looks uniformly gray. This form of cataract is known as Morgagnian cataract (*cataracta Morgagni;* Fig. 295).

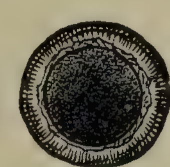

FIG. 295.—MORGAGNIAN CATARACT. Natural size. The pupil is dilated with atropine; the dark nucleus in this case is particularly big.

A liquefied lens, however, does not remain permanently unaltered, but inspissation occurs later through gradual loss of water, the disintegrated lens masses being at the same time in part resorbed. In this way the lens constantly diminishes in volume until, in cases in which there is no nucleus, it is transformed into a thin, transparent membrane (*cataracta membranacea*). In children, in whom resorption is carried especially far, the opaque lenticular masses disappear altogether in places. The two layers of the lens capsule, which has remained transparent, come into apposition, and thus there are formed perfectly transparent spots in the opaque lens, recognizable by reflected light as black gaps in the white pupil. The child begins to see again, a sort of spontaneous cure of the cataract having occurred.

When a hypermature cataract has lasted a long time, changes set in which lead to complications: (*a*) Cholesterin, or lime salts, are deposited in the lens mass. The former is recognizable with the naked eye under the form of glistening points in the opaque lens. Calcification of the lens (*cataracta calcarea* sive *gypsea*) takes place chiefly in complicated cataracts. It is characterized by a peculiar coloration, varying from chalk-white to yellow. (*b*) The anterior capsule becomes thickened by proliferation of the capsule cells, so that out of a simple lenticular cataract there is formed a *cataracta capsulo-lenticularis.* The capsular opacity presents itself under the form of a densely white, irregular spot upon the gray or brownish surface of the lens, usually occupying the central part of the anterior capsule, over an area about corresponding to the pupil (Fig. 296). (*c*) The lens becomes tremulous. The shrinking of the hypermature cataract affects not only its thickness, but also its equatorial diameter. In proportion as the latter diminishes in size, the zonula of Zinn is stretched, and thereupon undergoes

a corresponding atrophy of its fibres. Consequently, the attachment of the lens becomes imperfect, so that the lens shakes with the movements of the eye (*cataracta tremula*). Spontaneous luxation of the lens may even take place through partial or total rupture of the zonula. In consequence of these changes an operation for hypermature cataracts is often more difficult and gives rather less favorable results than the operation in the stage of maturity.

By a combination of thickening of the capsule with various degrees of consistence of the lens special varieties of cataract are produced. A liquefied lens in a thickened sac-like capsule is called *cystic cataract* (*cataracta cystica*). By the term *cataracta arida siliquata* is understood a shriveled cataract within a thickened capsule; deriving its name (dry, pod-like cataract) from its similarity to a dried pod (silique).

By the shriveling of the cataract in the stage of hypermaturity the anterior chamber becomes deeper, until finally the iris, instead of projecting forward in the shape of a cone, lies in a plane. If the diminution in the size of the cataract keeps on, the iris is not drawn backward so as to form a funnel, except when it is joined to the lens capsule by posterior synechiæ. Otherwise the iris remains stretched in a plane, and the shriveling lens becomes farther and farther removed from the iris, so that the latter deprived of its support, becomes tremulous. A dark interspace is then seen between the iris and the lens, and the iris again throws a shadow upon the latter (cf. § 649).

FIG. 296.—CAPSULAR CATARACT IN A CASE OF HYPERMATURE CATARACT. The centre of the dilated pupil is occupied by an irregular, brilliantly white capsular thickening, upon which can be recognized the fine wrinklings of the capsule. In the vicinity of the large capsular opacity are found thickenings of the capsule, which have just developed, and which form white dots contrasting strongly with the subjacent brownish and radially striate, opaque lens.

In the stage of hypermaturity in which the opaque layers become thinner through resorption, the sight often increases a little, so that, for example, the fingers can be again distinguished. Really serviceable vision sometimes comes on in young people, when the resorption goes on so far that spots are formed which are perfectly transparent. In senile cataract, in which a hard nucleus is present, it is extremely rare for a spontaneous restoration of sight to occur, although it may take place in the following ways: (*a*) By resorption, in exceptional cases, not only of the cortex but also of the nucleus to such an extent that nothing but slight opacities remain. (*b*) By the formation of a Morgagnian cataract, and the subsequent transformation of the fluid portion of the cataract into a clear, transparent liquid. Then the upper part of the pupil is transparent and black, while the brown nucleus is seen lying in its lower part. These cases are not so very rare, only formerly they were not correctly diagnosticated. Afterward the transparent liquid, and even the nucleus itself, may be resorbed, so that only a thin membrane is left. (*c*) By spontaneous dislocation of the lens, so that the pupil again becomes partly or entirely black.

652. Diagnosis of Stage.—As the diagnosis of the stage of a cataract is what determines the question of the performance of an operation, and hence is of great practical importance, the distinguishing signs of the separate stages will be summed up in the following words:

1. Cataracta incipiens. Anterior chamber of normal depth; transparent spots still to be found in the lens between isolated opacities.

2. Cataracta intumescens. Anterior chamber shallower; iris usually casting a shadow; lens bluish-white and having a silky lustre; markings of the stellate figure of the lens very distinct.

3. Cataracta matura. Chamber of normal depth; no shadow cast by the iris; markings of the stellate figure of the lens still recognizable.

4. Cataracta hypermatura. Anterior chamber of abnormal depth; no shadow cast by the iris (except in shrunken cataracts); surface of the lens appearing quite homogeneous (in the case of liquefaction), or showing irregular dots and spots in place of the radial markings of the lens-star.

653. Hard and Soft Cataracts.—According to their *consistence* total cataracts are distinguished into hard and soft (*cataracta dura* et *mollis*). This has reference to the nucleus of the cataract. By soft cataract we understand one having no distinct hard nucleus (Fig. 309), while those cataracts are known as hard which inclose a hard nucleus, although the cortex is soft (Fig. 308). The nucleus, in fact, does not usually become subject to cataractous changes, and therefore retains its natural consistence. The distinction between hard and soft cataract is made chiefly from a practical point of view. For the extraction of cataract from an eye a section must be made, the dimensions of which depend mainly upon the size of the nucleus. The wound must be large enough for the nucleus to pass easily through it, as otherwise the nucleus either cannot be removed from the eye at all, or, if it is squeezed forcibly through the wound, it crushes the lips of the latter. The soft cortex is stripped off from the nucleus, as the latter passes through the wound, and can subsequently be readily removed from the eye by stroking; it is unnecessary, therefore, to pay any regard to it in making the section. Hence, in soft cataract a small wound (simple linear extraction) suffices, while in hard cataracts the wound must be the longer, the bigger the nucleus is.

For these reasons it is of importance before undertaking an operation to diagnosticate whether the cataract contains a hard nucleus, and if so, about how large it is. For this purpose we must take into account the age of the patient and the appearance of the cataract. The age is to be considered, inasmuch as the development of the nucleus is in direct proportion to it in healthy, and hence also in cataractous, lenses. Cataracts of children and young people have no nucleus; in older persons the nucleus is in the main the larger, the more advanced the age is. Nevertheless, it is not to be forgotten that very considerable individual variations occur with regard to the size of the nucleus. A careful inspection of the cataract, however, enables us to see the nucleus directly. It can be recognized upon oblique illumination as a dark reflex emanating from the depths of the lens. From this we can estimate its size, and from its color estimate its consistence also, The darker—reddish or brownish—the nucleus is, the harder (and usually, too, the larger) it is.

It may happen that the sclerosis of the lens has advanced so far that it has been entirely, or all except a small residue, transformed into a nucleus. It is then converted into a hard, dark-brown, transparent mass. The pupil looks black, and it is only on careful inspection—especially with the aid of lateral illumination—that we recognize that it is of a peculiar dark brown. This condition is known under the name of *cataracta nigra* (or *brunnescens*). Properly speaking, it is not a cataract in the true sense of the term, but a far-advanced senile alteration (total sclerosis) of the lens. Such lenses are always large and hard, and require a large section for their removal.

To what has been said in regard to the nucleus of the lens and its behavior in cases of cataract, exceptions occur. There are cases of cataract in children, in which the lens, instead of being soft, has quite a hard nucleus or even has a wax-like consistence throughout. On the other hand, cases of senile cataract have been observed without nucleus. In exceptional cases, the nucleus and not the cortex may be the first part to become opaque in the lenses of adults (*cataracta nuclearis*).

C. Etiology of Cataract

654. (1) *Cataracta Congenita.*—The cause of this is either a disturbance of development or an intra-uterine inflammation of the eye. Both the stationary partial cataracts (particularly anterior and posterior polar cataract) and the progressive forms of cataract may be congenital. Congenital cataracts are usually bilateral and often inherited. Heredity, however, makes its influence felt in non-congenital cataracts also, and, in fact, even in senile cataract; there are families many of whose members become blind from senile cataract, and that, too, for the most part at an uncommonly early age.

It is only in exceptional cases that *congenital cataracts* are discovered immediately after birth, the rule being that they are not made out until the child is some weeks or months old; for newborn children have very narrow pupils, and moreover, because they sleep so much, keep their eyes shut most of the time, so that no notice is taken of the fact that their pupils are not black. Then, too, as such young children do not fix their eyes steadily upon objects, the fact that they do not see is not obvious. Partial congenital cataracts, if they do not cause any notable impairment of sight, are often not noticed until the patient is of quite a mature age, or perhaps are never discovered at all. Many congenital cataracts are complicated, as can be seen from the changes found at the same time in the iris, expecially posterior synechiæ. They are hence the result of a fetal inflammation of the uvea. The formation of the cataract must in many cases be dated pretty far back in intra-uterine life, since children sometimes come into the world with cataracts that have already become shriveled.

Congenital cataract is quite a frequent condition. Of 3300 cases of partial or complete blindness in children investigated by Harman, 284 were due to congenital anomalies of the lens.

655. (2) *Cataracta Senilis* is by far the most frequent form of cataract. Indeed, it occurs very frequently in old people, but not so regularly as to be regarded as a physiological attribute of age—as the turning gray of the hair is, for example—but rather as a pathological process. It usually does not make its appearance until after the fiftieth year of life, but is exceptionally observed in the years between forty and fifty. The fact of a cataract occurring in an elderly person does not of itself justify the diagnosis of senile cataract. An old man may get a cataract in consequence of traumatism, etc. Hence, to establish this diagnosis, it must be demonstrated that neither in the eye nor in the body in general are diseases which might account for the development of the cataract, so that the latter can be attributed only to the effect of age. Senile cataract always affects both eyes, but rarely at the same time, so that generally one eye is in advance of the other in respect to the development of its cataract.

The *cause* of senile cataract is still a matter of conjecture. It has been ascribed to—

(1) Purely *local causes*. In the process of transformation of the inner layers of the lens into nucleus (sclerosis) these layers diminish somewhat in volume. Under normal con-

ditions this process of shrinking is conducted so slowly and gradually that the cortical layers are able to adapt themselves to the diminished volume of the nucleus. But, if the shrinking goes on with exceptional speed or irregularity, there may be produced undue traction and subsequent separation of those layers of the lens which lie between nucleus and cortex. In this situation fine fissures are formed in which fluid accumulates; afterward the adjacent lens fibres themselves become opaque, and thus afford the initial impulse which leads to the opacity of the entire lens (Becker).

(2) Not very dissimiliar is the suggestion of Treacher Collins that cataract is due to *abiotrophy* or a want of vital force (§101), that causes the fibres outside of the nucleus (which latter represents fibres that have undergone a natural degeneration) to become degenerated before their time.

(3) *Altered composition of the aqueous*, resulting in injury to the epithelium of the anterior capsule. The alteration of the aqueous might be due to local (senile) changes in the ciliary processes which secrete the aqueous (Peters) or to disturbances of the general metabolism, e.g., such as occur in chronic nephritis (Michel) or to accumulation of toxic decomposition-products (cytotoxins) in the body (Römer).

(4) The action of *light*, especially light rich in ultra-violet rays. This view receives some substantiation from the fact that the first sectors of opacity develop in the lower portions of the lens which are most exposed to the sun's rays as they come from above, and from the experiments of Burge who found that lenses exposed to light became opaque, provided they were at the same time charged with sugar or certain salts of calcium, magnesium, or silicon. It further appears that cataractous lenses do contain an unusual amount of magnesium and calcium salts and in the case of Indian cataracts an unusual amount of silicon. If these findings are substantiated for cataract in general, it would seem that the development of cataract would represent the combined effect of light acting in a prolonged or excessive manner on the eye and of some disorder of metabolism acting to charge the lens with sugar or mineral salts. Van Der Hoeve points out that rays, especially ultra-violet rays, are not only converted into waves of longer wave length in the lens (causing fluorescence) but are also diffused (laterally dispersed). By this diffusion they are prevented from reaching and affecting the retina, but are thrown into the ciliary body, where, he believes, they set up morbid changes which produce disturbances of nutrition in the lens causing cataract (cf. § 743).

(5) *Heat.* The action of the infra-red rays is apparently responsible for *glass-blowers'* cataract. This is a very slowly developing posterior cortical opacity, occurring according to Legge in 30 per cent of glass workers over 40. It affects those especially who have for over twenty years been exposed to the hot rays emanating from the glowing glass and occurs especially in the left eye, which is habitually turned toward the latter.

656. (3) *Cataracts due to General Diseases and Toxic Conditions.*—In this category belong——

1. *Diabetes.* This mainly develops when the amount of sugar in the urine is high, and usually matures rapidly. It is always bilateral.

Diabetic cataract is probably attributable to morbid changes produced by altered composition of the fluids of the eye. Some have thought of these changes as consisting in the abstraction of water from the lens by the action of the sugar-charged eye liquids. But this view has been disproved. (Really dependent upon abstraction of water is probably the cataract that sometime develops in the last stages of *cholera.*) For the possibility that diabetic cataract may be due to the light acting upon a lens containing sugar see § 655.

The prognosis of diabetic cataract, as far as the operation upon it is concerned, is less favorable than in senile cataracts, because in diabetes wounds show less resistance to infection, and, moreover, diabetes predisposes to iritis. Hence, if we have to operate upon the diabetic cataract, we wait until by suitable treatment the amount of sugar in the urine has been reduced to the lowest possible point. It is said that in diabetic cataracts which have not advanced too far a partial disappearance of the opacities has sometimes been observed after a successful treatment of the diabetes.

Not every cataract that is seen in a diabetic patient is a diabetic cataract. Di betes being of common occurrence in advanced life, it often happens that opacities of t lens are found in patients affected with it. Such opacities must be regarded as sen cataract, if they exhibit the ordinary appearance of the latter and the slow developme characteristic of it.

2. *Tetany.* This in young children causes zonular cataract, in adults rapidly developing, soft, total cataract. In adults it usually develo between the ages of 30 and 40.

Tetany seems to be a fairly common cause of presenile cataract. As the diagno of tetany is sometimes difficult, this cause may be readily overlooked. Tetany and i resultant cataract may occur after removal of the thyroid or parathyroid glands.

Likewise occurring between 30 and 40 years of age is the total cataract produced *myotonia atrophicans*, a condition which like tetany is attributed to endocrine disorde

3. *Other Convulsive Diseases.* Cataract sometimes occurs in connectic with epileptic, eclamptic, and perhaps hysterical convulsions.

4. *Poisons.* A cataract, resembling an incipient senile cataract h been produced by *naphthaline* (e.g. in workers in naphthaline or by t application of napthol ointments). Other forms of cataract following t ingestion of poisons are those that appear in *ergotism*, *rhaphania*, an *pellagra*, being perhaps due to the convulsions that occur in these disease

657. (4) *Cataracta Traumatica.*—All injuries which make an openin in the lens capsule result in opacity of the lens. When the lens fibres com into contact with the aqueous, they become permeated by the latter, swe up, and break up into layers through a process of cleavage. When t traumatism affects the posterior capsule, the vitreous acts in the same wa as does the aqueous.

The opening of the capsule usually occurs through direct injur inflicted by means of a punctured or incised wound, through the penetra tion of a foreign body, and also designedly through an operation (discission Contusions of the eyeball also, which do not perforate its tunics, may caus opacity of the lens. In many of these cases it is likely that rupture of th capsule, probably in the region of the equator of the lens, is caused by th contusion. But it is also a fact that lenticular opacity is caused by simp concussion without opening of the capsule.

The development of cataract after injury of the capsule proceeds i the following way: As early as a few hours after the injury the lens is foun to be clouded in the vicinity of the capsular wound. Soon swollen len fibres protrude through the capsular wound, so as to project under the for of gray flocculi into the anterior chamber. Later on these break off and fa to the bottom of the chamber. Sometimes the entire chamber is found t be filled with the swelling and disintegrating fragments of the lens. Whil these prolapsed masses of lens substance are becoming gradually smalle through resorption and finally disappear, new flocculi keep protrudin through the capsular wound. At the same time the opacity spreads farthe and farther in the lens itself, so that usually within a few days the lens i opaque throughout. In favorable cases the lens may disappear com

pletely by gradual absorption, so that the pupil becomes clear and black, and thus a spontaneous cure of the cataract takes place. In most cases, however, resorption comes to a stop earlier from reclosure of the capsular wound. Then opaque portions of the lens still remain in the shrunken capsular sac and form a shriveled cataract, which requires an operation for the restoration of sight. So, too, when there is an actual solid nucleus, as is usually the case by the age of 30, this is not absorbed but remains in situ.

In younger subjects the portion of the lens which represents what is later the nucleus, being of greater consistence than the rest, may be extruded into the anterior chamber as a rather large opaque lentiform mass which takes a long time to absorb.

The younger the subject the more quickly and smoothly does the process of absorption go on. In infants it may be completed in a few weeks; in older children up to the age of puberty it takes one or two months.

In traumatic cataract it is the rule that the opacity of the lens becomes total, spreading rapidly from the site of the wound in the capsule to the rest of the lens. Exceptionally however, cases are observed in which the opacity of the lens remains *partial* or indeed actually *disappears* again. For this to occur, the capsule wound must be very small, so that it closes quickly and the aqueous no longer has access to the lens fibres. Most favorably situated in this regard are those capsular wounds that lie behind the iris, by the adhesion of which to the wound the latter is very soon closed up. In such cases it may happen that a circumscribed opacity remains confined to the site of injury, or, if a foreign body has penetrated the lens, is found only along the track of the wound. By resorption of the opaque portions the opacity itself may even in part clear up again. Sometimes, too, as a consequence of injury, stellate anterior or posterior *cortical cataracts* develop, which may likewise remain stationary or may even retrogress (see § 646).

When a soft cataract has been operated on by discission and is consequently swelling up and undergoing absorption, minute deposits are often found on the posterior surface of the cornea. These *lens deposits* resemble the precipitates of cyclitis (§ 677) but are essentially different in character; they are not agglomerations of exudation, but small rounded lens fragments, detached from the swelling lens and thrown against the posterior surface of the cornea, to which they then adhere. They are therefore in no sense to be regarded as a sign of inflammation; and this view of their nature is confirmed by the favorable course that such cases pursue.

A special form of traumatic cataract is that produced by *lightning* stroke or *electric* shock.

The *annular opacity of Vossius* is produced by contusion of the eye. It is a faint, gray ring on the front of the lens, having the size and shape of the pupil. Invisible by oblique illumination, it is seen best by axial illumination with the plane mirror. It consists of a deposit of blood (Hess) or pigment (Vogt) on the surface of the lens. It causes no symptoms and is absorbed after some weeks.

The course of traumatic cataract is unfavorable when complicated by either inflammation or increase of tension. *Inflammation* is, for the most part, the direct consequence of the traumatism, by which the membranes of the eye (particularly the uvea) either suffer a severe mechanical injury or undergo infection at the same time that the cataract is produced. Then the clouding of the lens and the inflammation (irido-cyclitis) go on simultaneously. The inflammation leads to the adhesion of the opaque lens to the neighboring parts, specially the iris and ciliary body (*cataracta accreta*), and by this adhesion the operation for the cataract is rendered difficult. In the severest cases the inflammation is so violent that alone it suffices to cause destruction of the eye, either as a result of panophthalmitis or through plastic irido-cyclitis terminating in atrophy of the eyeball.

Slight inflammations of the iris may also, it is likely, occur secondaril as a result of the swelling of the traumatic cataract, owing to which the iri is subjected to pressure or to traction; or, according to Verhoeff, uveal in flammation may be produced by the direct effect of the extruded len matter on an eye which is over-sensitive to lens protein (endophthalmiti phaco-anaphylactica).

Increase of tension may also be caused by a swelling traumatic cataract These cases are not very dangerous if they come under the observation o a physician, since the increase of tension can be done away with by timel interference (by paracentesis of the cornea, by removal of the lens, or by iri dectomy). But if such a case does not receive the proper treatment, th sight is usually destroyed through excavation of the optic nerve.

658. (5) *Cataracta Complicata.*—By this term we mean cataract occurring as the result of other diseases of the eyeball. What occurs in thi case is either an alteration in the character of the nutritive material whic the lens receives from the tissues surrounding it, or a diffusion through th lens capsule of deleterious substances produced by inflammation—the mai result of such diffusion being that the capsular epithelium is injured. Th affections of the eyeball most frequently leading to the formation of catarac are: (*a*) violent inflammations in the anterior sections of the eye, such a extensive suppuration of the cornea (particularly that produced by ulcu serpens) and irido-cyclitis, together with less active inflammations (especi ally heterochromic cyclitis); (*b*) sluggish inflammations in the posterio sections of the eye, such as chorioiditis (particularly irido-chorioiditi chronica), myopia of high degree, retinitis pigmentosa, detachment of th retina; (*c*) glaucoma in the stage of glaucoma absolutum (*cataracta glau comatosa*).

The diagnosis that a cataract is complicated may be made when ther is a disease of the anterior section of the eye, from the fact that morbi changes are found in the cornea or iris, and also adhesions between thes organs and the cataract. Even if the pathological changes which have le to the production of opacity in the lens appertain to the deeper portions o the eye, and are not visible from the outside, the cataract often, by it peculiar appearance, shows that it is complicated. Thus in chorioiditis an retinitis pigmentosa, stellate anterior and posterior cortical cataracts ar found (see § 646); and if the cataract is total, it is often distinguished b being liquefied or calcified, by the thickening of the capsule, by the presenc of a yellow or green discoloration, by tremulousness of the lens, etc. A very important evidence of complication in the deeper parts is furnishe by the functional tests. If the projection is faulty or the perception o light is defective or absent, the percipient organs must be abnorma (page 182 and § 638).

It is of practical importance to recognize the fact that a cataract i complicated, because by this fact the prognosis and treatment are influ- enced. The prognosis is less favorable than in uncomplicated cataracts both because the operation is more difficult to perform, and because the result, as far as sight is concerned, is less successful. This last is particu-

larly the case if the projection and light perception are abnormal, in which case generally an operation should not be attempted. Moreover, complicated cataracts frequently require special methods of operation. In fact, many complicated cataracts can not be operated upon at all.

D. Treatment of Cataract

659. In incipient cataract the *refraction* should be examined at frequent intervals and the glasses changed accordingly (see § 649). Proper correction not only improves the sight but renders the muscæ volitantes less disturbing.

In cases of central opacity with clear periphery the sight may be temporarily improved by the instillation of *atropine*. A one-per-cent solution may be instilled just often enough to keep the pupil dilated and thus enable the patient to see through the clear area.

Various remedies have been used to *clear* a cataract in its earliest stages.

Successes have been reported with dietetic and hygienic treatment in diabetic and plethoric patients; with radium; and with potassium iodide in the form of collyria (one-per-cent). No convincing proof has been brought that these remedies are effective in any but a very small minority of the cases.

For most cases, at any rate, the only remedy for a cataract is by *operation*. The indispensable pre-requisite for this is that the light-perceiving parts (retina and optic nerve) should be healthy, a matter which is determined by careful functional testing (see § 638).

The methods of operation at our command are mainly discission and extraction. *Discission* is chiefly adapted for the cataracts of young people which do not yet contain a solid nucleus (§ 653). It can be performed in every stage of the growth of the cataract, and hence, too, in partial opacities of the lens. Moreover, discission is indicated in membranous cataracts, not to effect their absorption, which would be impossible, but to cut a hole in them (§ 964). The indications for *extraction* will be given at the same time with the description of the methods for performing it (§§ 961, 963, 964, 966, 967). Extraction gives its best results when the cataract is ripe. Hence we prefer to put off the operation until this occurs. But if for any reason it is specially desirable, e.g., if the other eye is becoming clouded so that the patient is incapacitated from work, the cataract may be extracted even before it is fully ripe. Healing then takes place with a good result as in ripe cataract, except that layers of transparent cortex are more apt to remain adherent to the capsule during the operation. These afterwards become opaque, so that a secondary cataract is formed which requires a secondary operation (discission).

Cataracts which are *congenital* or which develop in childhood should be operated upon as early as possible. Children can be subjected to the operation of discission with good results at the age of a few weeks. If the cataract is not operated upon, the development of the retina is arrested and amblyopia ex anopsia is produced (§ 241). Consequently, the good result of a cataract operation that is performed at a later date is comparatively small so far as vision is concerned.

In *traumatic* cataract our first thought should be to combat the inflam mation which usually follows the injury. Iced compresses are of the mos service against this impending inflammation, and also against the grea swelling of the wounded lens. Removal of the opaque lens should not b performed at once except when, owing to its own great swelling, it is itsel the cause of inflammation or of increase of tension. Otherwise, it is bette to put off the operation until later, lest the inflammatory symptoms b aggravated or brought on anew by it. If we wait a long time, often a grea part of the cataract is absorbed spontaneously, so that instead of extractio a simpler operation (discission) can be performed. So, too, in *complicate* cataract associated with inflammatory symptoms we had better await th decline of the inflammation before operating, unless imperative indication compel us to an earlier performance of the operation.

Sometimes we operate for cataract even in an eye which has no longer perceptio of light and hence cannot regain any sight—doing this merely for cosmetic reasons i order to give the pupil its natural black hue again.

It is better to remove a ripe cataract even if the other eye sees distinctly, for, i the first place, by so doing we enlarge his field of vision. In one-eyed people the field c vision is limited toward one side by the nose, while the man who sees with two eyes has field which stretches far towards both sides. In the second place, we have given th patient an eye which can at once take the place of the other in case the latter should be come unserviceable. If we delay operation until something happens to the other eye, w may perhaps be obliged to remove later on a very hypermature cataract under ur favorable conditions.

While, however, it is proper to remove a cataract in one eye when the other sees wel we must warn the patient that in general the vision in the eye operated on will be ver different from that of the other and usually quite blurred and that it will be difficult c impossible for him to correct this eye with glasses. For, on account of the considerabl loss in refractive power produced by removal of the lens (§ 193), the aphakic eye ha regularly a very different refraction from the other and requires a much stronger glas and in adults, at least, such an unequal correction is usually not well borne.

III. Changes of Position of the Lens

660. Anatomical Cause.—Changes of position of the lens always hav their anatomical cause in changes of the zonula of Zinn. This in the norma eye is tightly stretched, and holds the lens so firmly that the latter remain perfectly immovable even with the most violent motions of the heac Hence, any tremor of the lens, and still more any displacement of it from it natural position, presuppose a relaxation of the firmness of this attachment Such a relaxation can take place either from a simple elongation and loosen ing of the fibres of the zonula, or else from their rupture or complete des truction. Changes of this sort may affect either single portions or the entir circumference of the zonule.

661. Objective Signs. The objective symptoms of a change of positio differ according as the lens is only displaced a little (subluxation), or ha left its place in the fossa patellaris altogether (luxation).

(*a*) *Subluxation* may consist in the lens's being a little tilted, so tha one edge of it looks somewhat forward, the opposite one somewhat back ward. This is recognized from the *unequal depth of the anterior chambe* Another sort of subluxation is produced by lateral displacement of the lens

so that it no longer lies in the centre of the fossa patellaris. In this case, too, the anterior chamber is unequally deep. If, for example, the lens is somewhat depressed, the anterior chamber would be found to be deeper in its upper half, shallower below (Fig. 297). Furthermore, when the pupil is dilated (and, if the displacement is marked, without this) we can see the edge of the lens. This, in the example above selected for depression of the lens, would run transversely across the pupil, forming an arch which is convex upward. That part of the pupil which is situated above it, and which has no lens (Fig. 297, *a*) would be a deep black, while the lower part (*l*) which contains the lens would be faintly gray. This arises from the fact that even the most transparent lens reflects some light.

In both cases—that is, when the lens is tilted and when it is laterally

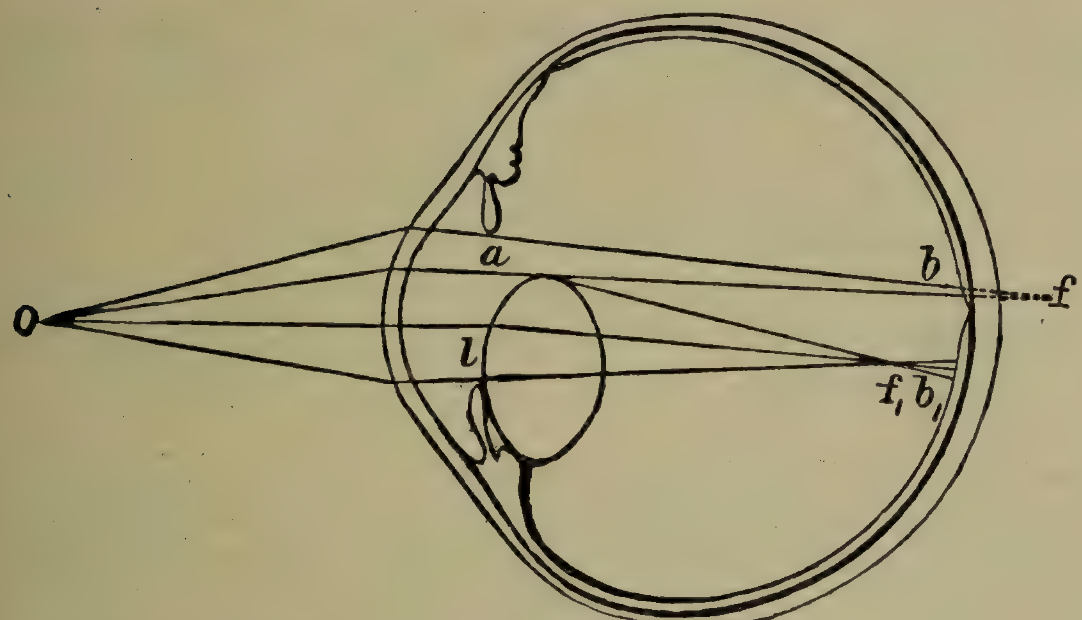

FIG. 297.—SUBLUXATION OF THE LENS. SCHEMATIC.

The lens has sunk so far downward that its upper edge is visible in the pupil. In consequence of the relaxation of the zonula, the lens bulges strongly, and is in contact by its lower border with the ciliary processes; moreover, the lower half of the iris is pressed forward by it. Above, on the contrary, the anterior chamber, owing to recession of the iris, is abnormally deep. Of the beam of rays emitted by the luminous point *O*, a portion goes through the aphakic part, *a*, of the pupil; these rays, on account of the absence of the lens, are insufficiently refracted, so that they come to a focus behind the retina at *f*, and form upon the retina a diffusion circle, *b*. That portion of the beam passing through the section, *l*, of the pupil, which contains the lens, undergoes excessive refraction on account of the increased convexity of the lens, so that the rays intersect in front of the retina at f_1, and form upon the retina a diffusion circle, b_1. This latter gets to lie below the fovea centralis (and below the diffusion circle, *b*), because all rays passing through the lens undergo a deviation downward on account of the prismatic action of the latter. Thus two images of the point *O* are produced upon the retina. The monocular diplopia thus caused is not, therefore, the result of the two sorts of refraction present in the area of the pupil, but is the result of the prismatic action of the margin of the lens.

displaced—conditions which are often combined—there occurs in movements of the eye tremor of the inadequately attached lens and with the lens of the iris also (*iridodonesis*).

By *reflected light*, a transparent, luxated lens appears faintly gray, and its edge has a golden lustre, almost as if it were a self-luminous body. This is so because the rays of light that come from in front and enter the marginal portions of the lens undergo total reflection at the posterior surface of the latter; for at this spot they pass from a denser medium (the lens) into a rarer medium (the vitreous), and hence are refracted away from the normal of incidence; but as, in consequence of this, they fall very obliquely upon the posterior lenticular surface at the edge of the lens, they undergo total reflection. They accordingly do not continue their course into the interior of the eye, but return to the observer, who therefore sees the edge of the lens shine. By *transmitted light*—in examining with the ophthalmoscope—the edge of the lens for the same reason appears black, because

the light that is reflected from the fundus, wherever it traverses the lens near its edge, so greatly deflected by the strong prismatic action of the latter toward the opposite si of the lens, that it fails to reach the eye of the observer, in case he is stationed straig in front of the eye. Hence, the border of the lens appears unillumined. But if the obse ver's eye is made to pass slowly toward the side of the lens opposite the unillumined edg a point is finally reached where the rays pass that are transmitted through this edge; ar then the latter appears of a shining red, while the rest of the lens appears unillumin (Dimmer).

In making an examination with the inverted image, we may often, in case of di location of the lens, see some portion of the fundus—e.g., the papilla—double, and for t same reason that the affected eye itself sees external objects double.

(b) *Total luxation* of the lens consists in its leaving the fossa pate laris altogether, either by prolapsing into the anterior chamber or by r ceding into the vitreous.

A lens luxated into the *anterior chamber* is readily recognizable from i shape. This is more convex than usual because the lens is no longer ke flat by the tense zonula. It therefore assumes its maximum convexity, it does upon the strongest effort of accommodation. If the lens is tran parent, its edges appear like a curved line of golden lustre, so that it loo as if a great drop of oil were lying in the anterior chamber. The anteri chamber is deeper, especially below, where the iris is pressed backward b the lens.

A lens prolapsed into the anterior chamber produces through irritation of the iris spasm of the sphincter iridis. The pupil consequently contracts, so that the return of t lens into the posterior chamber is cut off. It may even happen that on account of th spasm the lens is held tight at the moment when it is endeavoring to make its way throug the pupil into the anterior chamber. The lens is then jammed in the pupil, and cons quently violent symptoms of irritation are at once set up. But there are also cases which the lens can slip through the pupil so easily that it is found sometimes in front o sometimes behind, the iris. Sometimes the patient is able to produce this change place voluntarily. He can bring the lens into the anterior chamber by bending his hea forward and shaking it, while to bring the lens back again behind the iris he has to lie upo his back. In this case, of course, we are always dealing with lenses of diminished diam eter, which can pass through the pupil without difficulty. In some instances suc movable lenses are still attached to the zonula, which is then greatly elongated. If i such a case we were obliged to extract the lens, we would first bring it into the anteri chamber by the appropriate manœuvre. Then if we cause the pupil to contra behind the lens by employing a miotic we imprison the lens in the anteri chamber, and will be able under ordinary circumstances to remove it with ease. Howeve these cases in which the lens shows such a great capacity for making excursions belong t the rare exceptions. The rule is, that a lens luxated into the anterior chamber stay there, and, in consequence of the violent inflammation which it excites, becomes attache by exudates to the cornea and iris.

Luxation of the lens into the *vitreous* occurs more frequently than lux-ation into the anterior chamber. The anterior chamber then is deep becaus of the recession of the iris, which is tremulous. The pupil is of a pure black. The lens, itself, if opaque, may sometimes be recognized deep down, even with the naked eye; in most cases, however, the ophthalmoscope is required in order to discover it. It is either attached to some spot of the fundus by means of exudates, or it floats about freely in the vitreous (*cataracta natans*).

662. Subjective Symptoms.—Every dislocation of the lens entails a considerable *disturbance of vision*. If the lens still lies *within the pupillary area*, the eye becomes very myopic, because owing to the relaxation of the zonula the lens assumes its maximum convexity. The convexity and hence also the myopia are particularly marked in congenital dislocation. Added to the myopia in dislocation is a considerable degree of astigmatism, arising from the fact that the lens, either from being tilted or being laterally displaced or (in cases of partial rupture of the zonula) from being unequally relaxed, refracts the light with unequal strength in the different meridians (longitudinal and more or less regular astigmatism); or the refractive power may even vary in different sections of the same meridian (latitudinal, irregular astigmatism).

The astigmatism attains its maximum when the lens is so greatly displaced that its edge is visible in the pupil; the latter thus consisting of a portion which does, and one of

Fig. 298.—Luxation of the Lens into the Anterior Chamber. Magnified 5 × 1.

The lens lies in the anterior chamber and moreover is displaced toward the right side (in the drawing), so that here, on the one hand, it abuts against the posterior surface of the cornea, and, on the other hand, against the ciliary body, the iris being interposed between it and the latter. The pupillary portion of the iris everywhere is jammed against the posterior surface of the lens and thus the posterior is cut off from the anterior chamber. On the other side of the lens the iris is pushed forward and bent at an angle by the aqueous which has accumulated in the posterior chamber.

which does not, contain the lens. In such a case, moreover, double vision—*monocular diplopia*—is present; for the marginal portions of the lens act like a prism, whose refracting edge corresponds to the equator of the lens. By reason of this the rays passing through the lens are deviated, so that two images (b and b_1, Fig. 297) of one object (O) are formed upon the retina. Neither of these is distinct. The image (b) produced by the aphakic portion of the pupil corresponds to that formed by a very hypermetropic eye, and would require a convex lens to make it distinct. The image (b_1) appertaining to that part of the pupil which contains the lens is that of a myopic eye, and could be made distinct by means of a concave lens. Furthermore, disturbance of vision may be produced by the development of opacity in the subluxated lens.

In luxation of the lens *into the vitreous* the eye acts like an aphakic one, and, if no further complications are present, sees well with the correcting convex glasses. Indeed, in the old method of cataract operation by depression, a luxation of the lens into the vitreous was produced designedly in order to increase the sight.

663. Sequelæ.—Dislocations of the lens usually entail secondary consequences which may be extremely disastrous to the eye. Subluxations often in time change into complete luxations, the vibrating lens constantly pulling upon the zonula and gradually causing it to atrophy. While subluxated lenses often remain transparent for a long time, luxated lenses usu-

ally soon become opaque. Moreover, dislocated lenses are often opaque to start with, this being particularly the case in spontaneous luxations. The worst complications are irido-cyclitis and increase of tension (secondary glaucoma). The most dangerous form of luxation of the lens is that into the anterior chamber. In this case the cornea becomes opaque wherever the lens is applied to its posterior surface, and the eye, for the most part, undergoes speedy destruction through irido-cyclitis or increase of tension. On the other hand, luxation of the lens into the vitreous is the form best tolerated, especially if the lens, as time goes on, becomes smaller through resorption. In fact, in the depression of cataract, one used to count upon this tolerance of the eye toward the lens when depressed into the vitreous.

664. Etiology.—Dislocations of the lens may be congenital or acquired.

(*a*) *Congenital* dislocations consist in a lateral displacement (subluxation) of the lens, which is known as *ectopia lentis* (Fig. 299). The displacement is caused by the fact that the zonula is of unequal width in different directions. Most frequently the lens is displaced upward, the fibres of the zonula being shortest above, longest below. For the most part, too, the volume of the lens is somewhat smaller. In after years the ectopia usually increases, and even passes over into a condition of total luxation. Ectopia of the lens is ordinarily present in both eyes and symmetrically in both. Very often it is of hereditary origin.

FIG. 299.—ECTOPIA PUPILLÆ ET LENTIS. RIGHT EYE.
The pupil is pretty wide (5 mm.), not regularly circular and is displaced temporally. The broader nasal portion of the iris displays the contraction furrows. The lens is slightly cloudy, smaller than normal, and displaced in a direction opposite to that of the pupil, that is, nasally.

(*b*) The *acquired* dislocations of the lens develop either as the result of trauma or spontaneously. *Traumatic* dislocations are principally caused by contusion of the eyeball (for the mechanism of the luxation, see § 725). Every variety of subluxation and luxation may be thus produced, according as the zonula is simply torn into or is entirely torn through. If the tunics of the eye are ruptured, the lens may even be expelled entirely from the eye. Among traumatic luxations in the more extended sense of the word may be reckoned those which develop when perforation of a corneal ulcer takes place very rapidly; in this case if the perforation is large enough, the lens may even be discharged through it externally. *Spontaneous* dislocations take their origin from a gradual softening and disintegration of the zonula. The lens then owing to its weight sinks gradually deeper and deeper, and at length undergoes complete prolapse into the vitreous. The atrophy of the zonula develops as a result of liquefaction of the vitreous, and hence occurs especially in myopia of high degree, in chorioiditis, and in detachment of the retina. Again, the shrinking of a hypermature cataract may cause stretching of the zonula with consequent atrophy of it, and thus give rise to spontaneous dislocation of the lens, so that the sight which has been abrogated by the cataract is restored without an operation. If for any reason the zonula is already atrophic, the final impulse leading to total luxation is frequently afforded by a very insignificant traumatism—in fact, even by bending over, sneezing, etc.

Spontaneous dislocation of the lens not infrequently occurs in *ectasiæ* either of the eyeball as a whole or of its anterior segments—hence in buphthalmus, in staphylomata of the cornea, and in anterior staphylomata of the sclera. The luxation takes place because, as a result of the bulging out of the wall of the eyeball, the space between the edge of the lens and the ciliary body becomes enlarged, so that the zonula is stretched and finally atrophies. It may even happen that the lens has become adherent to a corneal cicatrix, so as to become more and more tilted as the cicatrix expands. So, too, the lens is sometimes drawn out of its place by exudates in the vitreous, which attach themselves to its posterior surface and afterward shrink; and it may be dislocated by tumors (gliomata and sarcomata) pressing upon it (Fig. 325).

665. Treatment.—*Treatment* in uncomplicated dislocation of the lens consists in the prescribing of suitable glasses.

In total dislocation the refractive state is that of aphakia (§ 193) and is treated accordingly. In subluxation the myopia and regular astigmatism can be corrected by glasses but the irregular astigmatism can not be. If the dislocation of the lens is so great that a part of the pupil is aphakic, we have the choice of correcting either the aphakic portion of the pupil with a convex glass or the portion of the pupil that contains the lens with a concave glass. We choose in this case the correction which gives the better sight. Sometimes for the sake of better correction, it is indicated to enlarge the aphakic portion of the pupil by an iridectomy, and so make the eye like one destitute of a lens.

If symptoms of irido-cyclitis or of secondary glaucoma are caused by displacement of the lens, extraction of the latter, if feasible, is indicated. Extraction is most readily performed in luxation of the lens into the anterior chamber; in this case, too, it is absolutely required, since otherwise the eye is lost. For extraction of a lens floating in the vitreous see § 975. In subluxation, the removal of the lens is often difficult or even miscarries altogether, because prolapse of the vitreous occurs on account of the defective structure of the zonula. Discission of a subluxated lens may be tried, but it is not often successful, because the lens, being imperfectly attached, gives way before the discission needle. In cases in which the removal of the lens is difficult or impossible, all we can do is to combat the inflammation or the increase in tension by means of an iridectomy. If an eye which is already blind is the seat of inflammation and pain due to luxation of the lens, enucleation is the best means of relieving the pain and averting the danger of sympathetic affection of the other eye.

Congenital and Other Anomalies of the Lens

666. Congenital anomalies of the lens include congenital *cataract* (§ 654) and *dislocation* (ectopia lentis—§ 664). Furthermore, whether ectopic or not, the lens may be smaller as a whole (*microphakia*), in extremely rare cases may be entirely absent (congenital *aphakia*), or may show a partial defect (*coloboma lentis*), which may take the form of a notching, a flattening of the edge, or an elongation and compression of the lower end of the lens. Coloboma of the lens is often associated with the coloboma of the chorioid or iris. *Coloboma of the zonula* is a congenital, usually triangular defect in the zonule of Zinn.

In *lenticonus* the lens presents a conical protrusion on its anterior or posterior surface. *Anterior* lenticonus is extremely rare and still rarer is *lentiglobus* (Dybus-Jaborski) in which the anterior protrusion is spherical instead of conical. *Posterior* lenticonus is more frequent. In both forms of lenticonus, skiascopy shows that the area occupied by the protrusion is highly myopic, the difference in refraction between it and the surrounding

peripheral zone being sometimes as much as 15 or 16 D. As evidence of the abrupt change of curvature in the lenticular surface the anterior lenticular image (Fig. 176, *b*), in the case of anterior lenticonus, and the posterior lenticular image (Fig. 176, *c*), in the case of posterior lenticonus, suddenly elongates or doubles (cf. § 612). When brought to the margin of the myopic area this abrupt change in the lenticular reflex distinguishes true from *false* lenticonus (central lenticular myopia, bifocal crystalline lens), in which there is a like highly myopic central area but no change in the curvature of the lens surface and hence no change in the reflex (Dimmer). Since posterior lenticonus is seated at the posterior pole of the lens, the myopic area, just as in the case of an opacity situated there, coincides with the corneal reflex and shifts with the latter as the eye is moved (§ 398). The apex of the protrusion often shows a slight opacity. Hess regards the more marked forms of posterior lenticonus as due to fetal rupture of the lens capsule.

In *pseudophakia fibrosa* (Czermak) the lens is replaced by a very vascular mass of connective tissue which is adherent to the iris and derives its blood-vessels from the latter. This very rare condition evidently is attributable to a hyperplasia of the tunica vasculosa. Such a hyperplasia may also be found when the lens is present, forming a dense deposit on the posterior surface of the latter (Lane). In life these hyperplasias look like a pseudo-glioma (Salzmann).

IV. Diseases of the Vitreous

667. Opacities.—These are sometimes small and sharply circumscribed, sometimes of large size. The smaller opacities are apparent to the patient himself as black specks of various shapes (muscæ volitantes) floating before the eyes (*myodesopsia*).[4] Such small, isolated opacities may not affect the visual acuity at all; larger or more diffuse opacities reduce the vision more or less considerably, and in very dense opacities the sight may be absolutely abolished.

When the opacities are numerous, the patients often say that their sight shows great variations within short periods of time. This fact is also noticed when tests of vision are made. Thus at first the patient may not make out the larger letters on the test card and yet later after looking fixedly for a time may distinguish even the small letters. Then all at once he sees much worse again. This comes from the mobility of the opacities, which, when the gaze is kept steadily fixed sink to the bottom of the vitreous, so that the central portion of the latter becomes clear; then any great movement of the eye stirs them up again.

To a certain extent vitreous opacities exist in every eye (*physiological* opacities).

The embryonic vitreous contains a great abundance of cells, and is hence opaque. The cells afterward disappear, but opaque remnants of them remain in the vitreous, and may be perceived entoptically as muscæ volitantes. These physiological opacities of the vitreous appear under the form of transparent filaments or of strings of pearls or of small flocculi, which move not only with the eye, but also spontaneously. We see this very readily if we look suddenly upward and then hold the eye still, when the opacities sink slowly down. They are thus distinguished from the entoptic images which are produced by opacities in the lens, as these always remain in the same place in the field of view. Physiological muscæ volitantes are not at all obvious, so that most men are not aware of their existence. To perceive them we look at a uniformly illuminated surface—for instance, the sky—through a stenopæic aperture. They are usually better perceived by myopic eyes. As soon as such muscæ volitantes become so distinct as to continually force themselves upon the attention and to become troublesome to the patient, they excite the suspicion of their being pathological. They then become visible with the ophthalmoscope, which is not ordinarily the case with physiological opacities.

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[4] From μυῖα, a fly, and ὄψις, vision; hence properly written myiodesopsia.

Pathological opacities are due to the exudates or hæmorrhages deposited in the vitreous and derived from the uvea or retina. A large part of the impairment of sight in diseases of the uvea and retina is due to opacities of this kind. Grave inflammations of these organs form large plastic or purulent exudates, sometimes filling the vitreous. The exudates produced by less severe inflammations appear as floating opacities which may be dust-like and diffuse or larger and isolated.

Hæmorrhages in the vitreous occur after injuries, and also, spontaneously in chorioiditis, retinitis, and myopia of high degree, in diseases like diabetes associated with altered composition of the blood, and in old people with atheromatous vessels.

Recurrent Vitreous Hæmorrhages—In young men, less often in young women, hæmorrhages in the vitreous, often with simultaneous hæmorrhages in the retina or sometimes with epistaxis, may appear spontaneously and recur repeatedly—permeating the vitreous so completely that even perception of light is lost. The most common cause of this affection seems to be tuberculosis, although an important primary or contributing factor apparently is gastro-intestinal disturbance with constipation. Syphilis, oxaluria, disorders of the blood and circulation, and menstrual disturbances have been held responsible for some cases. There is often marked tortuosity or periphlebitis of the retinal veins; in one case tuberculous disease of the sheaths of the retinal vessels was found on autopsy. If the hæmorrhages recur often, the vitreous never clears up perfectly, but masses of connective tissue ultimately form in it which may vascularize, and detachment of the retina may occur. The sight is thus seriously and permanently affected or even absolutely annihilated (cf. Retinitis Proliferans, § 784 and Fig. 355.)

668. Character of Vitreous Opacities; Prognosis and Treatment.—The *appearances* presented by vitreous opacities vary according to their nature and amount.

Opacities in the vitreous are made out with the *ophthalmoscope*. When the opacities are faint, it is necessary to use a plane mirror and place behind it a strong lens in order to enable us to get as near as possible to the eye we are examining. Generally, too, artificial dilatation of the pupil is required. Opacities of the vitreous are often best seen by the direct method, convex glasses of different strength being successively used so as to bring into view the opacities at different depths. From the strength of the glass that makes an opacity distinct, an idea can be formed of the distance of the opacity in front of the retina. Seen with the ophthalmoscope, opacities of the vitreous appear like dark dots or filaments or membranes floating about in the vitreous. Very minute opacities afford the picture of an extremely fine stippling of the vitreous ("vitreous dust"). If the opacities are still more minute, they can no longer be perceived as discrete points; nothing but a uniform obscuration of the fundus is observed (diffuse opacity of the vitreous). Such an opacity is observed in different diseases of the fundus (syphilitic chorio-retinitis etc.), and a peculiar form, whitish and occupying the posterior layers of the vitreous was observed during the war in cases of injury by foreign bodies (Salzmann). The more numerous the opacities are, the more hazy the fundus appears, the pupil at the same time looking redder than usual (as any bright background—e. g., the rising sun on a cloudy morning—appears red behind a cloudy medium). With very dense opacities, nothing is got with the ophthalmoscope but a feeble red reflection from the pupil, or the latter may even be perfectly dark. Massive plastic or purulent exudates appear by oblique illumination as gray or yellowish masses situated behind the lens.

Blood extravasated into the vitreous is found there under the form of flocculi or larger masses. When observed with the ophthalmoscope these either look simply black or show a faint reddish gleam. If they occupy the anterior section of the vitreous, they may even be recognized with lateral illumination (provided the pupil is widely enough

dilated) through the dark-red reflex emitted from the depths of the eye. In some cases of vitreous hæmorrhage a red streak of blood is seen on the posterior surface of the lens, usually parallel with the equator of the latter (Dimmer). In rare instances, especially in traumatic hæmorrhage the coloring matter of blood exuded in the vitreous dissolves, making the whole eyeground appear bright red. The aqueous, too, in such cases is colored red so that the iris looks as though seen through a ruby glass. Unabsorbed hæmorrhage may also cause siderosis bulbi (§ 858).

In *synchysis scintillans* particles are seen that look like golden spangles floating about in the vitreous, and which fall like a shower of gold to the bottom of the eye when the eye is held still. These are formed of crystals whose surfaces being smooth reflect the light strongly. They usually consist of cholesterin, sometimes also of tyrosin, margarin, and phosphates. These crystals are found in connection with various diseases of the eye but sometimes also in eyes that are otherwise healthy (especially in elderly people) without causing any essential disturbance of vision.

The *prognosis* depends upon the size and the age of the opacities. Recent opacities may be absorbed, so that the vitreous becomes perfectly clear again. Old opacities, on the contrary, usually resist all treatment. As regards hæmorrhages, the smaller ones may be completely absorbed, but large-sized ones nearly always leave considerable and permanent opacities. In any case hæmorrhages take a long time to absorb.

The larger exudates and hæmorrhages, if not absorbed become organized into membranes, cords, or even pretty large masses of connective tissue. New blood-vessels also may be formed which run from the retinal vessels into the vitreous, and can be made out there by means of the ophthalmoscope. The massive plastic exudates produced by the graver types of inflammation become organized, shrink up, and thus lead to atrophy of the eyeball, while the purulent exudates for the most part are transformed into a panophthalmitis, i. e., are discharged externally after perforating the sclera, and terminate in phthisis bulbi.

The *treatment* which generally is successful only in recent cases, consists in the employment of absorbent remedies, e.g., potassium iodide or other remedies containing iodine, especially syrup of hydriodic acid (De Schweinitz), mercury, diaphoretics, and mild saline purgatives. Repeated paracentesis of the anterior chamber may also be of service by stimulating tissue metamorphosis; and subconjunctival injections of a 5-to 10-per-cent salt solution or of mercury oxycyanide act in the same way. Good results have been had from aspiration, a hypodermic needle being introduced into the vitreous and a half or whole syringeful of turbid fluid being withdrawn (Zur Nedden).

In a recent case of vitreous hæmorrhage, rest in bed, kept up for several days is very important, and, if the hæmorrhage is due to injury, iced applications should be added. Dionin is also sometimes helpful. If the patient is anæmic, iron should be given. Constipation should be relieved, the diet regulated, and any remote source of infection sought for and removed. In arteriosclerosis the blood pressure should be reduced when this can be done safely. The iodides and other absorbent remedies mentioned above should be used and kept up for a long time. If this is done, sometimes old and even massive hæmorrhages may be absorbed. In persistent bleeding the subcutaneous injection of a 2-per-cent solution of gelatin or of defibrinated

human blood may be tried (Leber), or calcium lactate (0.30 gm. three times a day) may be administered (Wilmer).

669. Liquefaction of the Vitreous (**Synchysis**[5] **Corporis Vitrei**).—When observing opacities of the vitreous with the ophthalmoscope, we see that most of them float about freely in the vitreous. It follows from this that the framework of the vitreous must have been destroyed, so that this body itself is converted into a perfectly liquid mass. In operations we often have an opportunity of directly convincing ourselves of the liquefaction of the vitreous, which we see flowing out under the form of a viscid, usually yellow-colored liquid. Liquefaction of the vitreous occurs as a simple senile change, but when of greater extent it is the result of disease of the adjacent membranes, which are concerned in maintaining the nutrition of the vitreous—that is, it occurs chiefly in disease of the ciliary body, also in retinitis, chorioiditis, myopia of high degree, ectatic eyes, etc.

The most important consequence of liquefaction of the vitreous consists in the gradual diminution in volume, which the altered vitreous may undergo and which manifests itself in a diminished tension of the eye. In such cases, detachment of the retina and afterwards even atrophy of the eyeball may supervene. Another consequence of the liquefaction of the vitreous is that the zonula becomes softened and atrophic. This produces tremulousness of the lens, and later on even its spontaneous dislocation.

670. Wounds and Foreign Bodies.—These and their consequences will be considered under the head of injuries of the eye (Chap. XXX).

Wounds opening the vitreous chamber are often associated with *prolapse of the vitreous.* If the vitreous is fluid it escapes as a sticky liquid. The healing of the wound is not affected, and the fluid is soon replaced. It is otherwise if the vitreous is of normal consistence. Then it does not escape but lies between the lips of the wound, causing gaping of the latter and delayed healing and often affording an avenue to the entrance of infecting germs. If the protruded bead is excised more protrudes. As the wound heals the protrusion is gradually pinched off and converted into a whitish filamentous mass. The vitreous within the eyeball remains adherent to the scar (*anterior vitreous synechia*). The vitreous framework, if destroyed, is not replaced (Salzmann).

Hernia of the vitreous is a sac-like prolapse into the anterior chamber occurring sometimes in aphakia or subluxation of the lens.

Among *foreign bodies* may be reckoned lenses luxated into the vitreous and *entozoa.* The latter include the filaria, the ecchinococcus, and the cysticercus cellulosæ. All these occur but rarely, although the cysticercus, which is the scolex of the ordinary tape worm (Tænia solium), was formerly frequent. The eggs of the tænia get into the stomach either with the food or drinking water or, it may be from the intestine, (in case the patient himself harbors a tapeworm). In the stomach, there develop from the eggs embryos, having hooklets, by means of which they penetrate the stomach walls and get into the blood vessels. The blood current then carries them into different parts of the body, where they again leave the vessels, bore into the tissues, and there grow into cysticerci. In the eye the cysticercus most often gets into the vessels of the chorioid and when it leaves there passes at once beneath the retina, detaching it from the chorioid (see Fig. 360). When it has reached a certain size it perforates the retina and gets into the vitreous. But the cysticercus may also find its way into a vessel of the retina or the ciliary body, and from there may enter the vitreous directly, without a preceding detachment of the retina. In the vitreous the cysticercus becomes visible as a bluish-white bladder. If the head and neck are drawn in, they appear under the form of a brilliant white spot; but if they are

[5] From σύν, together, and χέω, I pour.

protruded they can be recognized quite distinctly, and it is even possible to make out in the head the suctorial discs and the crown of hooklets. The animal makes spontaneous, often very active, movements. It is rare, however, for the cysticercus to be seen with perfect distinctness when in the vitreous. For membranous opacities very soon form which so envelop it that nothing can be discerned through them but a dense white mass. In such cases the diagnosis of a cysticercus is difficult, and can indeed be made with certainty only when upon long and attentive observation we make out spontaneous movements taking place in the white structure. Ultimately the eye undergoes destruction from irido-cyclitis. Hence a cysticercus when discovered in the vitreous must be removed (§ 978).

671. Detachment of the Vitreous.—This consists in the accumulation of liquid, either in front of the vitreous, between it and the lens, or behind the vitreous between it and the retina. (In the region of the ciliary body the vitreous is so firmly adherent that it never becomes detached). According to Lister, it is not infrequent and if occurring when partial attachment of the vitreous to the retina has been produced by injuries, hæmorrhage, or inflammation it may cause folding and detachment of the retina and tears in the retina. Sometimes also a wrinkling of the hyaloid membrane or an actual *hole* in the latter is produced.

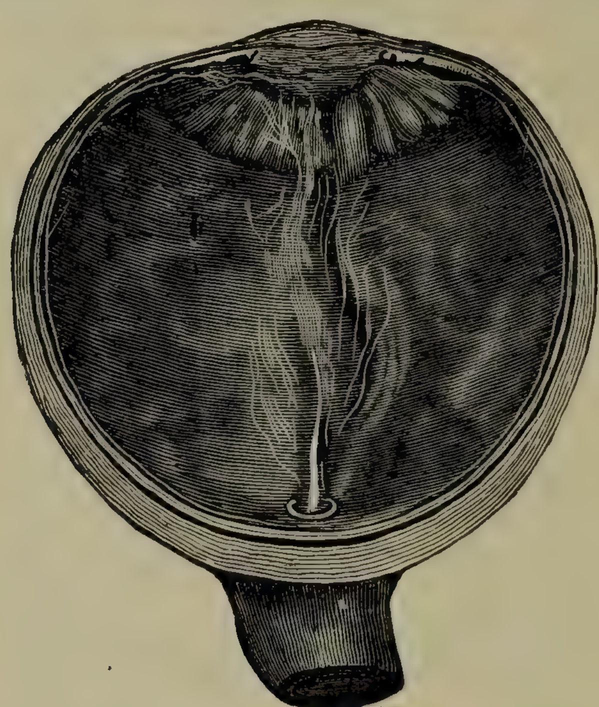


FIG. 300.—PERSISTENT HYALOID ARTERY (Lang and Collins). From Norris and Oliver. Shrunken globe in which a tag of a persistent hyaloid artery was found adherent to the optic nerve on pathological examination.

672. Congenital Anomalies.—The main congenital anomaly of the vitreous is the *persistent hyaloid artery. Coloboma of the vitreous* is a notch in the lower part of the vitreous, sometimes extending all the way from the optic disc to the ciliary body. It is caused by the projection of vascular connective-tissue strands into the interior of the eye.

Of the hyaloid artery normally the only thing that is left in the newborn infant is a short and slender cord, which, moreover, disappears during the first year of life. Exceptionally, however, larger remnants of the artery remain for life. The artery then ordinarily appears under the form of a gray filament that stretches from the papilla out into the vitreous, and may even reach to the posterior pole of the lens. In typical cases it is possible to demonstrate the connection between the filament and the central vessels that emerge at the papilla, and in this demonstration is found the surest means of distinguishing between this remnant of fetal life and pathological opacities of the vitreous, which may in other respects have a similar shape and position. Sometimes instead of a filament a wider tubular structure is observed, extending out from behind forward in the axis of the vitreous. This represents the *hyaloid canal* (also called *Cloquet's canal*), whose walls, owing to some abnormality in their structure, are visible with the ophthalmoscope. This congenital anomaly, as well as the persistent hyaloid artery, is frequently associated with opacities in the posterior portions of the lens (posterior polar and cortical cataract). In many animals—e.g., in the frog and in many snakes and fishes—the vessels of the vitreous persist during life.

CHAPTER XXV

DISEASES OF THE IRIS AND CILIARY BODY

I. Inflammation

673. Iritis, Cyclitis, Irido-cyclitis.—The iris and the ciliary body form a continuous whole, inasmuch as the iris springs from the ciliary body; both, moreover, are supplied by the same blood-vessels. It is hence quite easy to understand that both organs are very frequently diseased at the same time. Unmixed inflammation of the iris (iritis) or of the ciliary body (cyclitis) is rare; in most cases we have to do with a combination of the two (irido-cyclitis). For practical reasons, however, it is advisable to describe the symptoms of iritis and of cyclitis separately, and then show what sort of clinical picture is produced by their combination.

674. Symptoms of Iritis.—The symptoms of iritis are partly referable to the hyperæmia of the iris, partly to the formation of exudation.

Hyperæmia of the iris is usually the first manifestation of iritis. It may occur also in cases in which no iritis develops, i.e., there is no exudation. In that case the hyperæmia disappears without leaving a trace of its presence. Simple hyperæmia is due to the same causes as iritis itself, whenever the irritation they set up is not great enough to provoke actual inflammation. The most frequent cause is some affection, particularly small ulcers or foreign bodies, of the cornea.

Hyperæmia manifests itself chiefly by the discoloration which causes a blue or gray iris to appear greenish—a change which is particularly striking when comparison is made with the iris of the other eye, in case this is healthy. In dark eyes the discoloration is less pronounced. Sometimes, with the aid of a magnifying glass, we can clearly distinguish the separate dilated blood-vessels under the form of red striæ or maculæ. Furthermore, the pupil is contracted, and does not react as well as usual. The contraction is a necessary result of the distention of the iris due to the increased fullness of the vessels; besides, there is a spasm of the sphincter produced by the irritation. For these reasons the reaction of the iris to light is diminished, and atropine also acts less promptly and less thoroughly than usual. Hyperæmia of the iris, whether simple or inflammatory, is accompanied by conjunctival and ciliary injection, photophobia, and increased secretion of tears.

675. *Exudation* takes place partly into the tissue of the iris itself, partly into the anterior and posterior chambers, and is accordingly characterized by varying symptoms:

(1) *Exudation into the tissue of the iris* makes the latter, since it is filled with an abundance of round cells, appear swollen and thicker than usual. The *discoloration* is still more pronounced than in simple hyperæmia, and the clear-cut markings upon the anterior surface of the iris are obscured.

It is easy to understand that the rigid and swollen iris should react bu insufficiently to light; the pupil is greatly contracted.

676. (2) *Exudation into the anterior chamber* manifests itself first b *turbidity of the aqueous,* in which numerous exudation cells are suspendec The turbidity is best recognized upon the dark background of the pupi which in this case looks gray instead of being a pure black. Gradually th formed constituents floating in the aqueous sink to the bottom of th chamber, where they produce a *hypo pyon* (Fig. 245, A and B). When ther is very great hyperæmia, rupture of th blood-vessels in the iris may take plac with an extravasation of blood, whicl also sinks to the bottom of the anterio chamber (hyphæma).

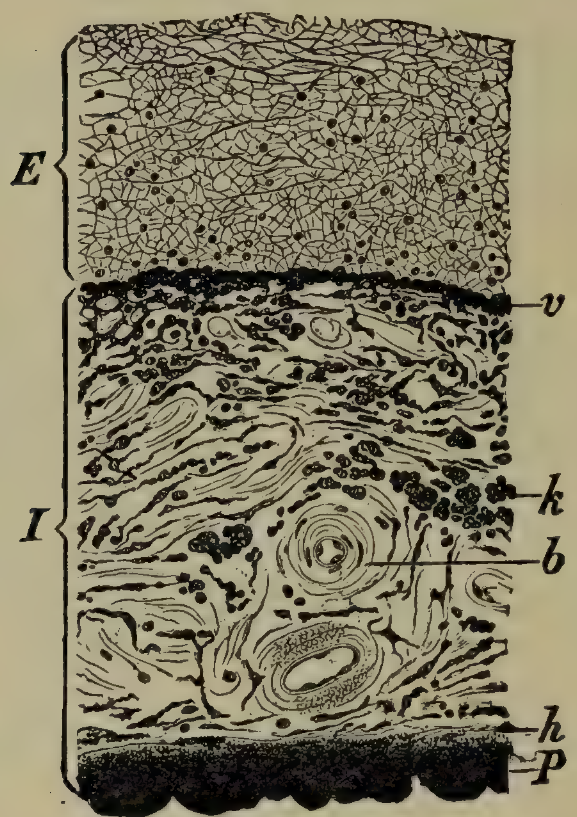


Fig. 301.—Iritis. Magnified 116 × 1.

Upon the surface of the iris is a layer of exudate, *E*, consisting mainly of a fine network of coagulated fibrin, in which lie scattered pus corpuscles. The iris, *I*, is bounded in front by the anterior limiting layer, *v*, behind by the posterior limiting layer, *h*, and the retinal pigment layer, *P*. In the tissue of the iris are recognized the cross sections of blood-vessels, *b*, with very developed adventitia, and a great quantity of pigmented cells (the specimen is taken from a dark-brown iris). Most of these (chromatophores) still retain their elongated and branching shape, but others, at *k*, are transformed into shapeless agglomerations of pigment, as is usually the case in inflammation of the iris. In the anterior layers of the iris many small cells (emigrated leucocytes)—likewise an evidence of inflammation—lie between the pigment cells.

Even when there is no hyphæma th presence of blood in the anterior chamber ma be shown by the reddish or yellowish hue c the white reflex seen when the angle of th chamber is examined with the ophthalmos cope by Salzmann's method (§ 390).

Besides the exudates suspende in the aqueous, a layer of exudate i also found covering the walls of th anterior chamber. Owing to the exu date deposited on its posterior sur face (Fig. 313, *f*) the cornea appear faintly and uniformly clouded. More over, the layer of exudate deposite upon the iris (Fig. 301) contributes es sentially to the hazy appearance of th iris markings. The layer of exudat extends from the iris upon the anterio capsule of the lens (Fig. 302, *e*), an covers the latter throughout the area o the pupil, which consequently appear gray.

If this exudate becomes organized a membrane is produced which closes the pupil and is connected with th pupillary margin of the iris (Figs. 303 and 304, *e*). This is called a pupill ary membrane, and the condition thus brought about is called *occlusi pupillæ* (Fig. 308). It is evident that this condition must result in a ver considerable impairment of vision.

A peculiar appearance is furnished by the presence of a pretty large *fibrinou* exudate in the anterior chamber, such as is at times observed in any sort of acute iriti If much fibrin is deposited in the aqueous, it may become coagulated into a uniforml gray, translucent mass (hence the name gelatinous exudate and also lenticular exudate because with its rounded edges it sometimes looks like a half-transparent lens that ha

been dislocated into the anterior chamber). The exudate speedily shrinks, since the fibrin in contracting expels the liquid from its meshes (for which reason we also speak of it as a spongy exudate and of the iritis as a *spongy iritis*). After a few days the exudate either completely disappears or is reduced to a thin pellicle lying in the pupil and often still connected with the pupillary margin by one or two slender filaments.

677. (3) A special form of exudate that is found particularly in the chronic cases, are the *precipitates* (deposits) upon the posterior surface of the cornea. These are small dots, no bigger than a pin's head, of a light

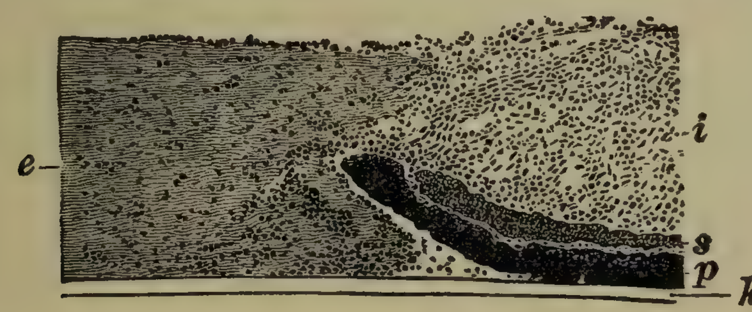


FIG. 302.—RECENT IRITIS WITH PUPILLARY MEMBRANE. Magnified 55 × 1.

The iris, *i*, is greatly thickened by swelling and infiltrated by numerous round cells. Attached to the pupillary margin is an exudate, *e*, which fills the whole pupil, and which, on the one hand, pushes its way somewhat beneath the pigment layer, *p*, and, on the other hand, extends up on the anterior surface of the iris. That it is a quite recent exudate is evident from its thickness and from its being composed of a network of fibrin, inclosing scattered pus corpuscles. The number of the latter increases toward the surface of the iris. *k*, capsule of the lens.

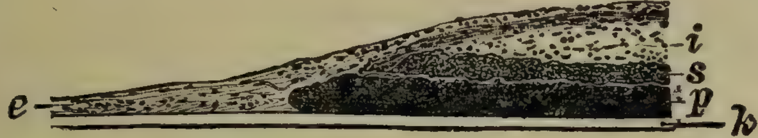


FIG. 303.—OCCLUSIO PUPILLÆ TWO MONTHS AFTER A PERFORATING INJURY. Magnified 55 × 1.

The exudate, *e*, has become converted into connective tissue, and has, in consequence, shriveled down to a thin pellicle, which, becoming constantly more and more attenuated, can be traced over the entire surface of the iris. The iris itself has become thinner from atrophy, and where it ends at the pupillary margin tapers off owing to its being pulled upon by the pupillary membrane. The pigment layer, *p*, is the part most drawn out into the pupil, so that it projects a good bit beyond the sphincter, *s*, and the stroma of the iris, *i*. Hence the margin of the pupil in the living eye, when looked at from in front, seems as though encompassed by a broad brown rim, which appears to attach the edge of the pupil to the capsule of the lens.

FIG. 304.—OCCLUSIO PUPILLÆ THREE MONTHS AFTER A PERFORATING INJURY. Magnified 55 × 1.

The exudate, *e*, is converted into a thin membrane of connective tissue, which, at the pupillary border of the iris, extends mainly beneath the latter, and can be traced as a delicate pellicle for a long distance between the pigment layer, *p*, and the lens capsule, *k*. The tension set up by the pupillary membrane upon the border of the atrophic iris, *i*, has drawn the latter down over the pigment layer, so that the sphincter iridis, *s*, which is solidly united with the pigment layer, has its anterior border turned back in a hook-shaped bend. In the living eye the margin of the pupil appeared encompassed by a gray rim which merged gradually into the less gray, because more transparent pupillary membrane.

gray or brownish color, which lie upon the posterior corneal surface (*p*, Fig. 308). If the deposits are large, they are generally few in number and scattered irregularly over the cornea (Fig. 305; if they are small they are proportionately more numerous and occupy the lower half of the cornea, in which they cover a surface having the shape of a triangle. The base of the triangle corresponds to the lower corneal margin, and its apex is directed upward toward the centre of the cornea. It is frequently observed that the deposits diminish in size from the base toward the apex of the triangle (Fig. 306).

42

The peculiar arrangement of the deposits is readily explained by their mode o origin. The deposits are conglomerations of cells, agglutinated into masses by means o fibrin (Fig. 313). At first they are suspended in the aqueous, and by the movements o the eye are thrown by virtue of centrifugal force against the posterior surface of the corne and adhere to it. In so doing they arrange themselves according to weight, the larges being lowest down. The triangular shape of the arrangement is a result of the move ments of the eyeball, by which the deposits are cast upon the cornea. We have only t think of what happens when we throw sand through a wire screen or shake grain in sieve. The little fragments of stone or of grain always form a pointed figure with th apex, which contains the finest particles, running upward, while successively coarse particles follow in order below. The same is the case with the deposits. By thei peculiar arrangement the deposits are generally easily distinguishable from macula opacities in the cornea itself (in keratitis punctata, see §§ 571 and 575). Other distinguish ing marks are the clearer outline and frequently the brownish color of the deposits, which moreover, do not lie at different depths like maculæ in the cornea itself, but lie all in th same plane—that is, on the posterior surface of the cornea.

The deposits are easilv overlooked, because they are often so extremely *minute*. W

Fig. 305.

Fig. 306.

Fig. 305.—Precipitates. Besides minute deposits, there are found also large ones, which ar light gray and lardaceous looking.

Fig. 306.—Precipitates. These are small and disposed in the form of a triangle.

should, therefore in every case in which there is a suspicion of the existence of an affec tion of the uveal tract, look for them with a strong magnifying glass. Sometimes the can be made out well by using the direct method and putting up a 16 to 20 D convex len in the ophthalmoscope. In slight cases of chronic cyclitis (see § 683) a couple of ver minute precipitates are often the only objective sign of the disease, and if we overloo them we are often unable to account for the patient's subjective troubles, for the sensi tiveness of the eye to light, for the readiness with which it tires, or for the occasiona slight pain or moderate reddening of the eye. It is important also to make the diag nosis in another class of cases, quite frequent, in which cyclitis causes increase of tensio and glaucomatous symptoms (the appearance of colored rings about a light, pain, dila tation of the pupil etc.). If in such a case the precipitates are overlooked the patien may be subjected to an iridectomy in the belief that the condition is one of primary glau coma, when he might have been relieved without the performance of this operation.

Besides the ordinary precipitates above mentioned it is possible to make out wit the *corneal microscope* and slit lamp extremely minute deposits forming a faint haze on th posterior surface of the cornea (Koeppe).

In contradistinction to the scarcely visible deposits, spoken of above, there are also found specially *large* gray lardaceous-looking deposits which as the disease progresses change their shape, enlarge, and coalesce with neighboring precipitates to form biscui or trefoil-shaped masses of exudation (Fig. 305). Large deposits of this sort occur pre-eminently in the tuberculous or scrofulous inflammation of the uvea.

On careful inspection with the loupe we sometimes find deposits in the pupillary area and particularly *on the iris*. It is true, they are hard to see, in this situation, especially on a light-colored iris with which they do not contrast well. The deposits probably get on the iris at night, when the patient lies for a long time quietly on his back, so that the agglomerations of exudate floating in the aqueous are not thrown against the cornea by the movements of the eye, but sink gradually down upon the iris.

Most deposits get *darker*, as they get older. For, the cells of the exudate break down and become absorbed, so that the pigment granules, which lie between them and which are left behind, come more and more clearly into view. Ultimately there remain dark brown or black dots, which may readily be confounded with small black foreign bodies, if no heed is paid to their deep situation. In rare cases the deposits are dark brown, even when recent, if at the outset they have carried much pigment with them from the uvea.

For *lens deposits*, see § 657.

678. (4) Exudation poured out into the *posterior chamber* is not accessible to direct observation, but manifests itself only by the adhesions which it causes between the iris and the capsule of the lens (*posterior synechiæ*). These adhesions develop principally at the spot where the iris and the capsule of the lens are in contact—i.e., at the pupillary margin. They form at the time when the iritis is at its height, and when, therefore, the pupil is greatly contracted. When, after the iritis has run its course, the pupil tends to resume its usual mean width, this is only possible over the area in which the pupillary margin has remained unattached. The portions that are adherent to the capsule of the lens cannot retract, but remain as tags of greater or less size, projecting in toward the centre of the pupil. The pupil thus acquires an irregular shape which is still more obvious if atropine is instilled; for, as the iris then retracts strongly at its unattached portions, the synechiæ stand out in the clearest possible way (*a* and *b*, Fig. 307). Atropine is hence a very valuable agent for the diagnosis of posterior synechiæ.

In the formation of posterior synechiæ, it is not the stroma of the iris, but the layer of the retinal pigment (Figs. 302, 303, and 304, *p*) covering its posterior surface, that becomes adherent to the capsule of the lens. When the iris starts to retract, the pigment layer is held back at the points of adhesion, and is thus exposed to view more extensively than usual. Hence in dilatation of the pupil, especially by atropine, the tags jutting out into the pupil look brown (Fig. 303). From the traction exerted by the iris, rupture of the synechiæ may result. This sometimes is effected spontaneously by the traction which is constantly being made upon the adhesions during the ceaseless movements of the iris; for the most part, however, rupture is produced artificially by the instillation of atropine. In that case we see, at the spot where the synechia has been set free, a brown spot remaining upon the anterior capsule of the lens. This is the pigment layer whose pathological adhesion to the capsule of the lens is firmer than its physiological connection with the tissue of the iris. If several synechiæ have been ruptured, we find remains of them in the shape of a corresponding number of brown dots arranged in a circle upon the anterior capsule of the lens (Fig. 307, between and on either side of *a* and *b*). This circle is narrower than the mean diameter of the pupil, because the synechiæ were formed at the time when the pupil was contracted by iritis. The dots of pigment never disappear, and hence during the whole life give evidence of an iritis that has once existed.

It happens sometimes that posterior synechiæ develop in the presence of a dilated, instead of contracted, pupil; as, for example, when they form in an eye under the influence of atropine. In this case the margin of the pupil becomes adherent to the capsule of the lens at some peripherally located spot, so that afterward, when the pupil assumes its mean width, the synechia is marked, not by a projecting process, but by a re-entrant angle.

Posterior synechiæ may sometimes be formed *without inflammation of the iris*, e.g., when the latter comes into contact with a wound of the lens capsule or with swelling lens matter (Salzmann).

679. If the adhesion of the iris to the capsule of the lens is not confined to single points, but comprises the whole extent of the pupillary margin, we speak of an *annular posterior synechia.* No projecting tags are then apparent, because the iris is no longer able to retract at any spot, the pupil remaining unchanged all the time, even after the instillation of atropine. There is generally a brown rim (pigment) or a gray rim (exudate) investing the pupillary margin (Figs. 303 and 304). An annular synechia is seldom formed all at once; it is for the most part the result of a number of recurring attacks of iritis, which little by little produce a more and more extensive adhesion of the iris to the lens. The direct consequence of an annular synechia is the shutting off of the anterior from the posterior chamber— shutting off of the pupil (*seclusio pupillæ*).

The two sequelæ of iritis, shutting off of the pupil (seclusio pupillæ) and shutting up of the pupil (occlusio pupillæ), very often occur together owing

Fig. 307.—Posterior Synechiæ and Remains of Fetal Pupillary Membrane. Magnified 2 × 1.
The pupil has been dilated by the instillation of atropine. The dilatation, however, is unequal because the upper part of the pupillary margin is fixed by means of adhesions to the anterior capsule of the lens. At *a* there is a slender synechia which is drawn out into a fine black point; at *b*, on the other hand, is found a broad and but slightly elongated adhesion, such as frequently occurs, especially in syphilitic iritis. Between the synechiæ and by the side of them, the capsule of the lens is covered with minute black dots arranged in a semicircle. They correspond to the situation of the pupillary margin when the pupil was contracted, and have been left by the rupture of the adhesions. From the lower part of the iris a filament, *c*, runs straight upward. This rises from the trabeculæ of the circulus iridis minor, and passes in the region of the pupil to the anterior capsule of the lens, where it is attached to a small, round, capsular opacity. This filament is not a posterior synechia, but a remnant of the fetal pupillary membrane. It does not prevent the iris from retracting properly under atropine, but is simply stretched and drawn out thin itself.

to the fact that the exudate which attaches the pupillary margin to the lens may also extend over the entire pupil (Fig. 308). But they may also occur separately and then have very different consequences. Occlusion of the pupil occurring by itself produces very great diminution of sight, without, however, entailing any dangers for the future. Seclusion of the pupil in itself does not affect the sight, if the pupil is free from membrane, but subsequently induces changes (increase of tension) which cause blinding of the eye.

For the diagnosis of *seclusio pupillæ*, the employment of atropine is often indispensable. It frequently happens that the pupillary margin is thought to be adherent to the capsule all round, when, nevertheless, upon the employment of atropine it retracts at one small spot. At this unattached portion, which is most frequently situated above, there is thus formed a curved or horseshoe-shaped indentation of the pupillary margin. Similarly we may infer the existence of a small opening if, after the case has been under observation for a long time, no protrusion of the iris takes place, since in true seclusio pupillæ such protrusion never fails to appear. Of course, we must be able to exclude the existence of a total posterior synechia, in which case evidently protrusion of the iris could not take place.

Seclusio pupillæ appears to occur frequently without there being any simultaneous formation of membrane in the pupil (occlusio pupillæ). This, however, is only apparent, as a rule. Upon careful examination we usually notice that the gray fringe of exudate which runs along the adherent pupillary margin projects far into the pupil, becoming gradually thinner as it does so, so that perhaps only the centre of the pupil appears to be quite free. Nay more, if, after performing an iridectomy, we compare the pupil with the pure black coloboma, we can almost always convince ourselves that really no part of the pupil is quite free from a membrane.

Much more frequently than seclusion without occlusion, the converse—i.e., occlusion without seclusion—occurs. That is, there is a membrane in the pupil, and sometimes quite a thick one, which is not connected with the pupillary margin all round, but only at separate spots.

The presence of a well-marked reaction of the pupil to light is not altogether conclusive evidence against the existence of seclusio pupillæ. If the tissue of the iris is not yet atrophic, and a fair perception of light at the same time exists, the anterior layers of the iris, when the illumination is varied, move quite perceptibly over the fixed posterior pigment layer.

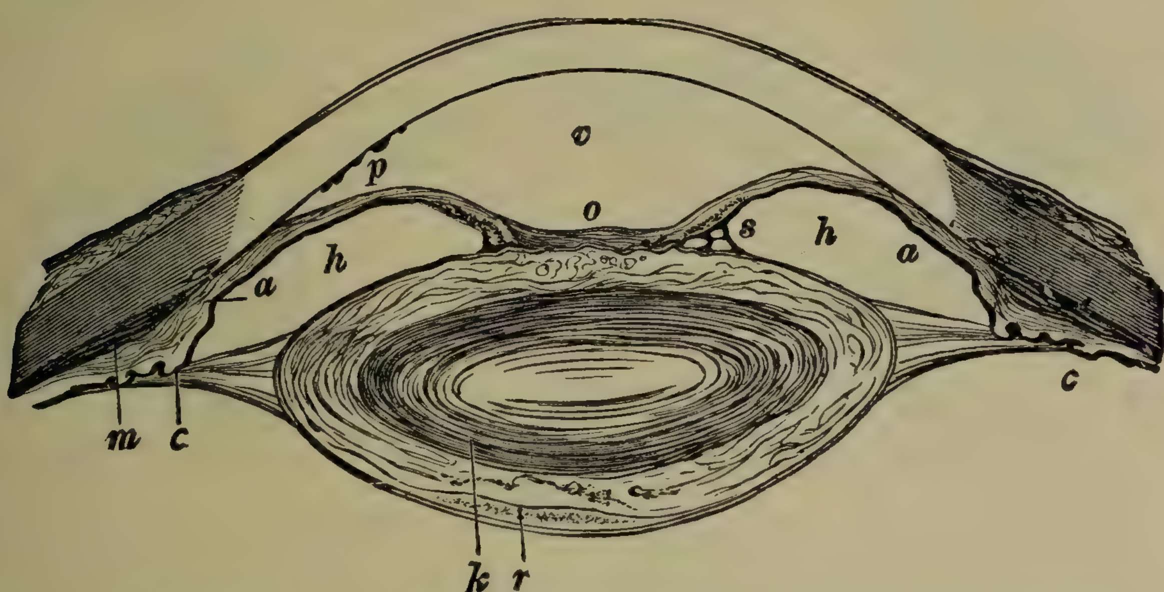


FIG. 308.—SECLUSION AND OCCLUSION OF THE PUPIL. Magnified 5 × 1.

The iris is adherent by its entire pupillary margin to the lens, but elsewhere is pushed forward. The posterior chamber, *h*, is thus made deeper, the anterior chamber, *v*, shallower, especially at the periphery where the root of the iris, *a*, is pressed against the cornea by the increase of tension. In consequence of the traction made upon the iris, its retinal pigment is beginning to separate (at *s*) and to be left upon the capsule of the lens. The pupil is closed by an exudate membrane, o, by the shrinking of which the anterior capsule is thrown into folds. In the lower part of the anterior chamber there is matter, *p*, precipitated upon the posterior surface of the cornea. In consequence of the increase in tension, both the ciliary processes, *c*, and the ciliary muscle, *m*, are atrophic and flattened. The cortex of the lens has undergone cataractous disintegration, and at *r* is separated from the capsule by liquor Morgagni; the nucleus, *k*, of the lens is unaltered.

680. Symptoms of Cyclitis.—Exudation from the ciliary body, apart from the infiltration of the tissue itself, takes place partly into the anterior chamber, but most of all into the posterior chamber, and the vitreous:

1. The exudate produced by the ciliary body may get into the *anterior chamber* directly, in case there is implication of the most anterior portion of the ciliary body—i.e., that covered by the ligamentum pectinatum and lying at the confines of the anterior chamber. Thus a hypopyon may be poured out by the ciliary body, and especially there may be produced the precipitates, which are so frequent in chronic cyclitis. Particularly characteristic of cyclitis are the gray or grayish-white exudates that develop in many cases and which appear to grow out in the form of spongy masses from various portions of the angle of the chamber, both below, like the precipitates, and also from other directions.

2. Exudation into *the posterior chamber* (Fig. 312), if excessive, cause adhesion of the whole posterior surface of the iris to the anterior capsul of the lens (*total posterior synechia*, Fig. 309). This adhesion of the surfac of the iris is distinguished from the annular synechia, in which only th pupillary border of the iris is attached to the capsule, chiefly from th altered form of the anterior chamber. The exudate, as it shrinks, draw the iris everywhere up to the anterior surface of the lens, so that the pos terior chamber is completely obliterated. The anterior chamber is henc proportionally deeper, above all at the periphery, where the iris is displace farthest backward (*b*, Fig. 309).

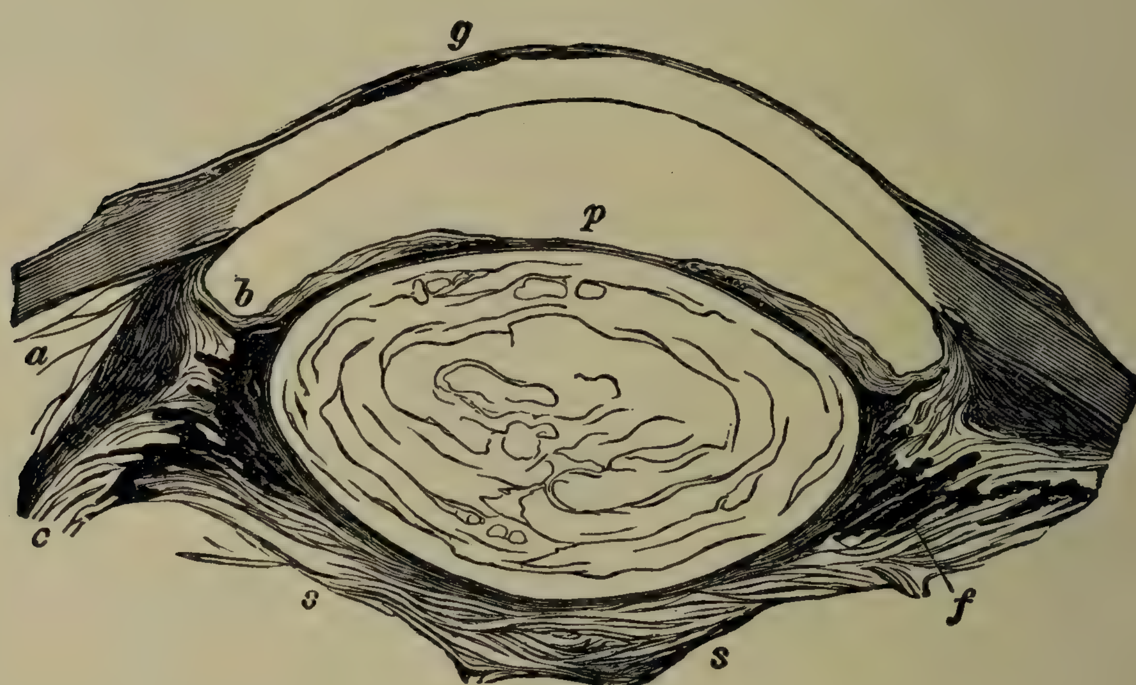


FIG. 309.—TOTAL POSTERIOR SYNECHIA. VERTICAL SECTION THROUGH THE EYE. Magnified 5 × 1.

The iris is adherent by its posterior surface to the capsule of the lens and also the anterior surface of the ciliary body. The posterior chamber consequently is obliterated and the anterior chamber deepened at its periphery, *b*; at this spot the iris is strongly retracted and at the same time is here the most thinned through atrophy. The exudate connecting the iris with the lens also stretches as a thin membrane, *p*, across the pupil. The hull of exudate, *s*, springing from the ciliary body, envelops the posterior surface of the lens and by its shrinking draws the ciliary processes toward the centre. As a result of this, a separation of the ciliary body, *c*, from its bed has already taken place below, and in the intermediate space are seen the disjoined lamellæ of the suprachorioid membrane, *a*. The pigment epithelium, *f*, of the ciliary processes has undergone proliferation. At the lower part of the cornea there is a zonular opacity, *g*. The lens is swollen and is opaque throughout; there is no hard, undisintegrated nucleus (i.e., it is a soft cataract).

3. Exudation *into the vitreous* appears under the form of opacities of the vitreous, which can be made out with the ophthalmoscope if the condition of the refracting media permits. Moreover, they manifest themselves by a corresponding diminution of the vision. In bad cases, an exudation of large size is present in the anterior division of the vitreous (*s*, Fig. 309), and under favorable circumstances can be seen with oblique illumination as a gray mass behind the lens. The sight is then almost completely abolished, and atrophy of the whole eye is subsequently produced by the shrinking of the mass of exudation.

The *tension* of the eye, which in iritis is usually unchanged, is often altered in cyclitis. It is not infrequently elevated in the beginning of the cyclitis; indeed, such a great elevation of tension may be developed that blindness is produced by it. In the later stages of cyclitis, on the contrary,

diminution of the intra-ocular pressure is more frequent, and is the result of the shrinking of the exudate while in process of organization.

Since the aqueous in irido-cyclitis is more albuminous, it filters with more difficulty through the ligamentum pectinatum, especially if the gaps in the latter are partially blocked by coagula or leucocytes. In this probably consists the cause of the frequently occurring increase of tension. In this case, in consequence of the retention of the aqueous, the anterior chamber is deeper than normal, while in true glaucoma it is shallower. The increase of tension in irido-cyclitis is rarely so lasting that the eye would be blinded by it without an operation; usually the increase of tension is but transient, although it may occur repeatedly in the course of the disease.

681. Subjective Symptoms of Iritis and Cyclitis.—Both iritis and cyclitis are associated with *symptoms of inflammatory irritation*, consisting of ciliary injection, photophobia, lacrimation, and pain. The pain is not only situated in the eye itself, but radiates to the parts in the vicinity, especially the supra-orbital region. The intensity of the irritative symptoms depends on the greater or less acuteness with which the case runs its course. Chronic cases occur in which inflammatory symptoms are wanting altogether, so that the eye is at no time reddened or painful; on the other hand, there are cases of irido-cyclitis in which the pain reaches an absolutely intolerable pitch and is associated with vomiting and a febrile movement. Sometimes (particularly in syphilitic iritis and irido-cyclitis) the pain sets in with special violence at night.

Vision is always diminished through turbidity of the aqueous or through exudation in the region of the pupil or in the cavity of the vitreous.

In recent acute iritis and irido-cyclitis examination of the sight discloses sometimes the existence of a moderate degree of *myopia*, which did not exist previous to the inflammation, and which, moreover, gradually disappears after the latter has run its course. This has been attributed to spasm of the accommodation due to irritation of the ciliary muscle and to increased refractivity of the aqueous due to exudation. Neither explanation is satisfactory. In testing the vision in iritis it is important to remember that myopia of this sort may be present and to see if the poor vision found can not be improved by a concave glass of moderate strength; otherwise, we may think that the sight is poorer than it really is.

In severe cases of irido-cyclitis (especially of sympathetic origin) *blanching of the cilia* on the lids of the affected eye is sometimes observed.

682. Diagnosis of Iritis.—Iritis, especially in the earlier stages, is often confounded with conjunctivitis and with glaucoma.

From *conjunctivitis* a hyperæmia or actual inflammation of the iris is distinguished by the discoloration of the iris (often made evident by comparison with the other eye) and the small size of the pupil which reacts but feebly to light and is dilated imperfectly by mydriatics. Moreover a slight turbidity of the aqueous can be recognized from the fact that the pupil is not as pure a black as in the other eye.

Confirmatory signs are the injection, which is predominately circumcorneal (anterior conjunctival and ciliary) with exemption of the retrotarsal fold, and the presence of actual pain.

In the earliest stages of iritis when there are simply hyperæmia and cramp of the iris, the diagnosis is sometimes difficult. Homatropine should then be used. (If there

is a suspicion of glaucoma, the homatropine may be replaced by euphthalmine o even cocaine. In that case if increase of tension occurs with the dilatation, this ca easily be controlled by eserine, and if, on the other hand, the euphthalmine or cocaine fail to act, they can at once be replaced by homatropine.) If one instillation of a 2-per-cen solution of homatropine combined with 4-per-cent cocaine fails to dilate the pupil ir twenty minutes, repeated instillations should be made until the pupil does dilate, or it is evident that it will not do so. In the latter case atropine should be instilled at once. I under either homatropine or atropine the pupil dilates sluggishly, imperfectly or unevenly and if, furthermore, when the dilatation is finally effected the pain and photophobia are relieved, the diagnosis of iritis is confirmed. Adrenaline helps, too, by dispelling a superficial injection and so bringing to view a deep injection which the drug does not much reduce.

Congestive *glaucoma* shares with iritis the symptoms of discoloration of the iris, turbidity of the aqueous, ciliary injection and pain, but is distinguished by the dilated, immobile pupil, the shallow anterior chamber, and the distinct increase in tension. The last, in particular, serves to distinguish a glaucoma from an iritis in which the pupil has been dilated by atropine.

683. Diagnosis of Cyclitis and Irido-Cyclitis.—That the ciliary body is pathologically altered in most cases of apparently simple iritis is put beyond a doubt by anatomical investigations. But, since we can not see the ciliary body directly, slight changes in it escape diagnosis. We hence make the diagnosis of *irido-cyclitis* only in those cases in which in addition to the symptoms of iritis, positive evidences of involvement of the ciliary body are also present. This is the case—

1. When the inflammatory symptoms reach a considerable pitch, and especially if œdema of the upper lid is associated with them—a thing which does not occur in simple iritis.
2. When the eyeball in the ciliary region is painful to the touch.
3. When from the presence of a pupillary membrane, of many posterior synechiæ, or of an annular or a total adhesion of the iris to the lens capsule, we can infer that the exudation is especially great.
4. When the disturbance of vision is more considerable than one would expect from the opacities within the confines of the anterior chamber. We are then justified in inferring the existence of opacities in the vitreous causing diminution of vision.
5. If the tension is altered—either elevated or lowered.

The participation of the ciliary body in the inflammation of the iris makes the disease a far more serious one and renders the prognosis worse. Not only is the inflammation more violent, but it induces changes which are much more difficult to remedy. The exudates, such as posterior synechiæ and pupillary membranes, produced by iritis in the confines of the anterior chamber, may be attacked successfully by operative procedures. But the exudates left by cyclitis in the vitreous, so far as they do not become absorbed spontaneously, are in no way susceptible of removal. Severe cyclitis leads to destruction of the eye (atrophy of the eyeball)—a thing that never occurs with iritis alone.

Simple cyclitis without iritis occurs only in the chronic form. The inflammatory symptoms are slight or absent, the iris is of normal appear-

ance, and the pupil is generally somewhat dilated. The chief symptoms are the presence of deposits upon the cornea and opacities in the vitreous.

Simple cyclitis with precipitates on the cornea is often called *serous iritis*. But this term should be rejected for the precipitates are not a serous exudation, and the conditions is not properly speaking an iritis but a cyclitis.

684. Course of Iritis and Irido-Cyclitis.—With regard to the course, a distinction is made between acute and chronic cases.

Acute iritis is associated with marked inflammatory symptoms, but runs a fairly rapid course. But even in the acute cases—if we except the very slightest ones—it takes four weeks or more before the inflammation entirely subsides. The first signs of a change for the better in the inflammation are the decrease of the injection and of the pain, and especially the prompt action of atropine, while at the acme of the inflammation the pupil is so spasmodically contracted that atropine has little or no effect.

In **chronic iritis** the products of exudation—the synechiæ, pupillary membrane, deposits, and opacity in the vitreous—develop and gradually increase, while the eye either shows no symptoms of irritation at all, or else there is now and again simply an insignificant injection and some sensitiveness to light. There is always a disturbance of sight, which, indeed, is the thing that attracts the patient's attention and takes him to the physician. Chronic iritis occurs in a light and in a grave form. The *light* form is characterized mainly by the precipitates, occurring either alone or in combination with opacities in the anterior portion of the vitreous. If at the same time no changes of any kind can be observed in the iris, the disease must be called a sluggish cyclitis (cf. § 683). Often the posterior portion of the uvea is likewise affected, and foci that can be made out with the ophthalmoscope appear in the chorioid. The more the inflammation affects the whole uvea, the more closely this form approximates to the second, severe form, into which it may pass and from which it is not separated by any sharp line of distinction.

The most serious complication of the ordinary cases is the *increase of tension*. We then find the anterior chamber deep (a sign that the increase of tension is caused by a hindrance to the escape of the aqueous) and the pupil somewhat dilated. Often the increase of tension abates spontaneously in a short time, but if it remains for a long time without being attended to, it may cause blinding of the eye. Apart from this, the disease usually runs a mild course. It often extends over a period of years, but finally disappears either without leaving any trace of its presence or else leaving a disturbance of sight due to vitreous opacities.

In the *grave* form of chronic iritis symptoms of irritation are absent just as they are in the light form, but, on the other hand, the exudation is much more extensive and affects all parts of the uvea, for which reason this form is called *chronic irido-chorioiditis* or better *chronic uveitis*, since the ciliary body, which is situated between the iris and the chorioid, is likewise involved. The patients come on account of the steadily increasing disturbance of vision. Examination of the eye shows posterior synechiæ

which slowly increase in numbers until finally seclusion of the pupil is developed. Almost always, too, there is a thin membrane present in the pupil. The iris becomes atrophic early, and afterward, when seclusion of the pupil develops, bulges forward in hump-shaped projections. Hypopyon is never present, but instead there are very frequently fine deposits which point to the implication of the ciliary body, an implication which also manifests itself through the presence of opacities in the vitreous. As these opacities continually increase in number and the vitreous at the same time becomes liquefied, the latter is finally converted into an opaque, mucilaginous liquid. Subsequently opacity of the lens is added, and atrophy of the chorioid and retina sets in. The interference with vision in these eyes is, therefore, always much greater than the optical obstructions in the anterior division of the eye (deposits, membrane in the pupil) would lead one to expect. This disease, which really affects all parts of the eyeball, in most cases terminates in complete blindness. In consequence of the seclusio pupillæ, increase of tension sets in with blindness from excavation of the optic nerve; afterward the eyeball may become ectatic. In other cases, blindness takes place under the guise of a gradually developing atrophy of the eyeball; the eye becomes softer, and the retina undergoes total detachment.

Chronic iritis (irido-cyclitis, irido-chorioiditis) almost always attacks both eyes. It runs a very chronic course, and the light form often passes gradually into the grave form, so that at the outset of the disease one can never say what its duration and outcome will be.

685. Recurrences of Iritis.—Inflammations of the iris and ciliary body often show a great tendency to relapse. The recurrences are often less severe than the primary inflammation; but as in many cases they are quite frequently repeated, and as they leave a new exudate after them each time, they quite commonly lead ultimately to serious changes, such as seclusio or occlusio pupillæ.

It was formerly supposed that the recurrences were due to the irritation caused by the traction of posterior synechiæ left by the first attack. It seems certain, however, that they are due not to the syncehiæ but to a continuance of the constitutional condition which caused the iritis in the first place.

686. Termination—In light cases the outcome of inflammation may be a perfect *cure.* The synechiæ rupture, leaving behind tags of pigment attached to the anterior capsule, which are unproductive of injury to the eye. The hypopyon disappears by resorption. The deposits generally persist for a long time (for months), until they also are removed by resorption. In many cases they leave at the spot where they were situated a permanent opacity of the cornea in the shape of a gray speck, or the pigment that is contained in them remains permanently as a black dot. The slighter vitreous opacities may all disappear completely by resorption.

In most cases, however, sequelæ such as are described below remain, causing greater or less *impairment of function* or even *destructive changes* in the eye, with total blindness.

687. Sequelæ—These are posterior synechiæ, pupillary membrane, exudates between the iris and lens, atrophy of the iris, exudates in the vitreous, changes in the cornea, and opacity of the lens.

(1) *Posterior Synechiæ.*—These are the most frequent of the sequelæ of iritis. They either form permanent attachments between the iris and lens, or rupture leaving spots of pigment on the latter. If only a few are present, they cause no special injury to the eye and also produce little or no impairment of sight. Very much worse is the annular posterior synechia or *seclusio pupillæ.* By this the communication between the anterior and the posterior chamber is obliterated. The aqueous secreted by the ciliary processes can no longer pass through the pupil into the anterior chamber; it hence collects in the posterior chamber, pressing the iris forward. A hump-shaped protrusion of the iris is thus produced, which finally reaches as far forward as the cornea, while the pupil is represented by a crater-shaped retraction of the pupillary margin which is attached to the lens capsule (so-called *iris bombée,* Figs. 308 and 437). In consequence of being so greatly stretched, the iris becomes atrophic. To this condition, elevation of the intra-ocular pressure (secondary glaucoma; see §§ 847, 848) is added. The increased hardness of the eye can be established by palpation; the anterior ciliary veins are dilated; the cornea is dull and less sensitive to touch; the sight fails, with a decrease in the field of vision starting from the nasal side, until at lenth the perception of light is entirely abolished. Then the formation of scleral ectasiæ takes place in the blinded eye in the shape of anterior and equatorial staphylomata of the sclera. Seclusio pupillæ, accordingly, if it is not remedied in time, regularly leads to blindness.

Sclerosis pupillæ is *not invariably associated with protrusion* of the iris. This is absent if the iris is totally adherent to the lens (total posterior synechia, 680) or if the secretion of aqueous is abolished, as happens sometimes when the ciliary body is involved in the inflammation and covered with exudate. In the latter case also there will be *no increase of tension.* It may even happen that the iris protrudes somewhat and yet, because the secretion of aqueous is checked, the tension fails to rise.

The *protrusion of the iris* in seclusio pupillæ does not take place uniformly, but with the formation of prominences separated by constrictions. The latter represent the more resistant, radial fibres which do not give way before the pressure of the aqueous until afterward. The condition in which the iris is found protruding in a greater part of its circumference, while one sector of it remains in its normal situation, is generally referable to the fact that at this spot an adhesion of the surface of the iris to the lens exists, preventing its protrusion (Figs. 337 and 338). It would, therefore, be a mistake to select just this spot for performing an iridectomy, although on other accounts it would seem to be very suitable for such a purpose, because of the greater depth of the chamber there. If the iris has been pushed forward as far as the cornea, it may become agglutinated to the latter in places, and in this way anterior synechiæ may be produced without there having ever been a perforation of the cornea (see § 609).

When in a case of seclusio pupillæ the iris is pushed forward as far as the cornea there is not, for all that, a complete *abolition of the anterior chamber,* the latter being still present in the region of the pupil. It is otherwise in those cases in which the seclusio pupillæ has been produced not by irido-cyclitis, but by inclusion of the whole pupillary margin in the orifice of a corneal perforation due to ulcer to injury. In this case the liquid which accumulates in the posterior chamber pushes the iris in its whole extent against the cornea so that all traces of the anterior chamber vanish. Afterwards the iris becomes agglutinated to the cornea and ultimately becomes so atrophic as a result of the

increase of tension that only its retinal pigment is left to cover the posterior surface of the cornea—a state of things which is particularly often met with in staphylomata of the cornea (see § 616 and Figs. 274-276). Finally complete abolition of the anterior chamber is produced in rare cases because the lens together with the iris is pressed against the cornea on account of a tremendous increase of tension in the vitreous cavity (glaucoma malignum, intra-ocular tumors). In all the cases above cited of abolition of the anterior chamber the intra-ocular pressure is increased. In this fact lies a diagnostic sign distinguishing them from other cases of absence of the anterior chamber, in which the intra-ocular pressure is diminished. To these latter cases belongs abolition of the chamber due to leakage of aqueous, either outside through a wound or fistula or into the suprachorioidal space in detachment of the chorioid (§ 760). Furthermore, the iris and lens may be pushed forward by a cyclitic membrane situated behind the latter, in which case, too, the eye is softer than normal.

688. (2) *Pupillary Membrane* (*Occlusio Pupillæ*).—This causes an interference with vision, the degree of which depends upon the thickness of the membrane.

689. (3) *Exudates Behind the Iris.*—These lie either between the iris and the lens (forming a flat or, when of greatest extent a *total posterior synechia*) or between the ciliary body and the lens and upon the posterior surface of the latter. In bad cases they form a coherent fibrous mass which completely envelops the lens, and which, on account of its solidity, is called the cyclitic hull (*s*, Fig. 309). This has a great tendency to shrink. In total posterior synechia there is evidently no hump-shaped protrusion of the iris—on the contrary, the anterior chamber in the beginning is usually deep at the periphery owing to the retraction of the iris (*b*, Fig. 309). Afterward, when atrophy of the eye sets in, the anterior chamber often becomes shallower again, because iris and lens together are pushed forward.

690. (4) *Atrophy of the Iris.*—This is characterized by a bleached-out, gray, or grayish-brown aspect of the iris (resembling gray felt or blotting paper); the delicate markings of the anterior surface have disappeared, and in their stead dilated vessels can often be recognized as reddish blotches upon the surface of the iris. The pupillary margin is thinned down, often looking as if it had been frayed out; the reaction of the iris is diminished or altogether lost. The great friability of the atrophic iris often renders the correct performance of iridectomy impossible.

The *causes* of atrophy of the iris are (1): Long continued or frequently recurring inflammation. (2) Increase of tension. Here the main agent is the compression of the blood-vessels at the root of the iris, which is pushed away from the ciliary body and against the sclera (Figs. 373 and 374). The atrophy often develops quickly—in attacks of inflammatory glaucoma, sometimes within a few days. (3) Iridodialysis, as the result of which the vessels running from the circulus arteriosus iridis major to the iris are ruptured. (4) Traction. This occurs when the iris is attached both at its ciliary and at its pupillary border, and the distance between these two points gradually increases. This is most frequently observed when the iris is incarcerated in a scar of the cornea, and subsequently this scar or the entire eyeball becomes ectatic. So, too, stretching of the iris occurs when the pupillary margin of the iris becomes attached to the capsule of the lens in childhood as the result of iritis, and later on, with the growth of the eye the distance between the ciliary and pupillary margins increases (Fig. 310). (5) Too thoroughgoing absorption; for example, when swelling fragments of the lens lie upon the iris and are gradually absorbed—a process, the result of which sometimes is that at

the spot where the fragments are situated a portion of the iris tissue also disappears. (6) Variola and xeroderma pigmentosum (see below). (7) Advanced age.

The *appearance* of the atrophic iris likewise varies, as follows: (1) After inflammation and increase of tension the iris is like gray blotting paper, since owing to the shrinking it has become flat and hence has lost its delicate relief, and since furthermore the pigment of the stroma in great part has been destroyed. For this reason, too, a brown iris by atrophy becomes gray. (2) As a result of a circumscribed effacement of the stroma pigment, light colored or even white spots are formed in the iris (in glaucoma and after absorption of lens fragments). In the cases associated with variola the iris has numerous little white spots on its anterior surface—*vitiligo iridis* (Müller, Fuchs). In xeroderma pigmentosum there are occasionally found spots of decoloration and atrophy of the uveal portion of the iris together with freckle-like pigment spots (Salzmann). (3) Black spots form, when not only the stroma pigment but also the stroma itself has disappeared, so that the retinal pigment layer lies exposed. (4) If the latter also is destroyed, complete gaps are formed in the iris. We can then throw light through these

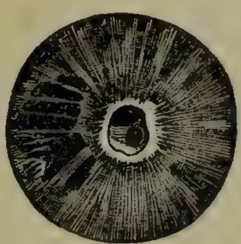


Fig. 310.—Gaps in the Iris. Magnified 2 × 1.

Left eye of a girl who as the result of hereditary lues had become affected in the first year of her life with a bilateral irido-cyclitis with seclusio and occlusio pupillæ. Up to the age of six the girl was almost blind; then owing to the spontaneous formation of gaps in the iris of both eyes the sight improved to such an extent that she was able to go to school. At the age of twenty-two she suffered from a bilateral parenchymatous keratitis. In the membrane which closes the pupil are visible two thin spots appearing as black gaps. On the nasal side the stroma of the iris has disappeared to such an extent that there are left of it only a few gray ridges corresponding to the larger vessels, which run from the ciliary border to the region of the sphincter of the iris. Between these ridges the iris looks black either because the retinal pigment is exposed to view or else because it is absent altogether. In the places where the pigment is absent we can look right into the interior of the eye with the ophthalmoscope. The rest of the iris is to a moderate extent atrophic and its radiating fibres are tensely stretched.

with the ophthalmoscope, and the patient, too, can see through them, thus regaining vision when previously he was blind because of closure of the pupil proper (Fig. 310). If the atrophy affects the root of the iris, which is a specially thin part even when the iris is normal, a spontaneous iridodialysis is produced (§ 723). One of the first evidences of disintegration of the pigment epithelium may be the liberation of the pigment which then coats the anterior surface of the iris or the structures at the angle of the chamber or becomes mixed with the precipitates (§ 696 and Fig. 313). This pigment deposit has been regarded as a source of glaucoma (Koeppe), but while occurring often in this condition, it occurrs in others as well. A peculiar degenerative change affecting the pigment epithelium is the dropsical swelling of the latter found in diabetes. (5) Diminution in size of the iris always occurs in conjunction with atrophy, when the pupillary margin is not so attached to the capsule that such a diminution in size would be prevented. In glaucoma the iris may become so small as to disappear either in part or altogether, because it is concealed behind the limbus. (6) When the pupillary margin is free, the shrinking iris stroma usually draws the retinal pigment layer gradually over upon the anterior surface. The brown rim lining the pupillary margin hence becomes progressively broader (*ectropion of the pigment layer*, Fig. 374, *e*).

The dilated vessels that are frequently visible in the atrophic iris have quite often a course that does not in any way correspond with the regular radial arrangement of the normal vessels of the iris. As a matter of fact, vessels of this sort, as anatomical examination shows, do not lie in the iris itself, but in a thin exudation membrane deposited upon it. Often they extend over the pupillary border into the pupil, i.e., into the pupillary membrane.

691. (5) *Plastic Exudates in Vitreous.*—Exudates in the vitreous (*c*, Fig. 311) which are capable of organization are called plastic exudates.

When they are of small amount it is possible for them to be absorbed entirel except for a few membranes left behind in the vitreous. Usually, howeve they become organized and in so doing shrink, thus causing diminution i volume of the vitreous. The first result of this is softening of the eyebal the next a detachment of the retina (*r*, Fig. 311) from the chorioid; in pa also this detachment is produced by direct traction, since the exudates a they shrink attach themselves to the inner surface of the retina and dra it out of its bed. In consequence of the detachment of the retina, complet blindness ensues. The eyeball itself gradually grows smaller and misshape This state of things, consisting of diminished tension of the eyeball, wit decrease in its size and with complete blindness, is known as *atrophy c the eyeball.*

In traumatic or postoperative irido-cyclitis an early sign of beginning atrophy is th indrawing of the scar in the cornea or sclera—this also being due to the traction exerte by the shrinking intra-ocular exudate. As atrophy proceeds the whole eye becomes c slightly quadrangular shape. This is because the four recti muscles, stretching across th equator of the eyeball, press the sclera in somewhat at this spot, and hence produc flattening of the four sides. With higher degrees of atrophy quite deep furrows a formed, so that the eyeball resembles a bale of goods grooved by the cord with which it tied. Other furrows, too, develop in the sclera, particularly one running around the ey behind the attachments of the recti muscles (Fig. 311, *S*). The cornea is smaller, ofte opaque and flattened; at other times, again, transparent, but abnormally protuberan or wrinkled. The atrophic iris is either pressed right against the posterior surface of th cornea, or an anterior chamber still exists. In the latter case, the chamber is bounde behind by a firm diaphragm in which the iris, which is imbedded in the mass of exudat is often but indistinctly recognizable. If the pupil is still distinguishable, a mem brane and the opaque lens are found in it. The eye is often sensitive to the touc In the later stages markedly hard spots (ossified exudates) may sometimes be fe through the sclera.

Atrophy develops gradually during a course of months and year The inflammation and the pain, which have been present for a long time disappear when the atrophy is complete. But even then secondary attack of pain occur, especially if the eye harbors a foreign body, or if ossificatio of the exudate takes place.

692. (6) *Suppuration in the Vitreous.*—In severe cases the exudate i the vitreous consists of pus and is incapable of organization. The pus eithe remains confined to the vitreous, forming an incapsulated mass (*abscess o the vitreous*) which afterward shrinks so as to produce atrophy of the eye ball; or it erodes and perforates the sclera (*panophthalmitis*) producin then *phthisis bulbi* (see for further §§ 754–758).

693. (7) *Opacity of the Cornea.*—The cornea in marked irido-cyclitis i always somewhat dull; moreover, it may suffer from deeply situated infil trates (keratitis pustuliformis profunda, see § 563), from the deposition on i of exudate (deposits, hypopyon), or from the contact of the iris, which whe lasting a long time produces corneal opacity (§ 588). In the stage o atrophy of the eyeball a zonular opacity frequently forms upon the cornea Keratitis bullosa and vesiculosa also often make their appearance in eye which are blinded by irido-cyclitis (§ 597).

694. (8) *Opacity of the Lens.*—This develops in consequence of the disturbed nutrition of the lens. If merely a few synechiæ are present, it is rarely observed; on the other hand, it is observed with proportionately great regularity when seclusio pupillæ has existed for a long time, and particularly so in the bad cases in which the lens is complete swathed in cyclitic exudates. Such a cataract is denoted as cataracta complicata or cataracta accreta (=grown fast to—i.e., grown fast to the iris). In atrophic eyeballs the lens is always opaque, and generally shrunken as well.

695. Morbid Anatomy.—Inflammation of the uvea is produced by ectogenous or endogenous infection (see §§ 97–100).

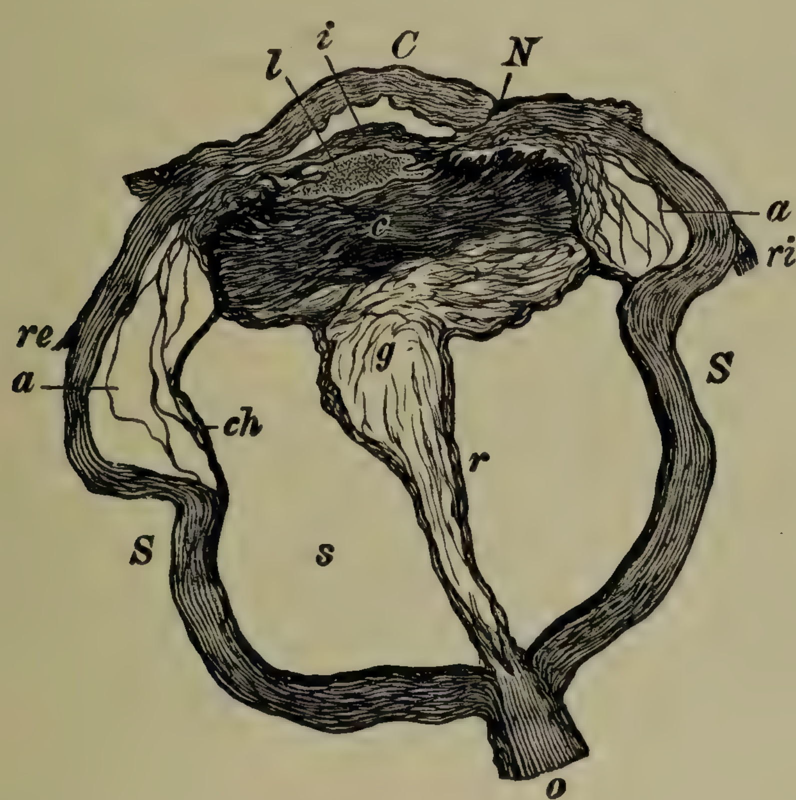


FIG. 311.—ATROPHY OF THE EYEBALL. (In part after Wedl-Bock.)

The eye is smaller and of irregular shape, chiefly from the grooving of the sclera, *S*, behind the points of attachment of the ocular muscles, the rectus internus, *ri*, and the rectus externus, *re*. The cornea, *C*, is diminished in size, flattened, and wrinkled, especially on its posterior surface. At its inner border it bears the depressed cicatrix, *N*, which was produced by the injury. The anterior chamber is shallow; the iris, *i*, is thickened and forms an unbroken surface, because the pupil is closed by exudate. Behind the iris lies the shrunken lens, *l*, and behind this is the great hull of cyclitic membrane, *c* the shrinking of which is the cause of the atrophy of the eyeball. By reason of this shrinking, the ciliary processes, the pigment layer of which has markedly proliferated, are drawn in toward the centre and, together with the adjacent chorioid, *ch*, are detached from the sclera; between the two structures are seen the disjoined lamellæ of the suprachorioid membrane, *a*. The retina, *r*, is detached and folded up into a funnel, which incloses the remains of the degenerated vitreous, *g*. The subretinal space, *s*, is filled with a fluid rich in albumin. The optic nerve, o, is thinner than normal and is atrophic.

(1) Ectogenous Infection.—Our best information with regard to the anatomical changes here is derived from the cases of infection produced by a perforating injury, (§ 98). The clinical and anatomical picture varies according as the inflammation begins in the anterior or posterior division of the eye, that is, the aqueous chamber or the cavity of the vitreous. The exudation thrown out into these cavities may be either plastic (capable of organization) or purulent. The description here given will apply mainly to the plastic form of irido-cyclitis; the purulent inflammations of the posterior segment of the uvea (purulent irido-chorioiditis) will be considered under diseases of the chorioid (§§ 752 et seq.).

696. (a) **Changes in Anterior Chamber.**—When an infection has taken place in the *region of the aqueous chamber*, either from the introduction of the bacteria into the aqueous

or into the tissue of the iris and ciliary body, the inflammation breaks out first in these two structures.

The iris is thickened, its blood-vessels distended. In its tissue lie uninuclear and multinuclear leucocytes in great abundance, and also a few red blood corpuscles; in particularly severe infection the tissue is permeated with extensive extravasations of blood and is often to a large extent necrotic. In the ciliary body it is mainly the lax tissue which adjoins the sinus of the anterior chamber that is infiltrated with leucocytes; and when the infiltration is great this tissue often can scarcely be differentiated from the hypopyon. From the inflamed tissue the exudate migrates into the aqueous chamber; the endothelial membrane, which lines the sinus of the chamber and the iris being at the same time exfoliated. The exudate coagulates on the surface of the iris, forming a layer which consists of a delicate network of coagulated fibrin, which encloses leucocytes in varying amounts (Fig. 301). This coating of fibrin extends, on the one hand, within the pupillary area over upon the anterior capsule (Fig. 302), and, on the other hand, it often also covers the posterior surface of the cornea (Fig. 313). The aqueous chamber may even be entirely filled by such an exudate (Fig. 312). The more severe the inflammation is, the more leucocytes the exudate contains, and the hypopyon consists almost exclusively of leucocytes.

In less severe cases in which we have to do with an exudate of a more fibrinous character, the latter may disappear without leaving a trace or may lead to only a partial adhesion of the iris to the lens capsule. In severe cases the exudate becomes organized into connective tissue. The iris is then covered by a connective-tissue pellicle (Fig. 303), which is often quite thick and not infrequently contains new-formed vessels, which have grown from the iris into the membrane. The surface of this pellicle is sometimes covered by a new-formed lamina vitrea which at the angle of the chamber becomes continuous with Descemet's membrane. From the surface of the iris the iritic membrane, crossing the pupillary margin which is solidly adherent to the lens capsule, passes into the pupil and closes it up (Figs. 303 and 304). The organized exudate often glues the periphery of the iris to the cornea and may also in places cover the posterior surface of the latter. The iris itself gets to be atrophic. It is thinner and its lax reticulate tissue is transformed into a rigid fibrillary connective tissue. The gracefully branched chromatophores are in large part changed to thick round cells filled with coarse pigment granules (Fig. 301). The blood-vessels have thickened walls of a hyaline appearance, and many are quite obliterated. The sphincter pupillæ and the retinal pigment offer the longest resistance to the atrophy.

A special form of exudation in the region of the anterior chamber are the *precipitates.* They do not occur at all in acute inflammation due to a perforating injury, and at most occur only when such an inflammation runs a chronic course (iritis traumatica serosa). On the other hand, they are an almost constant occurrence in chronic nontraumatic inflammations of the iris and ciliary body. Hence precipitates and hypopyon, which is an accompaniment of acute inflammation, are not usually found together. The precipitates are conglomerates of round cells, many of which contain pigment granules, thus showing their origin from the uvea (Fig. 313, *P*). The precipitates lie on the endothelium of Descemet's membrane, which at the outset is perfectly normal, and not till later on undergoes destruction beneath the accumulations of cells formed by the precipitate (Fig. 313, *e*).

697. (*b*) **Changes in the Posterior Chamber**—The more severe the irido-cyclitis, the more does the tissue that lines the posterior chamber share in the inflammation.

Then it is above all the ciliary processes and mainly the most superficial portions of their tissue that are infiltrated with emigrated leucocytes. The exudate that comes out on the surface detaches the two retinal layers of the ciliary processes and covers the surface of the processes (Fig. 312).

The exudate poured out into the posterior chamber afterwards becomes organized into the cyclitic membranes, which form solid adhesions between the lens on the one hand and the iris and ciliary processes on the other (Fig. 309). The ciliary processes which are imbedded in the membranes become atrophic; on their surface the unpigmented and the pigmented layers of the retinal lining undergo proliferation, and send long rows of cells

into the firm connective tissue of the membrane, which, moreover, contains new-formed vessels emanating from the ciliary processes. The lens gradually becomes opaque. If the lens capsule is injured either as a result of the traumatism itself or later as a result of its destruction by the exudate pressing against it, the lens substance disintegrates and gradually disappears and the exudate enters the capsular sac. This exudate here, too, becomes organized to form connective tissue, and may ultimately become ossified, so that the picture—to be sure, only an apparent one—of ossification of the lens is produced. The cyclitic membrane is often extremely tough, so that it grates when cut through; not

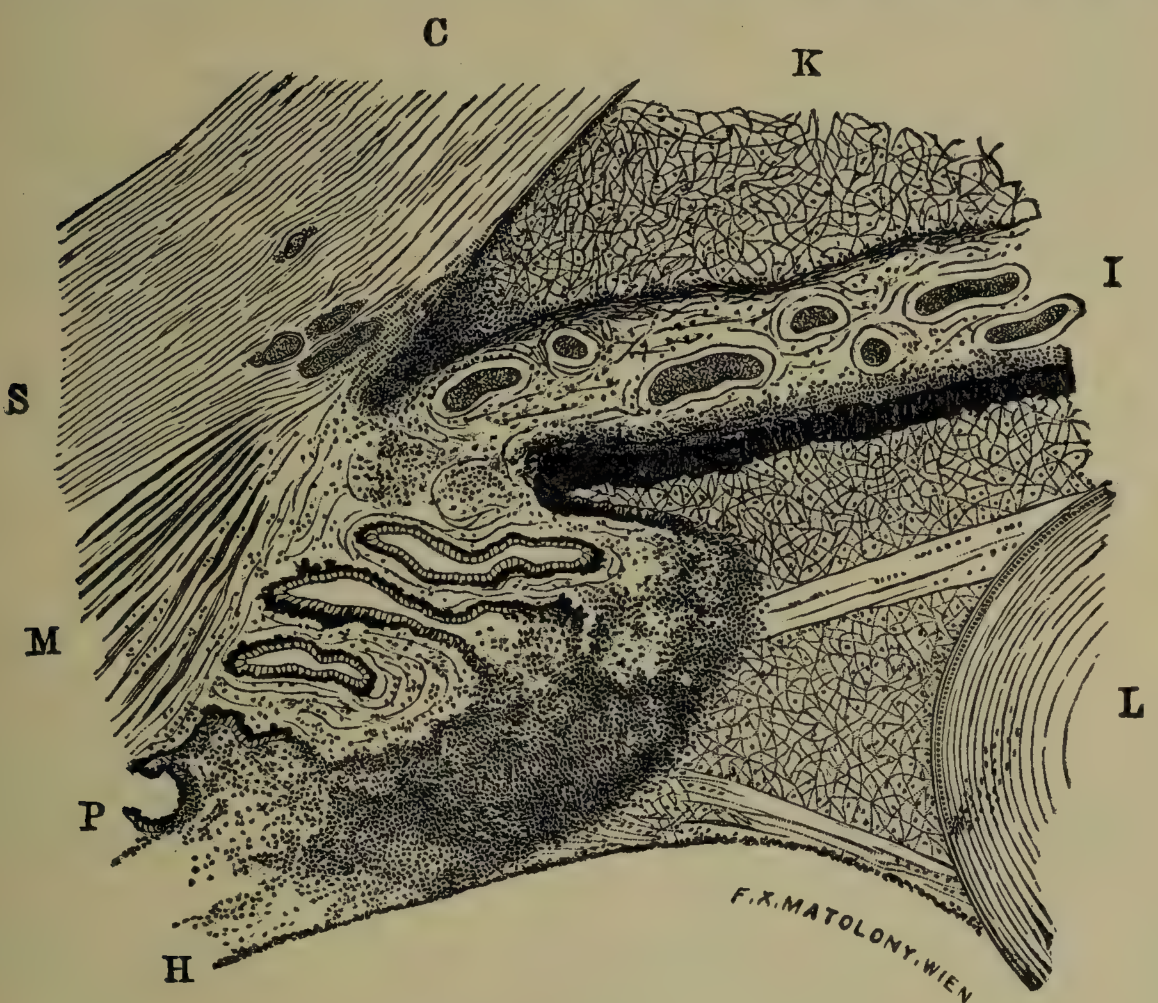


FIG. 312.—IRIDO-CYCLITIS AFTER PERFORATING INJURY. Magnified 30 × 1.

The cornea, *C*, and the sclera, *S*, are unchanged. Owing to the fact that the aqueous is rich in fibrin, and hence is coagulated by the hardening fluids used, the anterior chamber, *K*, is filled with a dense network of fibrin, in which lie isolated round cells (emigrated white blood corpuscles). These round cells are particularly numerous upon the surface of the iris, and above all in the sinus of the chamber; in fact, they fill the sinus completely, and form in it a low hypopyon which was visible in the living eye. A marked cellular infiltration can be seen surrounding Schlemm's canal and the cross sections of the anterior ciliary veins situated above the canal. The iris, *I*, appears broadened, as seen in cross section, this appearance corresponding with the swelling which existed in the living eye. Its vessels are dilated, and are distended with blood. In its stroma, particularly in the posterior layers, there are cells visible in great numbers; its retinal pigment layer is thickened and loosened. The greatest emigration of cells has taken place from the blood-vessels of the ciliary body, the direction pursued being a centripetal one—i.e., toward the interior of the eye. Hence the infiltration in the ciliary muscle, *M*, is but slight, in the ciliary processes, *P*, it is rather more pronounced, and upon the surface of the latter it is the most marked of all, so that the outer limits of the processes are concealed by the dense masses of cells. The hyaloid membrane, *H*, is pressed away by the exudate from the surface of the ciliary body. The contents of the posterior chamber, like those of the anterior, are formed of a fine network of fibrin with leucocytes imbedded in it, these latter being formed in specially large numbers along the hyaloid membrane. Two of the groups of fibres composing the zonule of Zinn are seen as light-colored bands, passing through the coagulated contents of the posterior chamber to the lens *L*. The latter displays the epithelium of its anterior capsule and the nuclear ring and shows no morbid change.

infrequently it contains small laminæ of bone. Owing to its contraction, the ciliary processes are elongated more and more and are drawn into the interior of the eye. This constant traction upon a tissue, so richly supplied with nerves, is one of the causes of the constantly present or constantly recurring pain that distresses the patient in so many cases of old irido-cyclitis.

698. (*c*) **Changes in the Vitreous**—Since that surface of the ciliary body which looks backward and inward adjoins the vitreous and is separated from it only by fibres of

the zonula, it is easy to understand that in case of severe inflammation leucocytes will pass from the ciliary body into the vitreous. So in many cases in which the infection at first has affected only the aqueous chamber, it happens that later the vitreous cavity is also attacked by the inflammation. In other cases the inflammation begins in the vitreous cavity, this occurring when the agents causing the inflammation have been introduced into the latter first. For inflammation thus originating in the posterior segment of the uvea see §§ 752-757.

The changes in the vitreous usually lead to either atrophy or phthisis of the eyeball. *Atrophy of the eyeball* is caused by the shrinking of connective tissue produced by the organization of exudates in plastic iritis or sometimes of connective tissue formed around an abscess of the vitreous. The connective tissue draws up to it first the retina, which is readily movable anyhow and is already detached, and afterwards the ciliary processes and through these the anterior portion of the chorioid. Ultimately there is formed in place of the vitreous a small-sized mass of tough connective tissue, bounded in front by the lens, which is usually opaque and shrunken, and by the iris, which is joined

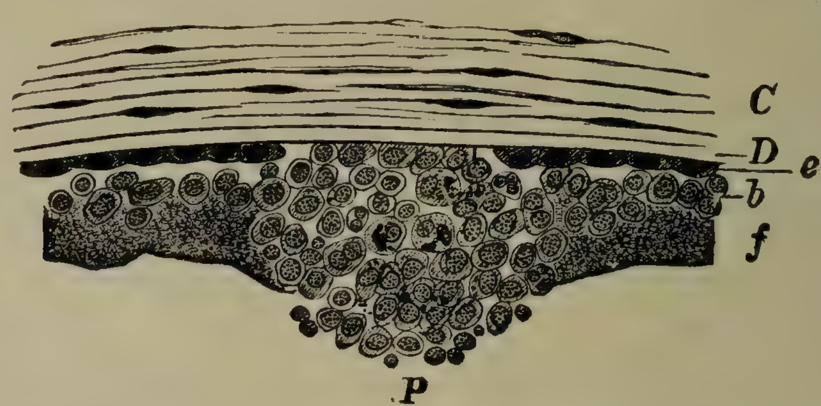


FIG. 313.—DEPOSIT UPON THE POSTERIOR SURFACE OF THE CORNEA. Magnified 140×1
The posterior surface of the cornea, *C*, is covered by Descemet's membrane, *D*, and the endothelium, *e*. The latter, which as a whole is of normal character, is wanting at the spot where the deposit, *P*, is situated. This deposit forms an accumulation of cells with interspersed pigment granules which are partly free and partly inclosed in the round cells. In the place where no deposits are situated the posterior surface of the cornea is covered by a layer of exudation consisting of two strata, an anterior one, *b*, composed of round cells, and a posterior one, *f*, formed of coagulated fibrin.

to the lens. These two latter structures are sometimes driven forward, sometimes drawn backward, the anterior chamber being thus either abnormally shallow or abnormally deep. The former condition results when the cyclitic membrane, which originally was concave anteriorly, is stretched into a plane by the process of shrinking; the latter condition develops when the shrinking makes its influence felt more in a direction from before backward. Laterally the cyclitic membrane is bounded by the ciliary processes, behind by the wrinkled retina. Beneath the latter is a transudate which is rich in albumin and often contains blood. A similar liquid is found beneath the chorioid between dissevered lamellæ of the suprachorioid. The chorioid itself is often but little changed. If, however, it has participated to a rather large extent in the inflammatory process, an organized exudate forms upon its surface. Subsequently ossification often occurs in the chorioid or in the exudate lying upon it, so that in atrophic eyes there are found here sometimes scattered laminæ of bone, sometimes a thin plate of bone of quite large size lining the posterior portion of the eyeball.

For *phthisis of the eyeball* and the changes that lead to it see § 755.

699. Changes in Sympathetic Inflammation.—A special variety of ectogenous infection is that which underlies sympathetic inflammation (§ 710). While in the ordinary cases of infection the exudation on the free surface of the coats of the eye plays the chief part, here, as in endogenous infection, the main thing is an infiltration of the tissue itself, which, moreover, is of a peculiar kind.

This infiltration has been especially studied in the exciting eye (that is the one which was injured and is the first affected). Here, in addition to the evidences of an

ordinary plastic irido-cyclitis, such as cyclitic membranes with detachment of the retina, we find the uvea distended with densely crowded lymphocytes and plasma cells. In most cases there lie in the midst of this uniform infiltration focal collections of large (epithelioid) cells, which not infrequently have giant cells between them (Fig. 315). Nodules are thus produced which often are like tuberculous nodules. This peculiar infiltration often is present in only a few spots, so that isolated nodules are seen here and there in the iris, ciliary body, or chorioid. In other cases the uvea is occupied by them either largely or wholly and thus often becomes extremely thickened so as to fill, more or less completely, the interior of the eye (Fig. 314). Sometimes the infiltration even makes its way into the sclera, which is permeated with scattered nodules (Fig. 314, *b*), and in this way perforation of the sclera and extra-ocular proliferation may result.

Up to the present time it has not been possible to demonstrate the presence of bacteria in the nodules. Nevertheless there is scarcely a doubt but that here too we are

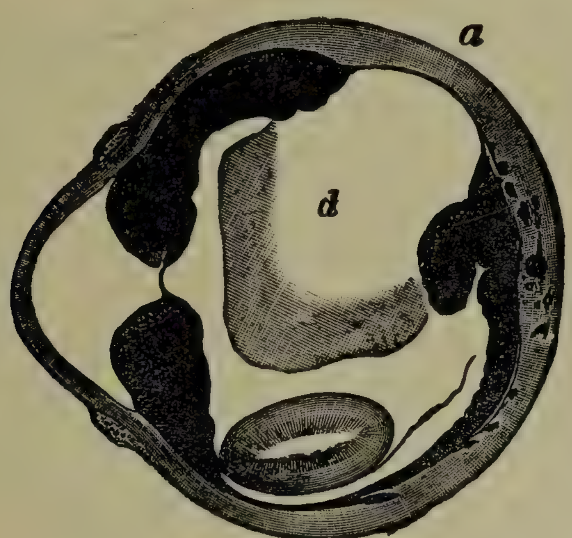


FIG. 314.—INFLAMMATION PRODUCING SYMPATHETIC OPHTHALMIA. Magnified 2 × 1.

A girl twenty years of age had a detachment of the retina, which developed in the right eye without any known cause. The eye gradually became blind but it was not till three years later that it was attacked suddenly by a very severe and painful inflammation, which, after four weeks, went over to the left eye. The latter presented the symptoms of a severe sympathetic irido-cyclitis and also became blind in spite of the immediate enucleation of the right eye. This was one of those rare cases in which a sarcoma, beginning in the chorioid, first produces blindness through detachment of the retina and afterwards becomes necrotic, and thus causes a severe inflammation in the eye. The vertical section of the eye shows the iris greatly thickened by an excessively dense infiltration. The pigment that is visible in it lies partly in the very numerous giant cells. The iris everywhere merges without any sharp line of demarcation into the also thickened ciliary body, and the latter again merges into the chorioid. Of the retina there is nothing to be seen. Below, at *c*, the lens which has been dislocated into the vitreous lies in contact with the chorioid. At *a* the chorioid is replaced by a mass, *d*, without distinct structure, which extends thence into the posterior cavity of the eye and fills a large part of the latter. This is the necrotic sarcoma. At *c* the infiltration of the chorioid passes along a vena vorticosa into the sclera; further back at *b*, nodules of the same tissue, but unconnected with it, lie in the sclera. The pupil is closed by a pigmented membrane.

dealing with an affection produced by bacteria, which, however, do not cause acute suppuration but, after the analogy of many other bacteria (for instance the tubercle bacilli), cause chronic proliferations. This inflammation has the property of being transmissible to the other eye. Of the sympathizing, that is the secondarily affected eyes, only a few have so far been got for examination, but then generally the same peculiar changes have been found.

700. (2) Endogenous Infection.—In endogenous infection the inflammatory agents get into the uvea from the blood-vessels and hence exert their primary effect within the tissue itself instead of upon its surface as in ectogenous infection.

The inflammatory agents may be the pyogenic bacteria as in the case of *metastatic ophthalmia* (see § 756), which produces purulent disintegration and necrosis, usually terminating in panophthalmitis, rarely in incapsulated abscess—in this bearing a clinical resemblance to purulent ectogenous infection (cf. § 692).

The forms of iritis and irido-cyclitis which accompany *acute and chronic infectious diseases* (cf. §§702–707 et seq) are without doubt in part also due to endogenous infection by bacteria, in part perhaps to the action of the toxins which are formed in the body. In any case the inflammations thus produced are not so violent and destructive as those due to infection by the pyogenic germs. The exudation develops more gradually and remains more confined to the tissue itself. The tissue is infiltrated chiefly with uninuclear leucocytes, which tend to accumulate about the blood-vessels under the form of scattered foci. Thus along with the diffuse infiltration there are formed nodular exudates which in the ordinary traumatic inflammations are only exceptionally met with. The nodules are generally so small that they are seen only on histological examination (Fig. 255), but they may be so large that they are visible clinically (papules, tuberculous nodules of the iris). In correspondence with the non-suppurative character of the inflammation is its outcome, which is also less serious. Panophthalmitis never results and even atrophy of the eyeball is a comparatively rare sequel, occurring only in particularly severe or in often recurring cases.

701. Etiology of Iritis and Cyclitis.—Iritis and cyclitis are either primary or secondary in their development. In the first case, the original site of the disease is in the iris or ciliary body itself; in the second case, there is an affection of neighboring parts, which has been transmitted to the iris and the ciliary body (e.g., in iritis with ulcer of the cornea).

We may classify the inflammations of the iris and the ciliary body with reference to their etiology according to the subjoined scheme. In it, as well as in the subsequent description, the expression "iritis" stands, for the sake of brevity, as the representative of iritis, cyclitis, irido-cyclitis, and irido-chorioiditis—that is, for all inflammations which affect predominantly the anterior part of the uvea:

- A. Primary iritis.
  - Iritis in consequence of general diseases.
    1. Iritis syphilitica.
    2. Iritis gonorrhoica and focal iritis.
    3. Iritis in acute infectious diseases (iritis metastatica)
    4. Iritis in disorders of metabolism.
    5. Iritis tuberculosa.
  - Iritis as a local affection.
    1. Iritis traumatica.
    2. Iritis sympathica.
- B. Secondary iritis.

This classification is evidently not an exhaustive one. There are many cases of iritis for which no cause can be discovered and which, therefore, cannot be placed under the classification given above. There is no doubt but that most of these cases, too, although called idiopathic, depend on general diseases, especially on anomalies of metabolism, which at present are unknown to us, or on obscure infections.

Iritis, whether primary or secondary, is due, in the great majority of cases, to *micro-organisms* or their toxins even though often we can not tell which micro-organism is acting. A tentative classification may be made as follows:

A. Infectious iritis, due to the—
1. Spirochæta pallida.
2. Gonococcus.
3. Staphylococcus and streptococcus.
4. Tubercle bacillus.
5. Other organisms.

B. Metabolic iritis, due to—
Diabetes, gout, hypothyroidism.

Even this classification is not altogether satisfactory. Many, and perhaps the majority of cases of iritis occurring in metabolic disorders, are probably due to micro-organisms or toxins, the altered metabolism simply lowering the resistance so that infection can take place. Traumatic iritis is undoubtedly almost always due to infection.

702. (*A*) **Primary Iritis.**—(*a*) *Due to General Diseases.* These are either infectious diseases (syphilis, etc.) or diseases of metabolism (gout, diabetes). In such cases both eyes, although not both at the same time, are often attacked by the iritis.

In cases where no general disease is discoverable, a cold is often accused of being the cause. It is certain that in a man predisposed to iritis exposure to cold may start

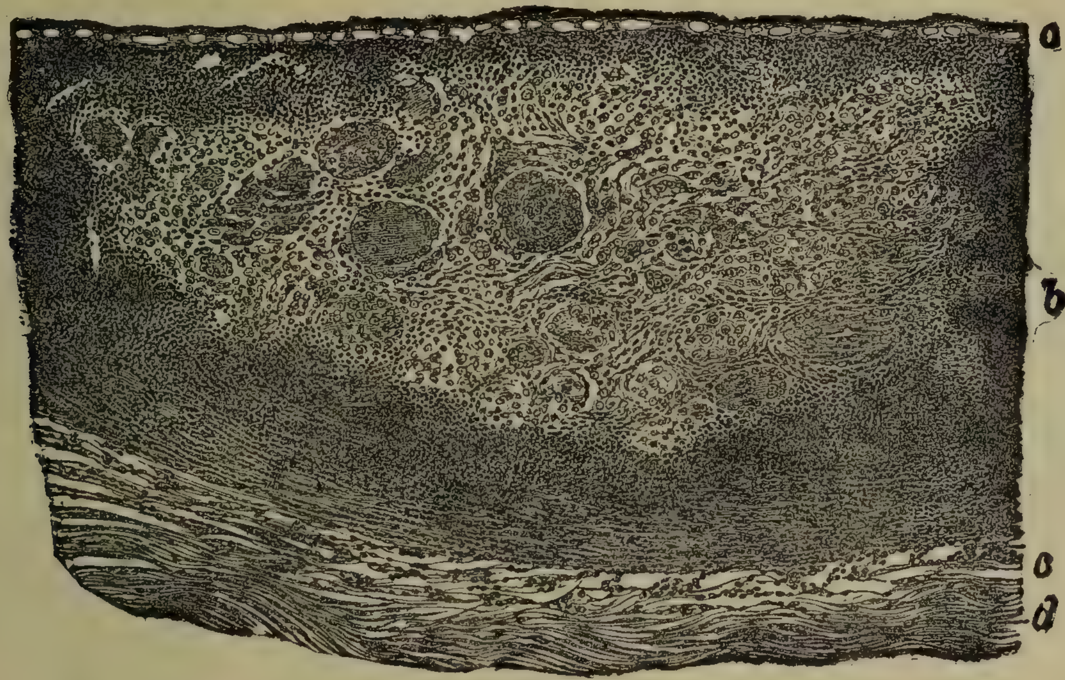


FIG. 315.—INFLAMMATION CAUSING SYMPATHETIC OPHTHALMIA.—DEGENERATED CHORIOID. Magnified 100 × 1.

Beneath the lamina vitrea can be recognized the lumen of the capillaries, *a*. The infiltration of the chorioid, *b*, begins beneath the chorio-capillaris and stops at the suprachorioid, *c*. *d*, innermost lamellæ of the sclera. The chorioid is very densely packed with lymphocytes. In the midst of this infiltration lies a large focus which on account of its lighter color can be seen even with the naked eye. It consists of large pale cells with a large nucleus (epithelioid cells) and of numerous large and small giant cells with the typical annular arrangement of the nuclei. A few giant cells lie outside of the focus and in the midst of the infiltration of small cells.

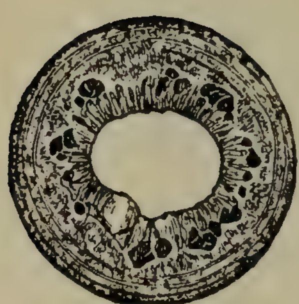
an attack, and recurrences of iritis are particularly apt to be caused by it. On the other hand, it is a question whether exposure to cold can by itself produce iritis.

Primary iritis is found mainly in adults; in childhood primary iritis is rare. Acute iritis (especially the syphilitic and gonorrhœal forms) more often affects men, chronic iritis more often affects women. The forms of iritis that are based on general diseases are:—

703. (1) **Syphilitic Iritis.**—Syphilis is by far the most frequent cause of iritis. For the most part, it is *acquired* syphilis that we have to do with. The diagnosis of syphilitic iritis is very easy in those cases in which the characteristic formation of nodules (iritis papulosa, Fig. 316) is present. The nodules have a yellowish-red color, are of the size of a pin's head or larger, and are situated either on the ciliary or the pupillary margin of the iris—rarely between the two zones in the mid-breadth of the iris. The nodules afterward disappear by resorption without any purulent disintegration taking place. At the places where they were situated, broad and firm

synechiæ are left, and there is often also a circumscribed atrophy of the iris. At other times no distinct nodules are found, these being so small that they lie concealed within the tissue of the iris, but several portions of the pupillary margin are greatly swollen, or at all events there are unusually broad synechiæ which do not yield to atropine (*b*, Fig. 307). Finally, in many cases iritis syphilitica presents absolutely no characteristic marks; the diagnosis, then, can be certainly established only by demonstrating the presence of syphilis, or by the favorable action of antisyphilitic remedies.

Syphilitic iritis generally belongs to the secondary stage of syphilis.



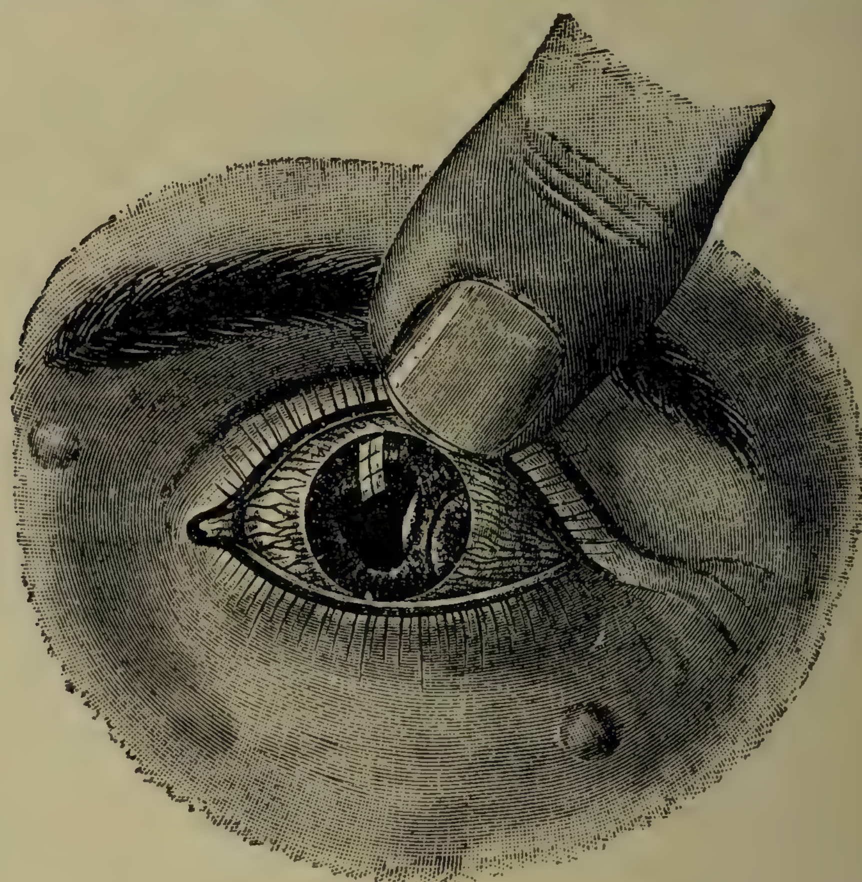


FIG. 316. FIG. 317.

FIG. 316.—IRITIS PAPULOSA. Magnified 2 × 1. In a woman 38 years of age the left eye had become inflamed eight days before. The woman was not conscious of having syphilis, and, except for some enlarged lymph glands, showed no symptoms of this disease, but her husband states that seven months previous he had had a hard chancre and soon after this an eruption. The eye is injected, and on the lower part of the cornea there are some minute precipitates. The pupil has dilated but little under atropine; the margin of the pupil in many places is adherent to the lens capsule and is hence indented. In the lower quadrant there is situated in the pupillary zone of the iris the yellowish-red, not very sharply defined papule which juts somewhat into the pupil. The dark spots at the periphery of the pupillary zone are crypts.

FIG. 317.—SYPHILITIC TUMOR OF THE CILIARY BODY. The patient, a man thirty years of age, acquired syphilis four months ago and got an exanthem one month later. The eye has been inflamed for one month. There are universal glandular swellings and a papular exanthem spread over the whole body. Of this some efflorescences are visible in the vicinity of the eye. The eye is markedly injected. The cornea has some precipitates on it. At its temporal side the margin of the pupil is contracted from an arc to a chord, because at this spot the iris is detached from its insertion and pushed towards the centre. In the area occupied by this iridodialysis there is exposed to view a yellowish-red vascular tumor which belongs to the ciliary body and whose light-colored apex is visible in the pupil.

It makes its appearance generally within the first year after infection has taken place and soon after the first eruptions upon the skin (macular or papular), for which reason we may compare the nodules in the iris to papules or to condylomata, and may designate the iritis as iritis papulosa (or condylomatosa). That these nodules are not gummata is shown not only by their appearance in the early stage of syphilis but also by the fact that they never break down nor suppurate. More rarely the iritis breaks out in the later stages of syphilis, and is then not usually associated with the

formation of nodules. In exceptional cases, however, nodules do show themselves in this late form also and they must then be regarded as gummata (iritis gummosa).

Syphilitic nodules occur in the *ciliary body* as well as in the iris. Of the little nodules that come to view on the ciliary border of the iris many certainly start from the ciliary body. Larger nodules in the ciliary body push the iris away from the cornea-scleral margin and can also be seen behind the pupil (Fig. 317); and they may grow through the sclera, and produce perforation and atrophy of the eyeball.

Iritis also occurs in consequence of *hereditary* syphilis, although far less frequently than after acquired syphilis. The parenchymatous keratitis, which is dependent upon hereditary syphilis, is often associated with iritis. Sometimes it happens that the iritis becomes, comparatively speaking, very conspicuous, while the keratitis attains but a slight degree of prominence; and there may even be iritis without any keratitis whatever. Iritis due to hereditary syphilis is a disease of childhood and youth, while iritis resulting from acquired syphilis is usually observed only in adults.

Syphilitic iritis also occurs during *intra-uterine life;* the children then come into the world with the remains of it, such as synechiæ, occlusion of the pupil, atrophy of the iris, and even atrophy of the eyeball.

Syphilitic iritis shows a great tendency to recur. Moreover syphilis, especially hereditary syphilis, instead of producing an acute inflammation, may set up a sluggish *chronic iritis* or irido-chorioiditis.

Syphilitic iritis is very frequently associated with syphilitic disease of the posterior section of the eye—that is, with inflammation of the chorioid. retina, and optic nerve.

Varying statistics indicate that from 20 to 70 per cent of all cases of primary iritis are due to syphilis. Perhaps Jenning and Hill's estimate of 61 per cent is nearest the truth. The proportion must vary, though, in different countries. The significance of syphilis and gonorrhœa as a cause of iritis is proved by the fact that in Palestine, where the former diseases are almost unknown, iritis is rare (Butler).

In the earliest stages of syphilis before roseola appears in the skin a very transient *roseola of the iris* may develop, manifested by a mottled or speckled appearance, due to a hyperæmia especially in the vessels of the lesser circle of the iris (Krückmann).

The diagnosis of syphilitic iritis will, of course, always have to be based upon the history of the case or upon the demonstration of the presence of syphilis in the patient particularly by the Wassermann test. But we must not call every iritis which has no characteristic marks, syphilitic, simply because it occurs in a syphilitic person. A syphilitic patient may acquire an iritis from other causes than syphilis. Among such causes one that requires special consideration is gonorrhœa, which almost all syphilitics have had at some time or other, and which is likewise a frequent cause of iritis. In doubtful cases, the indication in every case is to initiate an antisyphilitic treatment. This will, in most cases, cause rapid improvement in iritis of syphilitic origin, while other kinds of cases are but little or not at all affected; from which fact a conclusion may be drawn as to the source of the iritis. The effect of antisyphilitic treatment is also of assistance in making the diagnosis in those cases in which we are in doubt as to whether a nodule in the iris is of syphilitic nature or must be regarded as a new formation (sarcoma, tubercle). It must be noted that even in syphilitic iritis, the disease may be kept up or relapses caused by other infections (e.g., oral infections) (De Schweinitz). Cf. Dunn's theory of the causation of iritis in parenchymatous keratitis (page 572).

704. (1) **Focal Iritis**—In many cases iritis originates in a focus of purulent infection. Such focal infection may have its primary seat in the genito-

urinary tract, being then usually due to the gonococcus (*gonorrhœal iritis*), sometimes to the colon bacillus. In other cases it is derived from the tonsils, teeth (especially pockets at the roots of dead teeth), or, less often, from the accessory nasal sinuses. In these cases it is due usually to the streptococcus viridans (*streptococcic iritis*), less often to other germs (staphylococcus, influenza bacillus). Often associated with the iritis either as coincident or intercurrent affections, are inflammations of the joints or heart (arthritis, endocarditis). Other evidences of general toxæmia (mental and physical depresson, tendency to nervous exhaustion, impaired accommodation, great asthenopia, etc.) may be produced by the same infection. The iritis, especially when due to gonorrhœal infection often runs an acute course with marked inflammatory symptoms and with frequent recurrences. Such cases are hence often grouped under the name of *acute recurrent iritis*. In contrast with other—especially the syphilitic—iritides, the permanent changes produced by the initial stormy attack are often very slight. Frequently not a single synechia is left. Permanent and grave changes, however, may be induced by the repeated recurrences. Such recurrences are coincident with recurrences of the joint inflammations and also, in the case of gonorrhœal iritis, with recurrences of the urethral discharge. Because of its connection with joint symptoms and because of the disposition that the patients show to develop an attack on exposure to cold, the non-gonorrhœal form of acute recurrent iritis has usually been designated as *rheumatic iritis*.

In other cases focal iritis occurs under the guise of an obstinate chronic cyclitis or uveitis with often destructive changes; and in still other cases it complicates and prolongs an iritis due to other causes (cf. § 703).

From 8 to 20 per cent of the cases of iritis are due to *gonorrhœa*, so that probably at least 75 per cent of all cases of iritis are caused by either this disease or by syphilis. Gonorrhœa may also produce a purulent metastatic ophthalmia (see § 756).

In focal iritis complement fixation tests may reveal the presence of the exciting organisms when other evidence is lacking (*latent* gonococcæmia, etc.—Reber).

705. (3) **Metastatic Iritis.**—Allied to focal iritis is the iritis produced by metastasis in *acute infectious diseases*. In some cases this assumes the form of metastatic ophthalmia (see § 756) with suppuration and destruction of the eye; in other cases occurs as an ordinary plastic iritis. This latter form occurs especially in *relapsing fever* (in 2-12 per cent. of the cases). It sets in some weeks or even months after the last attack of fever and may either run an acute course with hypopyon, gelatinous exudation, and synechiæ, or as a chronic iritis. In both forms there are marked opacities of the vitreous. The iritis takes one or two months to run its course, but ultimately goes on to a cure.

Iritis also occurs frequently—in 44 per cent of the cases—in *Weils' disease* (icterus infectiosus). It sets in about the end of the first week of the disease and often in at most six weeks ends in a complete cure (Strassberger).

Iritis also occurs in conjunction with *parotitis*. A form in which uveitis is associated with parotitis and slight fever is the *febris uveo-parotidea* (Heerfordt). In about half the cases there is a peripheral facial paralysis and sometimes the lacrimal glands, the lymph glands, and other salivary glands beside the parotid are inflamed. Gray, yellowish, or

brownish-red nodules develop in the iris. The glandular disease clears up completely, but the eye usually suffers serious permanent injury and may even become blind. The condition is sometimes due to tuberculosis.

Other infectious diseases which exceptionally give rise to iritis are pneumonia, pertussis, intermittent fever, typhus, dysentery, influenza, variola, erysipelas, purpura hæmorrhagica, and peliosis rheumatica. The iritis, moreover, that sometimes accompanies herpes zoster should probably be put down under this head. Some few cases are known in women in whom a transient iritis with hypopyon has recurred regularly with menstruation.

706. (4) **Metabolic Iritis.**—Iritis in some cases is caused by disorders of assimilation or nutrition or disturbance of the endocrine secretions. Thus iritis or uveitis may be caused by intestinal autotoxæmia and acute iritis by gout or arthritis deformans,—less often by diabetes.

Gouty iritis (iritis urica) is associated usually with marked hyperæmia and even chemosis of the conjunctiva. Recurrences are frequent. The children of gouty subjects occasionally develop a sluggish destructive iritis or rather uveitis, associated with disease of the vitreous (De Schweinitz).

It seems likely that in some cases iritis or uveitis may be due to *hypothyroidism.*

As already noted (§ 701), in iritis occurring with disorders of metabolism it is a question whether the connection is a direct or an indirect one. An iritis, for example, occurring with gastro-intestinal disturbance may be due not so much to absorption of the products of perverted metabolism as to the toxins of the infection that produced the metabolic disorder. Again, the iritis occurring with diabetes and hypothyroidism may be due to an infection, to which the system, because of the disorder of metabolism, offers less resistance. This, of course, does not mean that the metabolic disorder is not of considerable significance and that its removal does not exert a favorable effect on the iritis.

707. (5) **Iritis Tuberculosa.**—This may appear in the iris under the guise of miliary nodules or of a rather large solitary tubercle. The latter will be considered in § 729. Frequently, however, tuberculous and scrofulous persons, especially the young, suffer from an iritis or cycilitis, which is not accompanied by tuberculous nodules, but still must probably be regarded as tuberculous. This is often marked by the presence of large, lardaceous-looking deposits or by lardaceous exudation masses, which appear to grow out from the sinus of the chamber. It is found in childhood and in youth, in scrofulous persons and also in those suffering simply from anæmia.

In other cases tuberculosis produces a chronic uveitis.

In tuberculous iritis extremely minute grayish nodules may be made out in the iris with the higher powers of the corneal microscope. Such nodules, if transient, are denoted as *tuberculides* (Gilbert). A positive diagnosis that an iritis is tuberculous can in general be made only from the focal reaction with tuberculin (§ 140).

708. Heterochromic Cyclitis.—If the two eyes have a different color (e.g., one has a brown the other a blue iris—a condition called heterochromia iridis), a very chronic cyclitis with deposits on the cornea and with the formation of cataract sometimes develops in the lighter colored eye. Sometimes a marked cyclitis with degenerative changes may be present when the difference in color is extremely slight, one iris being simply a lighter blue or gray than the other, and the cyclitis may begin several years before the difference in color of the irides is noticed. Some of these cases, at least, seem to be due to tuberculosis; in others the cause is unknown.

709. (*b*) *Iritis as a Local Affection.*

(1) **Iritis Traumatica.**—The causes of this are traumatism of all kinds, especially if perforation of the eyeball has taken place, and particularly if a

foreign body is left in the eye. Operations upon the eyeball are, of course to be counted among the traumatisms, and of these the most dangerous with regard to iritis and irido-cyclitis are the cataract operations.

The irido-cyclitis that follows traumatism is frequently of the most serious nature, so that atrophy of the eyeball speedily ensues; in contradistinction to iritis due to constitutional causes, which even when violent often gets well without leaving any ill results worth mentioning.

The exciting cause of traumatic iritis in cases of the sort just adduced may be of three kinds; either mechanical injury (traction, contusion), chemical irritation (e.g., in the case of contact with swollen portions of the lens or in the case of a cysticercus); or, finally, infection from without. The last-named cause is without doubt the most frequent.

In traumatic (including post-operative) irido-cyclitis, exacerbation of the symptoms may occur periodically and often at quite regular intervals (every 3, 5, 7 days) (De Schweinitz).

710. (2) **Irido-cyclitis Sympathica.**—When an eye is affected with irido-cyclitis in consequence of an injury, either symptoms of irritation or else an actual inflammation may develop in the other eye.

Sympathetic irritation (irritatio sympathica) consists in photophobia, lacrimation, or actual pain. Sometimes also weakness of accommodation is present, so that when the patient tries to do fine work the vision gets indistinct and the work has to be given up. Such symptoms, however, are to be called by the name of sympathetic irritation only when objective signs of inflammation are absent, for when once these make their appearance, it is a question, not of sympathetic irritation, but of sympathetic inflammation. A characteristic sign, furthermore, of sympathetic irritation is that it disappears at once and forever when the primarily affected eye is removed.

711. *Sympathetic inflammation* (ophthalmia sympathica) consists in the development of an irido-cyclitis in the second eye. The eye primarily affected is called the "exciting eye," that which is affected secondarily the "sympathizing eye." Sympathetic inflammation develops sometimes in immediate conjunction with preceding symptoms of sympathetic irritation, sometimes without any intermediary symptoms at all and quite unforeseen.

The beginning is often insidious. In conjunction with an insignificant reddening of the eye, deposits—which are never wanting in the beginning of a sympathetic ophthalmia—appear upon the cornea. Then a few posterior synechiæ and fine opacities in the vitreous develop, and the ophthalmoscope shows hyperæmia of the retina and optic nerve. In favorable cases the disease reaches no higher pitch and at length gets well, leaving either no trace of its presence or a few posterior synechiæ. Unfortunately such mild cases are the exception. The rule is that the symptoms of inflammation slowly or quickly increase; more and more adhesions of the iris develop, and the visual power becomes more and more reduced. Hypopyon, however, ordinarily does not occur even when the inflammation is severe. Although sometimes intervals in the inflammation with some improvement in sight occur, yet these are not lasting, and finally, owing to the constantly recurring

inflammation, there ensues in spite of all treatment the formation of a pupillary membrane and of an annular or total posterior synechia. Then, in consequence of the seclusion of the pupil, increase of tension may set in, but this is commonly not of long duration, since cyclitic membranes have also formed in the vitreous, and by their shrinkage the intra-ocular pressure is once more lowered and finally atrophy of the eyeball is produced. As a rule, therefore, the sympathizing eye is lost.

In view of the great gravity of sympathetic ophthalmia, it is important to know its early danger signs, i.e., those that occur before the stage of actual exudation. According to Brownlie, these are a contraction of the visual field, a spindle-shaped enlargement (elongation of the vertical diameter) of the blind spot, congestion of the optic disc and retinal vessels, loss of visual acuity, paresis of accommodation, and changes in the blood count (marked increase in the large uninuclear leucocytes, some increase in the lymphocytes, decrease in the polymorphonuclear cells). According to Gifford, however, the blood changes, although well-marked in sympathetic ophthalmia, are not pathognomonic since they are found frequently in chronic uveitis and like conditions.

For blanching of the eyebrows in sympathetic ophthalmia, see § 681.

It is supposed that the sympathetic disease may appear not only in the form of an irido-cyclitis, but also under some other guise. The greatest variety of affections have been described as sympathetic. Among non-inflammatory affections, cases of paralysis of accommodation, of amblyopia, and of blepharospasm have been adduced as sympathetic; among inflammatory affections in the posterior division of the eye, neuritis, chorioiditis, and glaucoma; and in the anterior division of the eye, conjunctivitis and keratitis. However, the only conditions in which the connection has been surely proved are sympathetic neuritis and chorioiditis. The former occurs very rarely and gives a comparatively good prognosis. As regards the chorioiditis, this probably is present in all severe cases, of sympathetic irido-cyclitis, but cannot be diagnosticated, because the cloudiness of the media prevents ophthalmoscopic examination. Hence we get the ophthalmoscopic picture of chorioiditis only in those comparatively mild cases, in which the anterior segment of the uvea is affected but little or not at all; and such cases are rare. Sympathetic chorioiditis is marked by the presence of numerous small yellow patches which occupy especially the periphery of the fundus (see § 743). In other cases observers have often gone too far in taking the sympathetic nature of the disease for granted. The fact that an eye has been destroyed through traumatism by no means justifies us in regarding, without further proof, any subsequent disease of the other eye as sympathetic. This assumption should be made only when such disease presents the characteristic clinical picture of sympathetic irido-cyclitis, or when, upon the enucleation of the eye first diseased, the symptoms in the second eye recede too rapidly to be accounted for upon any other assumption than that the affection of the second eye was caused by that of the first. The converse of this inference does not hold good—that is, the fact that enucleation of the first eye does not influence the course of the disease in the second is no argument against the sympathetic nature of the lesion; indeed, it is a well-established fact that when sympathetic ophthalmia has once broken out, enucleation of the eye first diseased is not generally able to cause much change.

712. The affection of the *exciting* eye, which gives rise to a sympathetic inflammation, is always an irido-cyclitis, and is, in fact, almost without exception, an irido-cyclitis traumatica, due to a penetrating injury. In this category, of course, are to be reckoned the operations that are attended with opening of the eyeball, in case they are followed by inflammation.

As a matter of prognosis and treatment, it is important to know that, in spite of there being a violent inflammation of the primarily diseased eye, sympathetic ophthalmia occurs very rarely in (1) suppuration of the cornea (in ulcus serpens, after acute blennorrhœa, etc.) and in its sequelæ, phthisis

corneæ or staphyloma of the cornea, and in (2) panophthalmitis and t phthisis bulbi that follows it; and it never occurs in (3) absolute glaucom

The *point of time* at which the greatest danger of the transmission the inflammation exists is when the irido-cyclitis in the injured eye is at i height. Hence sympathetic inflammation makes its appearance, in mo cases, from four to eight weeks after the injury to the first eye has tak place. (It may occur as early as four days after the injury—Cahillous cit by De Lapersonne). Later than this, when the traumatic irido-cyclitis h subsided and the eye has fallen a prey to atrophy, there need be generally fear of sympathetic inflammation, so long as the atrophic eye is free from i flammation and is not painful, either spontaneously or to the touch. The da ger for the other eye does not develop again until the atrophic eye becomes on more the seat of inflammation and of pain—an event which, to be sure, ve frequently occurs. In this way an eye which has been carried for many yea in an atrophic state without causing trouble may suddenly become the cau of a sympathetic inflammation. While, therefore, the minimum period f the development of sympathetic irido-cyclitis is but a few days, no limits c be set to the maximum period; sympathetic inflammation has been seen appear forty years and more after the injury of the first eye. An eye whic has been destroyed in consequence of injury is therefore a constant sour of danger to the other eye.

To excite sympathetic inflammation it is not necessary for the injured eye to perfectly blind. Cases occur in which the eye has retained a remnant of visual pow after the injury and the irido-cyclitis following it, and has yet given rise to sympathe inflammation. In that case it may happen that the sympathetically affected eye unde goes complete destruction, while the injured eye is still used to see with.

Can an irido-cyclitis of *non-traumatic origin* be transmitted to the other eye? V very often see irido-cyclitis develop spontaneously first in one eye, then in the other. B we must not therefore at once conclude that the inflammation has been transmitted fro one eye to the other. It may be that we have to do with a deeply seated common caus generally of constitutional nature, which makes itself felt first in one eye, then in the othe We should therefore regard an irido-cyclitis occurring in the fellow eye as sympathet only in case the inflammation in the first eye is certainly a purely local one and not depe dent on constitutional causes. This is true of two varieties besides the traumatic, i.e of the irido-cyclitis which sometimes occurs after the perforation of a corneal ulcer, and that which develops in eyes with an intra-ocular tumor. If the latter becomes necroti a violent inflammation of the inner coats of the eye results. In these two cases a genuir sympathetic inflammation of the second eye is observed, even though it is rare (cf. ca described under Fig. 314).

713. The way in which the inflammation is *transmitted* from one eye t the other is as yet unknown. Not every severe traumatic inflammation c the eye leads to sympathetic disease of the other; whether this develops c not depends upon two conditions:

1. Upon the character of the inflammation of the injured eye. To a certain exten this has been known for a long time; thus we are aware that in cases in which the injur produces ulcus serpens or panophthalmitis, sympathetic inflammation usually does nc set in. To excite this an irido-cyclitis is required. But again it is not every traumati irido-cyclitis, even when destructive, that causes sympathetic inflammation, but only a irido-cyclitis of a quite well defined sort, which is marked by a peculiar, very characte

istic anatomical condition (compare § 699). Unfortunately, we have not as yet advanced so far as to be able to conclude from the clinical picture itself whether in a given case we are dealing with this particular sort of irido-cyclitis.

2. If now this particular kind of irido-cyclitis does really develop after an injury, a possibility is then afforded that this inflammation may pass to the other eye, but such passage does not necessarily follow. For it to occur, a series of conditions must be involved, affecting the paths by which the passage takes place. What these conditions and indeed what the actual paths are we do not know. By some the transmission has been thought to take place by the ciliary nerves. But since the ciliary nerves of the two sides are nowhere in direct connection, the transmission in this case could be effected not by direct migration, but only in a reflex way. As a matter of fact it is scarcely to be doubted that sympathetic irritation is effected by way of the ciliary nerves. In fact even in slight changes occurring in one eye (for example, a foreign body in the cornea) we observe lacrimation and photophobia in the other. But that an actual inflammation with serious anatomical changes should develop in a reflex way is, in our present state of knowledge, inadmissible. Hence others, including Mackenzie, who first taught us to know sympathetic ophthalmia, have regarded the optic nerves as the medium by which bacteria or toxins are transmitted from one eye to the other. But this hypothesis is opposed by the fact that sympathetic ophthalmia does not actually begin in the optic nerve and when changes are found in the latter these decrease instead of increasing as we go backward. Furthermore, the optic nerve of the exciting eye may be completely divided and yet sympathetic ophthalmia may develop. Hence the view has prevailed that the transmission of bacteria or other agents exciting the inflammation takes place through the blood (Berlin). It has been suggested that bacteria by prolonged lodgement in the exciting eye may acquire a special affinity for the uvea so that when finally let loose in the circulation they start up a peculiar inflammation in the uvea of the other eye but affect no other organ (Gifford, O'Connor).

Another view is that sympathetic inflammation is an *anaphylactic* disease. In this view the transmission of bacteria or of disintegrated uveal tissue from the affected eye so sensitizes the other that the latter falls a prey to infection from other sources (especially to focal infection from the teeth, tonsils etc.). There are a number of arguments in favor of this view, but it cannot yet be regarded as proved.

Investigation of this subject has been frustrated by the fact that we have been able in no way to demonstrate the presence of the bacteria that are supposed to excite the sympathetic inflammation. Furthermore we have had no opportunities for examining a sympathizing eye in the first stages of the disease, nor is experimentation on animals of any help to us either. Not only does sympathetic ophthalmia fail to occur spontaneously in animals, but the attempt to produce in animals an undoubted sympathetic inflammation experimentally has also failed of success.

Brownlie and others have pointed out the analogy that sympathetic ophthalmia shows in various ways to the protozoal diseases (syphilis, malaria, trypanosomiasis). This analogy is somewhat supported by the fact that salvarsan exerts a favorable effect on the disease.

714. (*B*) **Secondary Iritis and Irido-cyclitis** are the varieties which develop through transfer of inflammation from neighboring organs to the iris and ciliary body. Inflammations of the cornea are the lesions that most frequently give rise to it; above all the suppurative keratitides, which are very frequently indeed complicated with iritis. Old scars with incarceration of the iris may through a late infection excite a dangerous irido-cyclitis (§ 609). Of the varieties of scleritis, it is the deep form that leads to inflammation of the iris and the ciliary body. The lens may cause iritis, in case of its luxation and also when after the capsule has been opened, swelling masses of lens lie upon the iris. In these cases the iritis is not usually severe.

According to Verhoeff and Lemoine, intra-ocular inflammation resulting fro opening of the lens capsule is due to anaphylaxis taking place in an eye sensitized lens protein. That the eye is thus sensitized can be proved in advance by the fact th lens protein applied to the skin produces a positive reaction. The inflammation itse which the authors term *endophthalmitis phacoanaphylactica*, is marked by infiltration the iris with plasma cells, deposits of lymphoid cells on the back of the cornea, and inf tration of the lens first with phagocytes and pus cells, afterward with tissue deriv from the iris.

Of changes in the posterior division of the eye, which may lead iritis, the chief one is detachment of the retina. When this has lasted long time, irido-cyclitis very frequently ensues, sometimes insidiously, som times with such violence as to cause great pain and ultimately atrophy the eye. Likewise very severe and terminating in atrophy, is the course ru by the irido-cyclitis that sometimes occurs with intra-ocular neoplasms with intra-ocular cysticercus.

715. Treatment of Iritis and Cyclitis.—In every case of iritis an cyclitis it is incumbent upon us, on the one hand, to combat the local symp toms (*indicatio morbi*); on the other, to remove the causes lying at the foundation (*indicatio causalis*). In those cases in which an etiologic factor is not demonstrable, we are thrown back solely upon the symptomati treatment.

716. (1) *Symptomatic Treatment.*—*Atropine* is the most importan remedy in iritis. Inasmuch as it contracts the iris, it necessarily diminishe the amount of blood in its vessels, and hence directly counteracts the hype æmia. By paralyzing the sphincter it fulfills a second indication which re quires every inflamed organ to be put at rest; the constant to-and-fro move ment of the pupil is completely arrested by atropine. The third action o atropine consists in its rupturing posterior synechiæ which already exist an in its counteracting the formation of new ones, through the dilatation of th pupil that it causes. The amount of atropine administered must be care fully regulated according to the degree of the intensity of the iritis. (Fo the precautions in its use and the danger of poisoning, see § 126). Durin the period of increase of the inflammation it is usually difficult to obtai dilatation of the pupil, because a spasm of the sphincter exists. In thi case we must instil atropine several times a day. If this does no good, w place a little granule of atropine in substance in the conjunctival sac; this i better than to instil the solution too frequently, by doing which irritation o the conjunctiva (atropine catarrh) is readily set up. By the simultaneou employment of cocaine the action of atropine may be heightened (§ 130) When the inflammation is abating, atropine is instilled just often enough t keep the pupil constantly dilated.

In cases of irido-cyclitis in which there is marked implication of th ciliary body, and also in cases of pure cyclitis, atropine is not always wel borne. For, in proportion as the iris becomes narrower and its vessels ca contain less blood, the vessels of the ciliary body are overdistended, sinc they have to take up the blood which finds no lodgment in the iris. Hence, in such cases, we must be very cautious in the use of atropine, and must sus pend it whenever we find that the pain increases after the instillation. So

also, when an irido-cyclitis is combined with elevation of tension, the atropine must be stopped and, if necessary, replaced by a miotic.

Atropine should be used as early as possible in iritis in order to forestall the formation of synechiæ and put the inflamed iris at rest without delay. Hence, whenever the diagnostic application of homatropine (§ 682) shows an imperfectly dilating pupil, atropine should be instilled immediately, and the instillations should be repeated until the pupil is thoroughly dilated.

Atropine is useless in iritis if the pupillary margin is adherent to the capsule throughout, and the iris hence cannot retract.

In violent inflammation, *moist, hot compresses* afford the best results, especially for the relief of pain. Cold compresses are generally not well borne, and are suitable only for recent cases of traumatic iritis. *Dionin* introduced into the conjunctival sac in powder or in 5-per-cent solution, acts not only to relieve the violent ciliary pain, but also often exerts a favorable influence on the progress of the disease. Of internal remedies the main ones that act to relieve pain are the *salicyl preparations* (e. g., aspirin).

Free *bloodletting* by means of six to ten leeches applied to the temple, or by a Heurteloup artificial leech, may greatly diminish the inflammatory symptoms in the bad cases; not infrequently, directly after such a bloodletting the pupil for the first time yields to the action of atropine, while before this it had remained in a constant state of spasmodic contraction. If the disease lasts a long time, the bloodletting may, if needed, be repeated once or twice.

One of the most efficient means both of combating the inflammation and of absorbing the exudate is energetic *diaphoresis* (§ 124). For absorbing the exudate we may also try *mercurial* treatment (even in non-syphilitic cases) or *subconjunctival injections* of salt, or mercury—these last, however, only when there is no marked inflammatory irritation, which might be increased by the injections.

717. *The hygienic regulations* to be observed in iritis, especially when acute, require first of all protection from the light, not only on account of the photophobia that is generally present, but also because light excites the pupil to contraction. For the latter reason the protection against light must be applied to both eyes, since with the contraction of the pupil of one eye the pupil of the other also tends to contract. The patient is kept in a moderately darkened room, or is made to wear dark goggles. This is better than a bandage, which it would be difficult to apply to both eyes. The patient should be moderate in eating, and should abstain from spirituous beverages. Care should also be taken to secure bodily rest by the avoidance of all physical exertion and in severe cases by rest in bed. The healthy eye should not be submitted to strain of any kind, i. e., reading, etc. Moreover, it is very important to keep the bowels open.

Still more in chronic iritis, when we are often baffled in our attempts to locate or successfully treat the cause of the inflammation, are we compelled to resort to hygienic measures as our chief resource. In such cases we should try to increase the patient's resisting power by proper rest, by

improving his nutrition, by making him live out of doors, etc. In some cas thyroid extract is helpful.

718. (2) *Causal Indication.*—This requires a searching *prelimina examination* to discover the cause or causes of the iritis. In making tl examination we should, when the diagnosis is not otherwise certain, dete mine the condition of the gastro-intestinal tract, take skiagrams of t teeth and the nasal sinuses, use diagnostic injections of tuberculin, ai make complement fixation tests of the blood for the spirochæta (Wasse mann test), and when it seems necessary, for streptococci, gonococci, ai other organisms.

With regard to the etiological factor, *syphilitic iritis* gives the mo favorable prognosis, since it generally yields promptly to an energet antisyphilitic treatment. The chief requisite here is promptness of actio since we are dealing with a lesion in which a few days may produce gre and lasting damage (by forming a seclusio or occlusio pupillæ). Hen salvarsan or mercury is selected, and the latter is best applied by inun tion (from two to four gm. of blue ointment being rubbed in daily) or b intramuscular injections of an aqueous solution of mercury bichloride (an albolene emulsion of mercury salicylate. The inunctions or inje tions should be kept up until the diseased eye has become perfectly fr from discoloration and then potassium iodide (up to three gm. a day) ma be used for after-treatment. In iritis due to hereditary syphilis, besides th specific antisyphilitic treatment, special value is to be attributed to measur for strengthening the organism as a whole. In a case of syphilitic iritis w must not forget that there may be other causal factors besides the syphil that will require treatment (cf. § 703).

In gonorrhœal and other forms of *focal* iritis causal treatment is partic ularly indicated. In the former, treatment of the urethral gonorrhœa shoul be carefully carried out. In many cases gonococcus vaccine is used wit good results. In focal iritis due to disease of the teeth, tonsils, or sinuses removal of the diseased focus is the sine qua non of treatment, followed if necessary, by vaccine treatment. The latter is made, if possible, with a autogenous vaccine. The strength of the gonococcus and other vaccin injections and the interval between them are regulated by the local reactio at the site of injection, and the focal reaction in the eye. In gonorrhœa and other forms of focal iritis the salicylates, aspirin or the oil of winter green are often specially helpful. In iritis associated with a gastro-intes tinal disturbance the proper dietetic and eliminative measures must b faithfully carried out.

In iritis due to *metabolic disorders* treatment of the underlying caus (gout, diabetes) must be instituted. In stubborn cases of iritis adminis tration of the thyroid extract sometimes does good service, even thoug hypothyroidism is only an indirect cause of the iritis (see § 706).

For *iritis tuberculosa* the main thing is a correct and sufficiently lon continued treatment with tuberculin injections (see § 140) and, added t this, general hygienic measures for invigorating the patient.

In *iritis traumatica* the causal factor is first of all to be eliminated, i

case it still continues to act. Foreign bodies are to be removed from the iris; portions of the iris that are much contused or are incarcerated are to be excised; a swelling or luxated lens causing iritis must be removed from the eye. To combat the inflammation of the iris, iced compresses are employed in very recent cases in addition to the atropine. With respect to those traumatic iritides which follow an operation, the most important part belongs to prophylaxis. This consists in strict antisepsis during the operation; and, as a matter of fact, since this has been employed such iritides have become much more rare. In traumatic (including post-operative) iritis, mercury, either by inunction or in the form of calomel, kept up for a number of days, and the salicylates in doses of three to six gm. a day, are helpful (De Schweinitz) (cf. also § 721).

719. *Operative Procedures.*—These are for the most part indicated more in the sequelæ of iritis than in recent inflammation. *Paracentesis* is performed whenever increase of tension sets in. It may also be tried in cases of long-persisting inflammation which will not yield to other remedies. As the aqueous escapes, the deposits upon the cornea are often washed out with it, and their escape can be facilitated by rubbing the cornea; but the removal of such deposits is not the real aim of the paracentesis, the object being rather to improve metabolic conditions in the eye.

Iridectomy is not performed while inflammation still exists, save in very exceptional cases, since, if we perform it then we should have reason to fear that the newly made pupil would be closed up again owing to a continuance of the exudatave process. Hence we undertake an iridectomy only when we are compelled to do so because of the development of an increase of tension, or as a last resort in order to put an end to the iritis when all other means have failed. Otherwise we wait until the inflammation has run its course, and then perform an iridectomy, either because a seclusio pupillæ has developed, or as a prophylactic in relapsing iritis to prevent further recurrences. And, in fact, in many cases, an end is put once and for all to recurrences by this means; in other cases, the subsequent recurrences are, at all events, rendered lighter.

In cases of chronic irido-chorioiditis, iridectomy is not only mechanically efficient by removing the seclusio pupillæ, but it also has a favorable influence upon the entire nutritive condition of the eye. The vitreous clears up and the sight improves, often for quite a long time. If an operation is done upon eyes which have already begun to grow softer—that is, are on the road to atrophy—the eye in favorable cases fills out again and the ocular tension becomes normal.

720. *Enucleation* in general comes up for consideration only in those cases in which incurable blindness has already set in. It is then indicated: (1) If the eye is permanently inflamed and painful; (2) if we suspect that there is a neoplasm in the inflamed eye (§ 764); (3) in case of sympathetic irritation, this being surely and permanently relieved by the enucleation; (4) if sympathetic inflammation of the other eye threatens to occur. In the last case, enucleation has this great prophylactic value that it prevents with almost absolute certainty the outbreak of sympathetic inflammation. But if we wait to perform enucleation until the first signs of sympathetic disease

show themselves, we are generally too late. Hence, to advise enucleation a the right moment is one of the most important tasks that the physician ha to perform. To do this properly we must bear in mind the fact that th danger of sympathetic inflammation, is almost confined to the traumati cases. If it is a case of recent inflammation, enucleation is indicated as soo as we see that blinding of the injured eye is inevitable. We recognize that thi is the case by the increasing deficiency in the perception of light (this espec ially in the plastic cases; in purulent conditions, this not always an abso lute guide—see § 754). If the traumatic inflammation has already run it course, and a greater or less degree of atrophy of the eyeball has supervened enucleation is still indicated if the eye is sensitive to pressure or if it get inflamed often. It is only when the eyeball is entirely and permanently fre from irritation that the demand for enucleation is not imperative. But th patient ought to be warned that he should report immediately for enucle ation, if pain or inflammation happens to set in anew, and should be kep under periodic observation so that we can detect at once any evidences o inflammation or any of the danger signs of sympathetic disease (§ 711).

In phthisis bulbi enucleation is not generally required. Phthisis repre sents the outcome of panophthalmitis, of which, as experience shows, sym pathetic inflammation is only very exceptionally a sequel. Moreover phthisical eyes are usually free from irritation.

When sympathetic ophthalmia has already broken out, the effect o enucleation is uncertain. In the lighter cases it appears to exert a favorabl influence upon the course of the sympathetic inflammation; in severe cases on the contrary, it is often of no avail.

Although *enucleation* generally affords a sure safeguard against sympathetic inflam mation of the other eye, nevertheless a series of cases is known in which in spite o enucleation, inflammation has subsequently made its appearance. In every instance it has set in within a short time—from a few days to a few weeks—after the enucleation The longest interval so far observed is fifty-three days (in a case of Stephenson's cited b De Schweinitz). Yet even when this does happen, enucleation does not fail to exert a fav orable effect, since in the great majority of these cases the sympathetic inflammation run an unusually favorable course, probably because the removal of the first eye prevent the constant emission from it of new impulses for the production of inflammation.

721. *Sympathetic inflammation* itself is to be treated according to gen eral rules. Salvarsan has been used in several cases with distinct success Of good effect also is treatment with mercury and with salicyl preparation in the largest possible doses. Treatment, to be successful, must be persis tent and prolonged, and varied according to conditions. The patient, there fore, should be kept under constant observation. Atropine may have to b used for many months at a time. This may be combined with pilocarpin injections, and potassium iodide may be combined with the mercur (Burnham).

If the anaphylactic theory of sympathetic ophthalmia is correct, the latter may b prevented and its progress even may be checked by the removal of any form of disease i the teeth, tonsils, or other parts, which might act as an accessory agent in setting u inflammation in the eye if already sensitized. There seem to be some facts indicatin that this is indeed the case.

Operations in a case of sympathetic ophthalmia generally give a bad result, since they start up the inflammation again, so that the newly formed pupil is once more closed by fresh exudate. Hence operations are done only when it is absolutely requisite (e. g., when done on account of increase of tension); other operations, such as, for example, an iridectomy for optical purposes, are put off as long as possible, preferably for years.

722. Treatment of the Sequelæ—Isolated posterior synechiæ can often be ruptured by the employment of atropine either by itself or in combination with cocaine. Here it is not so much a long continued action as a very energetic one that is required, and this is most certainly secured by placing atropine in substance in the conjunctival sac after preliminary cocainization of the eye. Some instil a solution of atropine every ten minutes during an hour. Others use strong solutions (four per cent) and employ adrenaline as well as cocaine to promote absorption of the atropine. In all such attempts to secure a thoroughgoing effect, the precautions given in § 126 must be observed.

It is frequently possible to rupture synechiæ which are narrow and drawn out into a point, while broad synechiæ (such as occur after syphilitic or sympathetic iritis) withstand all attempts.

Annular posterior synechia (seclusio pupillæ) demands iridectomy unconditionally, the object being to restore the communication between the anterior and posterior chambers. The operation is often difficult on account of the shallowness of the anterior chamber (due to protrusion of the iris), and also on account of the atrophy of the iris. Accordingly, we must often be contented if we succeed in making a small opening in the iris. In hump-like protrusion of the iris this is best accomplished by transfixion (§ 954). Then the anterior chamber, in consequence of the restoration of the connection between the two chambers, regains its normal depth, so that an iridectomy can be performed later under more favorable conditions.

Total posterior synechia also requires iridectomy, which, however, is often void of result, as on account of the adhesion between the surfaces of the iris and the lens it is frequently impossible to excise a sufficiently large piece of the iris, or because the pigment layer of the iris, which has grown fast to the lens, remains attached to it. In such cases the only thing to be done is to remove the lens, too, even if it is still transparent (Wenzel's extraction, see § 970). If the lens is shrunken or absent, iridotomy is indicated (see § 954).

In *atrophy of the eyeball* enucleation is indicated subject to the conditions given in § 720. Sometimes enucleation of an atrophic eyeball is desired simply for cosmetic reasons, when the eye is disfiguring and, moreover, does not tolerate the wearing of an artificial eye over it.

If, after an iritis, one or two posterior synechiæ are left which do not rupture when atropine is energetically employed, we abstain from further treatment, since they generally do no harm to the eye. When, however, an annular synechia is present, we ought not to let it stay, but must make an *iridectomy*. This is also indicated in those cases in which the seclusion of the pupil is not yet indeed complete, but is on the verge of being so, only one small spot of the pupillary margin being free from it. For if we are dealing with

a chronic irido-cyclitis, we can count upon this small spot's soon becoming adherent to and in that case it is better not to wait for the seclusion of the pupil to become complete It is particularly advisable not to do so if the patient lives far from the physician, an might perhaps let the proper moment for the iridectomy pass by.

In seclusio pupillæ the iridectomy is made upward. If occlusio pupillæ is simu taneously present, we should, according to the rules laid down for an optical iridectom in § 951, make the coloboma to the inner side and below. But even in these cases it advisable to make the iridectomy upward, because it is so often the case that the len afterward becomes opaque, and in that case a coloboma situated above will be of advan tage in performing the extraction that will be required later.

II. Injuries of the Iris

723. In addition to what has been already said in the previous section in regard to injuries of the iris and their consequences, the following specia varieties of injury, which are most fre quently observed after contusion of th eye, may be mentioned:

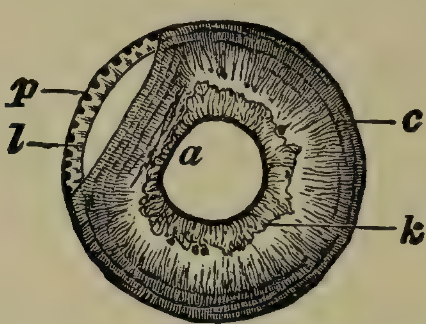


Fig. 318.—Iridodialysis. Magnified 2 × 1.

The pupillary margin at *a* is sloped off so as to form a straight line. At a point corresponding to this spot the iris is separated from the ciliary body and is narrower and somewhat wrinkled. In the interval between the iris and the margin of the cornea are seen the margin of the lens, *l*, and the apices of the ciliary processes, *p;* the fine radiating striation between the two structures represents the zonula of Zinn. *k*, circulus iridis minor; *c*, contraction furrows.

(1) **Iridodialysis.**[1]—By this is des ignated the separation of the iris fron the ciliary body. We then find on on side, at the ciliary margin of the iris a black crescent which is formed by th separation of the iris from its insertio at this spot, so that we can there look into the interior of the eye (Fig. 318) When the separation is pretty consider able, the edge of the lens (*l*), the ciliar processes (*p*), and the fibres of the zon ula of Zinn stretching between the tw can be recognized by means of lateral illumination in the gap that is thu produced. The pupil has lost its round form, owing to the fact that th pupillary margin toward the side of the iridodialysis has shortened s as to occupy the chord of an arc instead of the arc itself (Fig. 318, *a*) The cause of this inward displacement of the pupillary margin lies in the fac that the separated portion of the iris is stretched in a straight line by th contraction of the sphincter. By this means it is withdrawn from its inser tion in the ciliary body, and its reunion with the latter is rendered foreve impossible. The sight is but little affected by iridodialysis; the only thing being that, if the eye is not accurately focused, monocular diplopia may occur, owing to the formation upon the retina of an image both by mean of the pupil and also of the peripheral opening (see § 171).

Iridodialysis may occur to any extent, from a scarcely perceptibl tear to complete separation of the iris from its ciliary attachment (*total* o *circular iridodialysis*). In the latter case the iris rolls up into a ball which sinks to the bottom of the chamber, and which by the next day has shrunken into an inconspicuous little gray mass. If a rupture of the sclera has been at the same time produced by the injury, the separated iris may be expelled

[1] From ἶρις, iris, and διάλυσις, separation.

altogether from the eye through the scleral wound. In both cases complete absence of the iris (*irideremia*[2] or *aniridia*[3] *traumatica*) is produced. Again, a partial expulsion of the iris from the eye may occur, especially in cases of rupture of the sclera, and thus a *traumatic coloboma* is produced.

Iridodialysis is sometimes unintentionally produced in operations upon the iris (see § 948), and is also produced in a non-traumatic way, when neoplasms of the ciliary body grow out into the anterior chamber so as gradually to push the iris away from its insertion (Figs. 317 and 325), and is also produced by traction on the iris (§ 690).

724. (2) **Radiating lacerations** which start from the pupil. These may extend to the ciliary margin, so that the pupil at the site of laceration appears to be prolonged to the margin of the cornea in the form of a pointed Gothic arch (Fig. 319). Such large lacerations, however, are rare. Generally the pupillary margin is simply torn into a little, and the laceration

FIG. 319.

FIG. 320.

FIG. 319.—LARGE RADIAL LACERATIONS OF THE IRIS. Above lies a large laceration, whose edges gape widely and whose upper extremity, which does not reach to the ciliary border, is rounded off. The smaller laceration, which lies close to the larger one on the inner side of the pupil, retains its original acute-angled shape. The pupil is dilated, and for obvious reasons lacks the black rim of retinal pigment in that part of its circumference occupied by the lacerations.

FIG. 320.—SMALL RADIAL RUPTURE OF THE MARGIN OF THE PUPIL. Magnified 2 × 1. At the outside and above, the margin of the pupil is regular and is surrounded by the black line of the retinal pigment. At the inner side and below, this line is wanting; the margin of the pupil here is irregular, shows small indentations, and is retracted along its whole extent so that the pupil is dilated down and in.

gapes so slightly that it is discovered only upon careful examination, especially with the aid of a magnifying glass (Fig. 320). Such small lacerations are the most frequent cause of the dilatation of the pupil (*mydriasis traumatica*) which develops after contusions and which is due to the weakening or paralysis of the sphincter due to the laceration. In such cases, for the most part, a mydriasis of moderate degree remains permanently.

The ciliary muscle, too, may be paralyzed by a contusion (§ 727).

725. (3) **Inversion of the Iris** (Ammon) consists in its being turned back so as to lie upon the surface of the ciliary body (Fig. 322, *o*). The iris then can not be seen occupying its normal place, and looks as though it were absent altogether. Total inversion of the iris is very rare. Partial dislocation backward is more frequently observed. At the spot where this takes place the iris seems to be wanting, and a coloboma appears to be present just as if an iridectomy had been made.

To *account for* the above-described traumatic changes in the iris, two factors have to be taken into consideration. The first is the flattening which the cornea undergoes through the contusion, and because of which its circumference and consequently also the circle of insertion of the iris become larger. If this enlargement takes place suddenly, the iris can not adapt itself to it and tears away in places from it insertion, so that iridodia-

[2] From ἶρις and ἐρημία, loneliness, want. [3] From ἀ, privative, and ἶρις.

lysis is produced (Arlt). The second factor consists in the circumstance that the bl which strikes the cornea and flattens it out also pushes the aqueous backward. T latter tends to recede and pushes against the posterior wall of the anterior chamber, whi in the area of the pupil is formed by the lens and in the rest of its extent by the iris. T latter, when pushed backward, finds a support in the lens except at the marginal porti of the iris which lies outside of the margin of the lens. Here the posterior chamber deepest and is bounded posteriorly only by the feeble zonula of Zinn. The periphery the iris, therefore, forms the most yielding spot in the posterior wall of the anterior cha ber and the one which is the first to give way before the pressure of the receding aqueo Hence the iris is bulged out backward by the aqueous so as to form a sac extending far as the zonula, or, if this ruptures, extending even into the vitreous (Fig. 322, *u*). T direct consequences of this dislocati of the iris are threefold: marked stretc ing of the fibres of the iris in a radi direction; dilatation of the pupil; an finally, in extreme cases, rupture of t zonula. The first factor may result iridodialysis. The sudden dilatation the pupil may cause radiating lacer tions of the sphincter and consequent paralysis of the latter. The rupture the zonula causes tremulousness, su luxation, or luxation of the lens. If t backward dislocation of the iris and t consequent recession of its pupilla margin are of a sufficiently high degr to cause the latter to slip back over t edge of the lens, the lens being no long held in place by the zonula will sl through the pupil into the anteri chamber, where it is then held captiv by the iris, which again contracts behin it (luxation of lens into the anteri chamber). Finally, the sac-like intr sion of the periphery of the iris may b so extensive that the iris at one spot completely reversed and points straigh backward (Fig. 322, *o*) and inversion c the iris is produced (Forster).

Fig. 321.—Inversion of the Iris. Magnified 11 × 1.

Fourteen days previous a piece of wood had flown into the patient's eye while he was splitting wood. The eye showed a rupture of the sclera running close to the limbus. The iris had been turned over backward and made to lie on the surface of the ciliary body. It is retained in this position by a slight exudation membrane, *a*; this is attached to a projection of iris, produced by the bending of the pupillary portion of the iris so as to form an acute angle with the ciliary portion. Near the free margin of the iris the pupillary portion makes another turn backward, and so does the sphincter pupillæ, which is seen in cross section at *s*.

A small sharp-edged *hole* may be made in the iris by foreign bodies, particularl fragments of metal, entering the eye. If such a hole is not blocked by blood or exuda tion it never grows together but remains unchanged all through life. Upon this fac depends the possibility of effecting a permanent communication between the anterio and posterior chambers in seclusio pupillæ by making a small hole in the iris (transfixion § 954). For, provided it is not inflamed, the iris has no tendency to cover over raw sur faces by cicatrization, as for example, the cornea does. Thus in the iris stump which i produced by an iridectomy we find, even many years later, the tissue as smoothly divide and as bare as it was directly after the operation (Fig. 260).

726. Injuries of the Ciliary Body.—After injuries, especially concussions with rupt ure of the sclera, there is often produced *laceration of the ciliary body* at its attachment o even an entire separation of it from the sclera (*cyclodialysis traumatica*). This sort o injury is found quite often in anatomical specimens, and clinically it has been diagnos ticated by gonioscopy (§ 390.) By laceration of this sort the anterior chamber is place in direct communication with the space between the sclera on one hand and the ciliar body and chorioid on the other (perichorioidal space). It thus becomes possible for th aqueous to enter this space and detach the chorioid from the sclera (see § 760).

727. Injuries of the iris are generally accompanied by hæmorrhage into the anterior chamber. The blood which arises from the ruptured vessels of the iris sinks rapidly to the bottom of the chamber (*hyphæma*), and for the most part disappears by resorption within a few days. Then and not before, are we able to investigate completely the damage which the iris has suffered from the injury, and we find perhaps an iridodialysis or radiating lacerations. But often even then it is impossible to discover a solution of continuity in the iris, and so the source of the bleeding remains unknown. Iritis is generally not to be apprehended after injuries of the iris not associated with the perforation of the membranes of the eye.

Injuries of the cilry body are accompanied by *hæmorrhages* into its substance and by *paresis of accommodation.* In cyclodialysis, the ciliary body is approximated to the lens, so that the zonula is relaxed and the lens being released from compression distends so that the eye becomes more refractive. In this fashion a spasm of the ciliary muscle is simulated by a process purely mechanical (*false spasm of accommodation*). The condition is generally transient.

728. *Treatment.*—If the symptoms of irritation after the injury are particularly marked, we apply iced compresses for several days; in other cases it is sufficient to keep the injured eye under a bandage and to insure quiet on the part of the patient, secured, if need be, by rest in bed. If an iridodialysis can be made out, we instil atropine in order that the contracting sphincter shall not draw the iris farther away from its point of attachment; in radiating lacerations, however, atropine is contraindicated, because it would make the lacerations gape still more. For the reabsorption of large quantities of blood diaphoretic treatment may be initiated. With reference to the treatment of perforating injuries, see § 860.

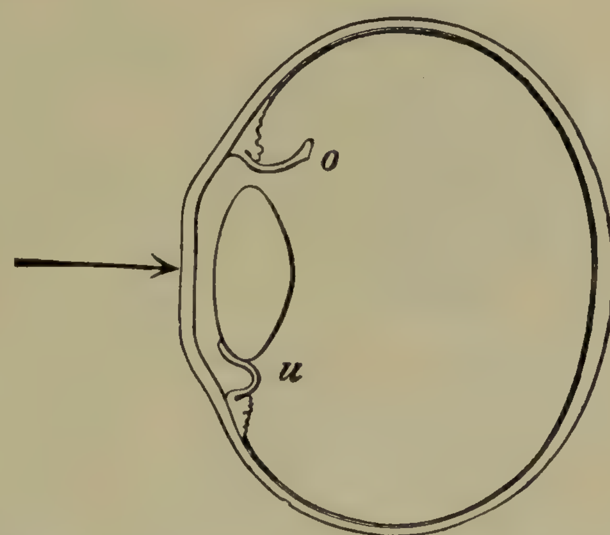


Fig. 322.—Inversion of the Iris, Schematic. The cornea is flattened, and consequently the aqueous is pushed backward by the force which acts upon the centre of the cornea. As a result of this, the iris in its lower part, *u*, bulges out in the form of a sac, but at its upper part, o, is completely reversed, so as to point straight backward.

In iridodialysis, the detached iris may be drawn up into a small incision into the limbus and fixed there either with or without sutures (Bulson).

III. Tuberculosis of the Iris.

729. **Tuberculosis of the Iris** is observed in children and young people. It occurs as disseminated (miliary) tuberculosis and as conglobated (solitary) tubercle—i. e., either in the form of small nodules or as a larger growth resembling a neoplasm. Tuberculosis may also cause an *iritis* or a *chronic uveitis* in which tubercle nodules are not demonstrable and yet the nature of the case is demonstrable by the diagnostic and therapeutic action of tuberculin (see §§ 707, 708).

Disseminated tuberculosis of the iris occurs in man under the form of an iritis, which presents as its characteristic feature small, gray, transparent nodules. These keep changing slowly, some disappearing while others are being newly formed. Recovery may finally take place; not infrequently, however, atrophy of the eyeball occurs in consequence of plastic irido-cyclitis. The disease often develops in both eyes. Removal of the individual nodules through excision by an iridectomy of the portion of iris bearing them is generally useless, since new nodules form afterward in other parts of the iris. Excision, moreover, is not free from danger, since it opens an avenue for the dissemination of the tuberculosis (Gamble).

The *solitary tubercle*, as so far observed, is confined to one eye. It either develops simultaneously with the miliary nodules, or, more frequently, without them and without any symptoms of iritis, so that it resembles a neoplasm; indeed, it was at first described as such by Von Graefe, under the name of *granuloma*, because Virchow, who made the anatomical examination of the tumor, described it as granulation tissue. Its subsequent course seems at first to confirm the diagnosis of a neoplasm, since the tumor keeps steadily enlarging, and finally, perforating the cornea near its margin, pushes out in the form of an exuberant growth. But then, instead of a larger tumor developing from this, which keeps growing on indefinitely, the growth breaks down, so that ultimately nothing is left of the eyeball but an atrophic stump. Haab was the first to bring proof of the fact that these tumors, formerly designated as granulomata, are tubercles. As far as treatment is concerned, however, this mistake in diagnosis would be of little significance, since enucleation is indicated alike in the case of a neoplasm and in that of a granuloma. The eye which harbors a granuloma is, in fact, lost for purposes of vision, and may become the source of a general tuberculous infection.

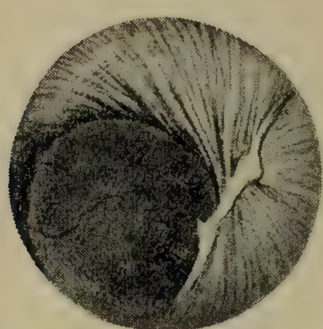


FIG. 323.—CYST OF THE IRIS. The cyst occupies the inner lower portion of the anterior chamber. It is round, gray, and translucent, and displays on its surface delicate, radiating fibres, which belong to the interior layers of the iris. Its upper and outer margin covers the somewhat distorted pupil, which appears as a dark oval spot showing through the cyst at a point about corresponding to the centre of the cornea. Bordering the cyst on the outer side is a vertical, white, linear cicatrix in the cornea, originating from a perforating injury that was caused by a kick from a hoof thirty years before. From all sides the iris is drawn into the cicatrix, and, owing to the tension thus produced, the part of the iris that runs upward shows a partial dehiscence of its fibres.

Solitary tuberculous tumors have also been observed in the ciliary body.

Experimental tuberculosis of the iris can be produced in animals by the introduction of aseptic tuberculous matter into the anterior chamber (Cohnheim). Fragments of tuberculous tissue, when introduced into the anterior chamber, excite there a slight irritation, which, after a few days, disappears again. As the fragments of tissue themselves are also rapidly absorbed, the eye soon appears perfectly normal again, as if the inoculation had remained without result. But in twenty or thirty days afterward the eye begins to become red once more, and the signs of iritis make their appearance; at the same time small gray nodules are noticed in the iris. These increase in number, become confluent, afterward fill the anterior chamber, and finally break their way through to the outside. Generally the animal experimented on perishes afterward from the development of general tuberculosis due to infection starting from the eye. Tuberculous iritis is employed as a means of demonstrating the tuberculous nature of excised pieces of tissue, for which purpose we introduce the latter into the anterior chamber of a rabbit's eye, and see if tuberculous nodules develop after the usual period in the iris.

The *diagnosis* of tuberculosis of the iris is made with the injection of tuberculin. A general or local reaction produced by the injection indicates tuberculosis, but a sure proof that the iris is affected is afforded only by the development of a focal reaction (increased hyperæmia of the iris or the development in it of new nodules).

Tuberculosis of the iris *originates* by secondary infection from a tuberculous focus elsewhere in the body.

As a matter of fact, in most cases of tuberculosis of the iris, signs of tuberculous disease in other organs (lungs, lymphatic glands, bones, etc.) are also found. In many cases, to be sure, the patients appear to have been perfectly healthy up to the time of their eye trouble, but even in these cases a primary tuberculous focus (e.g., caseous bronchial glands), although not demonstrable clinically, must be assumed to exist—in fact, the iris on account of its protected situation, cannot be infected by tubercle bacilli from without, as, for example, the conjunctiva can, in which primary tuberculosis is not so very rare.

In the milder cases recovery may take place, while in the severe cases the eye is usually lost. The *treatment* consists, on the one hand, in combating the local symptoms of iritis, and, on the other, in the general treatment of the tuberculosis by hygienic means, internal remedies, and injections of tuberculin. But if the disease, nevertheless, keeps on and blindness is impending, it is better to remove the eye by enucleation, that it may not be the source of a further extension of the tuberculosis.

730. Nodular Conditions Resembling Tuberculosis.—Besides occurring in tuberculosis, iritis with the formation of nodules also occurs in leucæmia and pseudoleucæmia. Under the name of *ophthalmia nodosa* is denoted the affection resembling tuberculosis produced by the entry of caterpillar hairs into the conjunctival sac. Some weeks or months later, nodules develop with violent inflammatory symptoms in the iris and often also in the conjunctiva and cornea. Examination of the excised nodules shows that they contain caterpillar hairs (Pagenstecher and others).

IV. Tumors of the Iris and Ciliary Body

731. Cysts of the Iris.—Very rarely, serous cysts develop within the stroma of the iris.

They appear under the form of grayish, transparent vesicles which are filled with a clear substance and whose anterior wall usually shows still one or two fibres of rarefied iris tissue, and also some pigment. They grow very gradually until they have reached the posterior surface of the cornea when they flatten out, while the cornea at the point of apposition becomes cloudy, as it always does when in contact with foreign tissue. Meanwhile the cyst has already reached the pupillary margin of the iris and pushes it forward into the pupil, so that the latter becomes kidney-shaped, and afterward even reduced to a slit. Moreover, the cyst keeps extending backward, too, and thus causes tilting and afterward opacity of the lens. All these factors give rise to disturbance of vision, which to be sure, is frequently unnoticed by the patient, whose vision has already been impaired by the injury. Later when the cyst has filled a large part of the anterior chamber increase of tension sets in, and the eye becomes entirely blind.

Sometimes cysts grow out into the *posterior chamber*, pushing the pigment layer of the iris before them, so that they appear as brownish tumors jutting out behind the iris (Salzmann).

Cases of congenital serous cysts have been observed; also cases of *pearl cysts*, which are distinguished from the serous cysts by their contents, which are pultaceous, tallowy, or like gruel; in rare cases hairs are also found in them. Some congenital cysts have been observed which spring from the pars ciliaris retinæ and contain vitreous. They are therefore *vitreous chamber cysts*, although they project round the edge of the pupil into the anterior chamber and may even become detached and float free in the latter (Spicer).

The walls of iris cysts are formed by iris tissue, while their inner surface is lined with epithelium, which secretes the serous contents of the cyst; in the pearl cysts the pultaceous contents are formed by the epithelial cells which are constantly thrown off from the inner surface and undergo fatty disintegration.

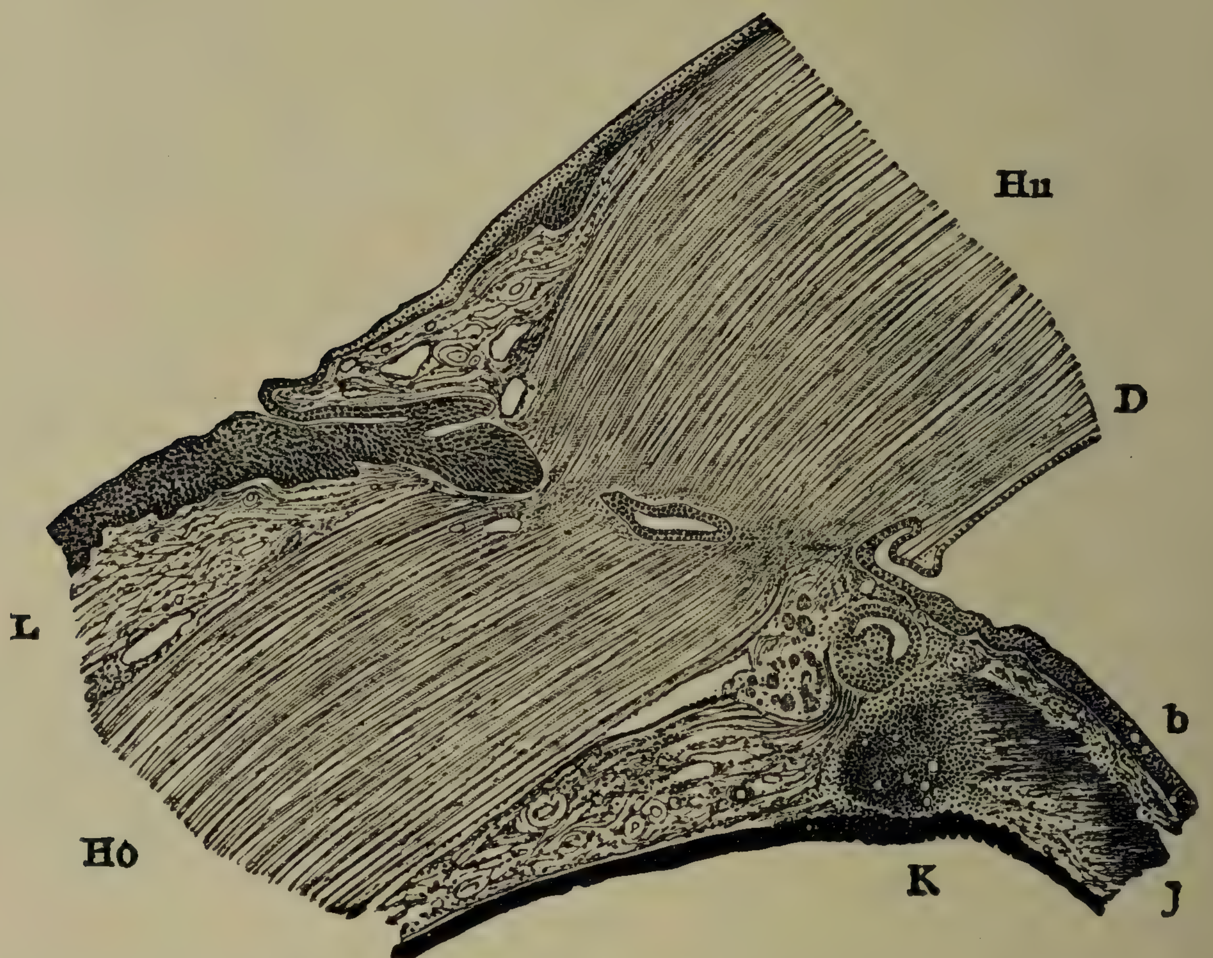


Fig. 324.—Migration of Epithelium into the Interior of the Eye. Magnified 30 × 1.

After an operation by flap incision for a senile cataract a good healing took place at first but after two years increase of tension set in. The eye became blind and had to be enucleated because of the constant pain. The figure shows the site of the scar which lies altogether in the corneal tissue, but, nevertheless, is covered by the limbus, *L*. *Ho* is the upper or peripheral, and *Hu* the lower or central lip of the wound. The epithelium of the limbus grows over both lips of the wound down into the deeper parts and hence forms a hollow tube whose lumen, however, in many places has disappeared owing to the free surfaces being in apposition. Since the epithelial tube does not extend in a straight line in its passage to the deeper parts, the section has not traversed it in its whole length but shows only separate portions of it. Thus we see the epithelial indipping on the surface; again, in the middle of the scar a portion of the epithelial tube; and lastly, far down, the opening of the tube into the anterior chamber at the spot where Descemet's membrane, *D*, was cut through. The epithelium winds over this cut edge and lines the posterior surface of Descemet's membrane in a single layer of cells. On the other side, the epithelium lines the surface of the iris, *J*, forming here a stratum consisting of several layers. The light spots, *b*, in the epithelium are produced by mucous degeneration of individual cells (beaker cells). Since the epithelium lines the anterior and posterior walls of the anterior chamber it forms an *anterior-chamber cyst* (Wintersteiner).

The upper edge of the cut in Descemet's membrane is turned somewhat forward and is separated by scar tissue from the lower edge. This scar tissue, on the one hand, juts somewhat into the wound canal and, on the other hand, it attaches the iris to the posterior end of the wound canal. In the iris we can recognize the muscular fibres of the sphincter pupillæ (at *J*), and behind this a nodular inflammatory infiltration, *K*. Between the latter and the scar tissue lies an islet of epithelium inclosing a small cavity. This epithelium is likewise derived from the epithelial tube which had entered the wound canal, but the connecting part between the latter and the epithelial vesicle has not been traversed by the section and hence this vesicle appears here like an islet of epithelium. The latter constitutes the first beginning of an *iris cyst*. If the eye had not been enucleated the cavity of the vesicle would probably have become progressively larger, so that the vesicle would have penetrated further and further into the tissue of the iris. The iris would thus have been divided into an anterior and a posterior layer of tissue which would have formed the anterior and posterior walls of the cyst. In time perhaps the epithelial tube lying within the corneal scar might have disappeared by a process of atrophy, so that the cyst of the iris would have appeared like an independent epithelial structure.

We have as yet no certain explanation for the *causation* of iris cysts except for the most ordinary kind, namely, the traumatic cysts which are the result of penetrating wounds of the eyeball. Normally there are nowhere in the iris either glands or epithelium, so that retention cysts are not to be thought of. The epithelium must have gotten into the iris from the outside. If in the formation of an anterior chamber cyst (§ 635) the iris abuts on the posterior orifice of the wound in the cornea, the epithelium as it grows

into the deeper part gets into it (Fig. 324); it pushes the layers of the iris further and further apart and becomes developed into an *iris cyst* (Stölting).

The *treatment* of cysts of the iris consists in their excision. An incision is made at the margin of the cornea, a forceps introduced, and the cyst is drawn out and excised. Cysts have also been destroyed by electrolysis.

Not to be confounded with the cysts of the iris are the *cysticercus vesicles*, which in very rare cases are observed in the anterior chamber. They either lie free in the latter or are attached to the anterior surface of the iris.

732. (2) **Melanomata.**—These are found under two different forms: The first consists in a blackish tumor which grows out from the stroma of the iris into the anterior chamber and arises from the proliferation of the

FIG. 325.—MELANO-SARCOMA OF THE CHORIOID AND CILIARY BODY. HORIZONTAL SECTION THROUGH THE LEFT EYEBALL OF A THIRTY-EIGHT-YEAR-OLD WOMAN. Magnified 2 × 1.

The tumor starts from the most anterior portions of the chorioid, *n*, and forms a hemisphere extending into the interior of the eye. The bright spots and striæ in the tumor represent cross sections of the numerous broad and thin-walled blood-vessels. At its anterior border the new growth, which has perforated the root of the iris, has pushed its way into the anterior chamber, where it was visible in the living eye as a brownish mass filling the sinus of the chamber. The iris is detached from its insertion by the tumor (iridodialysis). Behind the iris the sarcoma bulges forward nearly to the axis of the eye, the nasal portion of the lens having disappeared by erosion, and so given way to the advancing tumor. The lens has consequently suffered but an inconsiderable displacement to the temporal side—enough, nevertheless, to make its edge abut against the apices of the ciliary processes there and flatten them out somewhat. The lens is transparent, and the lens capsule uninjured. The retina is adherent to the surface of the new growth, but elsewhere is detached. There was no detachment, however, in the living eye, this condition having been caused by the shrinking of the vitreous in the hardening fluid. Before enucleation the eye had normal tension and a visual acuity of $\frac{1}{10}$.

chromatophores of the iris. The second kind of melanoma has its seat at the pupillary margin of the iris. It develops from the cells of the retinal pigment layer at the spot where it is reflected upon the anterior surface of the iris at the edge of the pupil. Here small blackish-brown nodules develop which project into the pupil.[4] Sometimes, in consequence of the alternating movements of the pupil, they become separated from the pupillary margin and then lie free in the anterior chamber.

A feature common to both kinds of melanoma is that they are benign tumors which reach only a certain size. Nevertheless, cases are known in which pigmented sarcomata have afterward developed from melanomata of the first kind.

733. (3) **Sarcomata.**—Sarcomata of the *iris* are for the most part pigmented, brown tumors, which grow at first slowly, afterward more rapidly,

[4] Such pigmentary outgrowths occur normally and greatly developed in the iris of the horse.

until they fill the anterior chamber, and finally, breaking through the envelopes of the eye, extend their growth exteriorly.

Sarcomata of the *ciliary body* remain for a long time unnoticed, since they are covered by the iris. It is not until they have reached a certain size that they are seen as a brown projection behind the iris.

With regard to their subsequent course, sarcomata of the iris and the ciliary body resemble those which spring from the chorioid, to which reference must be made for particulars (§§ 763-766).

Apparently originating in the ciliary body but really in the anterior part of the chorioid, are the tumors which pushing their way forward produce an iridodialysis and thus enter the angle of the anterior chamber (Fig. 325). Sarcomata of the ciliary body itself are rare and when they grow they push the iris forward against the cornea. In an annular sarcoma of the ciliary body no tumor is visible but only, when the iris is also involved, a thickening of the latter. As at the same time there is an increase of tension it is hard to distinguish these cases from those of an ordinary glaucoma.

The only treatment for these tumors is radical removal, which should be performed as early as possible. Very small sarcomata of the iris can be removed by iridectomy, those parts of the iris which bear the nodules of the tumor being excised. Larger sarcomata of the iris, and also sarcomata of the ciliary body, require the enucleation of the eye without delay.

734. Differential Diagnosis of Iris Tumors.—A *non-pigmented* nodular tumor in the iris may be a syphilitic growth (papule or gumma), a solitary tubercle, an unpigmented sarcoma, or a granulation tumor which has formed about a foreign body imbedded in the iris. The distinguishing marks are as follows:

1. The granulation tumors contain most vessels and are hence usually reddish. The vascularity of the sarcomata varies, but is often also quite considerable; the syphilitic growths have fewer, the tuberculous masses scarcely any vessels passing through them. In the case of the tubercles, small gray tuberculous nodules of characteristic appearance are sometimes found in the neighborhood of the large tumor.
2. Papules of the iris are generally situated at its pupillary and ciliary margins, while other tumors may take their origin from any point whatever of the surface of the iris.
3. With syphilitic and tuberculous tumors, iritis appears earlier than with sarcomata.
4. Tubercle is found, as a rule, only in people under twenty, while both the other kinds of tumors usually occur after that age.
5. Particular importance must be attached to the general examination of the patient, with the purpose of determining whether there is any evidence pointing to the presence of a foreign body in the iris, or symptoms of syphilis of tuberculosis are found in other organs, and for this purpose the diagnostic injection of tuberculin and the Wassermann test may be employed as an aid. In doubtful cases it is justifiable to initiate an energetic mercurial treatment, from the result of which a conclusion may be drawn as to the nature of the tumor.

Among the *pigmented* tumors, pigmented sarcomata and melanomata (of the first variety) resemble each other exceedingly. They can be distinguished with certainty only by determining, from the previous history or from observation, whether a process of growth is going on or not.

As tumors of very rare occurrence may be mentioned: Vascular tumors (Mooren, Schirmer); myomata (Lagrange), myo-sarcomata (De Wecker and Iwanoff, Dreschfeld, Deutschmann), and endotheliomata (Franke, Wintersteiner), all springing from the ciliary muscle; epithelial growths resembling adenoma and carcinoma, (Badal, Lagrange, Lawford, etc) and tumors of the type of a glioma (Emmanuel, Verhœf, Demaria etc), originating from the cylindrical cells of the pars ciliaris retinæ; metastatic carcinoma (Uhthoff); hypernephroma (Chance); and, lastly, lepra nodules (Bull and Hansen).

V. Congenital Anomalies of the Iris

735. Congenital anomalies of the iris are found, for the most part in both eyes. They are apt to be inherited, so that they are frequently found in several members of the same family; they are also met with in conjunction with other congenital anomalies. For the latter reason the disturbance of sight is often much more considerable than would be expected from the optical conditions. In such eyes there frequently exists very great myopia, hypermetropia, or astigmatism, or deficient development of the retina or even of the entire eye, the latter being considerably smaller than usual (microphthalmus). Such eyes, moreover, are more subject than normal ones to disease—(e. g., to irido-chorioiditis, glaucoma, and cataract).

Fig. 326

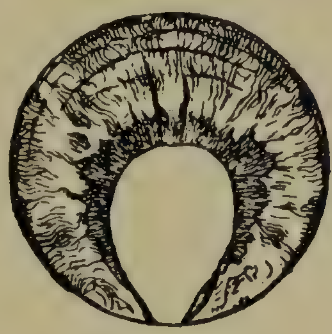


Fig. 327

Fig. 326.—Remains of the Pupillary Membrane. This rises as a small filament, *c*, from the circulus iridis minor, and runs to the pupil, in the centre of which it becomes attached to a small, round, white capsular opacity. In spite of the filament, the lower half of the iris has retracted widely under atropine, so that the filament is greatly elongated; but the upper part of the iris is prevented by two posterior synechiæ (*a* and *b*) from submitting readily to the action of atropine. (Cf. Fig. 307).

Fig. 327.—Congenital Coloboma of the Iris. Enlarged 2 × 1. The pupil as a whole is displaced downward, so that its upper border lies approximately behind the centre of the cornea. It is continued below into the coloboma, and is hence pear-shaped. The circulus minor grows more and more narrow below, so that at the lowest part of the coloboma it is no longer visible; on the other hand, the black fringe belonging to the retinal pigment of the iris is here proportionately broader. The contraction furrows of the iris are present only in its upper part.

736. (1) **Membrana Pupillaris Perseverans** (persistent pupillary membrane)—This consists of a gray or brown tissue which lies upon the anterior capsule of the lens in the region of the pupil, and is usually connected with the iris by brown filaments. Very frequently, however, there are simply a few brown dots present on the lens capsule or only one or two filaments that run from one portion of the pupillary margin to the opposite, and thus bridge over the pupil, or that pass from the iris to the capsule of the lens. They bear a great resemblance to the posterior synechiæ remaining after iritis, but do not, like the latter, rise from the pupillary margin itself, but outside of it, from the circulus iridis minor, which lies on the anterior surface of the iris (Fig. 326, *c*), and which, as embryology shows, gives off the vessels for the pupillary membrane.

Persistent pupillary membrane is of comparatively frequent occurrence in newborn infants, but afterward disappears, except in a few cases in which remnants of it persist during the whole life. The brown filaments stretching from the pupil to the capsule of the lens are blood-vessels which have been obliterated and enveloped in pigment. Where they are attached to the lens capsule, the latter frequently shows a punctate, densely white opacity (Fig. 326, *c*). These brown filaments do not hinder the free movement of the pupil. Moreover, under atropine the pupil dilates ad maximum without suffering any change in its roundness, because the filaments are so extremely extensible. This is another mark distinguishing them from the acquired synechiæ due to inflammation.

737. (2) **Coloboma[5] Iridis.**—Congenital coloboma of the iris is always situated below. The pupil is continued downward to the margin of the cornea, growing narrower all the time, so that it has the shape of a pear with its small end at the lower margin of the cornea (Fig. 327). The sphincter encompasses the pupil and the coloboma, too, as far as the apex of the latter. Congenital coloboma is thus distinguished from the artificial one made by iridectomy. In the latter the sphincter is wanting in the course of the coloboma, because it has been excised; it is seen to end with sharp edges at the dividing line between pupil and coloboma (cf. Fig. 436). Coloboma of the iris is very frequently combined with coloboma in the chorioid and in the ciliary body (see § 769 and Fig. 344), and sometimes also with a small notching of the edge of the lens (coloboma of the lens) at the spot corresponding to the coloboma in the iris (§ 666).

Congenital coloboma of the iris occurs under different forms. Besides the pear-shaped colobomata above described there are occasionally observed some in which the pupil has the shape of a keyhole, as in artificial coloboma. A special variety is the bridge coloboma. In this the pupil is separated from the coloboma by a narrow thread of iris tissue, which stretches like a bridge from one pillar of the coloboma to the other. Incomplete colobomata are of comparatively frequent occurrence; there is then simply a shallow notching of the pupillary margin, or the portion of the iris corresponding to the coloboma is distinguished by a different color, this being generally due to the absence of the anterior layers of the iris at this point.

With coloboma of the iris is often associated a pear-shaped appearance of the cornea, due to narrowing of the latter below.

Slit Pupil (cat's eye pupil) is observed as a rare congenital anomaly.

738. (3) **Irideremia** (**Aniridia**).—The iris may be wanting either altogether or all except a small residual portion. This defect is frequently complicated with congenital opacities in the cornea or in the lens.

739. (4) **Ectopia Pupillæ.**[6]—Even in the normal eye the pupil is usually placed a little below and to the inner side of the centre. While usually this can be noticed only upon careful examination there are cases in which the displacement is marked; indeed, the pupil may be situated quite eccentrically, in the neighborhood of the corneal margin. The displacement is most often upward and outward, and is frequently complicated with a dislocation of the lens (ectopia lentis), usually in the opposite direction (Fig. 299).

740. (5) **Ectropion Uveæ.**—As already stated (§ 27), the retinal pigment epithelium forming the posterior layer of the iris, turns over the pupillary margin so as to be apparent in front as a narrow, black rim encircling the pupil. Occasionally a sector of it extends further so as to form a black apron on the anterior surface of the iris (Samuels).

This occurs not only as a congenital but also as an *acquired* condition; in the latter case in atrophy of the iris (§ 690), glaucoma (see § 839), and other grave diseases of the posterior segment of the eye, especially those caused by traumatism (Stern).

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[5] From *κολόβωμα*, mutilation.

[6] Also called corectopia from *κόρη*, pupil, *ἐκ*, out of, and *τόπος*, place.

CHAPTER XXVI

DISEASES OF THE CHORIOID

I. Inflammation of the Chorioid

741. Inflammation of the chorioid (chorioiditis), if it remains confined to this membrane, produces neither external evidences of inflammation nor pain. The eye looks normal externally, and the disease manifests itself to the patient only through the disturbance in sight—to the physician, only through ophthalmoscopic examination. But if the disease passes over the anterior portion of the uvea it becomes recognizable exteriorly through the symptoms of cyclitis and iritis (*irido-chorioiditis*).

Chorioiditis is distinguished as non-suppurative or as suppurative, according as the exudates disappear again by resorption or lead to the formation of pus. The ordinary variety of chorioiditis is the non-suppurative form, which is known as chorioiditis exudativa. Non-suppurative irido-chorioiditis generally runs a chronic course, being known as irido-chorioiditis chronica (see § 684). In suppurative chorioiditis it is only in rare cases that the inflammation remains confined to the posterior division of the eye. As a rule, the condition present is a suppurative inflammation of all the membranes lining the interior of the eye—an inflammation, therefore, which, in bad cases, rightly bears the name of panophthalmitis.

A. Chorioiditis Exudativa (Non-Suppurativa)

742. General Characters.—Exudative chorioiditis appears for the most part under the form of isolated foci of inflammation scattered over the chorioid (Fig. 328 and 330). While still recent they appear, when seen with the ophthalmoscope, as yellowish, indistinctly outlined spots which lie beneath the retinal vessels upon the red fundus oculi. The spots are produced by an infiltration of the chorioid with exudate which hides the red of the chorioidal vessels; furthermore, the overlying retina is clouded and gray, and covers the subjacent chorioidal mass as with a faint veil (Fig. 328). In proportion as the exudate disappears by resorption the chorioid again comes into view, but in an altered state; it is atrophic, deprived of its pigment, and in part converted into cicatricial connective tissue. Hence, after the disappearance of the exudate, the diseased spot is seen to grow lighter in color. Where the chorioid has become altogether atrophic, a white spot is formed, because the white sclera then shows through; in other cases, remains of the vessels and of the pigment are recognized in the white cicatrix. Afterward the pigment often proliferates, so that the chorioiditic spots appear lined with black pigment or covered with black spots (Fig. 330). The decolorized or pigmented spots remaining after chorioiditis may properly be characterized as cicatrices of the chorioid.

The distinction between *recent exudates* and *old atrophic spots* in the chorioid i based upon the following signs: Exudates are of a yellow or yellowish-white color, d not have a sharp outline, and present to view no chorioidal vessels; retinal vessels which by chance run over them, show by their bending that a projection of the retin exists here, due to the prominence formed by the exudate. The atrophic spots are pur white, and have an irregular but sharp outline, often formed by a pigmented band Pigment spots also lie in the white spot itself; sometimes, indeed, the growth of pigmen becomes so excessive that the spots finally become entirely black. Moreover, remain of the chorioidal vessels are visible within the atrophic area. Such vessels not infre quently present thickened, white-look ing walls, or are even quite obliterate and converted into light-colored cords

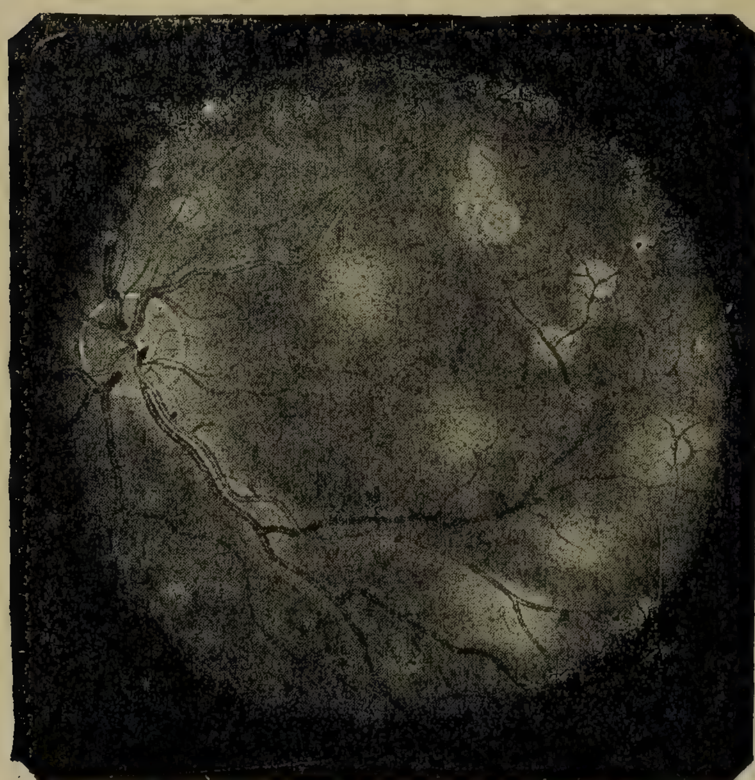


Fig. 328.—Recent Chorioiditis. (After Oeller.)

A man twenty-six years of age otherwise perfectly healthy had noticed for one or two weeks flashes of light and dark blue spots before the eye. Along the large retinal vessels starting from the papilla there is a whitish, œdematous cloudiness of the retina. The chorioiditic spots are arranged irregularly about the macula lutea. The most recent spots are the ones that are most centrally situated; they are grayish-yellow with hazy outlines and without distinct markings. They represent an inflammatory opacity of the retina over the chorioiditic focus which is hence concealed. In the older spots, on the contrary, which are most numerous in the outer upper quadrant, these foci show through the cloudy retina under the form of more sharply outlined discs of about one-fourth the size of the papilla and of a gray color with a lighter centre.

In cases of old retino-chorioiditi the pigment often migrates from th chorioid *into the retina.* That the pig ment is in the retina is obvious fron the fact that the retinal vessels in th spots where this pigment lies are cov ered by it, whereas they pass over pig ment that is situated in the chorioi and are hence not hidden by it.

There are cases in which th atrophy affects only the pigment epi thelium, which disappears. Then th stroma of the chorioid, with its vessel and pigmented intervascular spaces is exposed to view, and there is devel oped the picture of a *tessellated fundu* resembling that observed as a physio logical condition (Fig. 195) but much clearer cut (see Fig. 333). This occur both in diseases of the chorioid and als in diseases of the outer layers of th retina, to which the pigment epitheliun belongs. Besides being found in certain forms of chorioiditis, this change is als found in glaucoma, in myopia of high degree, in retinitis pigmentosa, retinitis syphilitica, etc.

Druses[1] are crystalloid outgrowths of the lamina vitrea of the chorioid, over which the pigment epithelium has undergone destruction. Their true origin is probably in the pigment epithelium. They often project far into the retina. In their typical form druses are discrete, small, whitish, or yellowish spots, unaccompanied by pigmentation or changes in the vessels or the papilla. Sometimes they look like scattered rice grains sown in the red background of the fundus or breaking their way through. Conglomerate and atypical forms occur. They are found especially in the periphery of the fundus and also massed in the maculo-papillary region. In the latter situation they have been called *chorioiditis guttata* (Fig. 329) and *Tay's chorioiditis* (although there is no real chorioiditis present) and *Hutchinson's disease.* The latter includes several varieties of central retinal degeneration (see § 795). The white spots in retinitis punctata albescens (see § 795) have been regarded as druses (Leber). Although usually occurring in the aged, druses

[1]The German synonym "Druse" has nothing to do with Drüse, gland. Both the English "druse" and its German equivalent are derived from the Bohemian druza and denote a cavity in a rock lined with crystals.

are found at times in the middle-aged and even in the young. They seem rarely in themselves to cause any special impairment of vision, the latter when it occurs being due to degenerative changes independently present.

743. Discrete Chorioiditis.—Chorioiditis occurring in *isolated foci of inflammation* is distinguished, according to the location of the latter, into different forms:

1. **Chorioiditis Centralis** is a term rather loosely applied to all sorts of inflammatory, degenerative, and atrophic changes occurring in the region of the macula lutea and characterized therefore by the development of a central scotoma. Owing apparently to the fact that the macula is a specially vulnerable portion of the fundus, these changes may be produced by a variety of conditions. A common cause is myopia, which, if of high degree, leads late in life, almost without exception, to changes in the yellow spot, which are mainly of an atrophic nature (Figs. 333 and 334). Inflammatory changes at this spot are often found in syphilis, in which the region of the macula is sometimes occupied by a large exudate, transformed later into a bluish-gray mass of connective tissue. Circumscribed disease of the macular region may also develop as a result of injuries affecting the whole eyeball, such as contusions, the entry of foreign bodies into the vitreous, etc. Finally, there is observed in old people a disease of the macula which usually affects both eyes about equally, and consists commonly of grayish, reddish, or whitish irregular spots of varying size often surrounded by pigment (Haab), sometimes of a single large whitish area, sometimes of very slight irregular markings. This causes marked impairment of sight, especially for near work, even when but little change is discoverable by the ophthalmoscope. According to Van Der Hoeve it is due to the long continued action of ultra-violet light. He believes that in eyes the lenses of which act to diffuse light (i.e., scatter the rays laterally) the injurious effect of the ultra-violet rays is exerted on the ciliary body causing nutritive changes, which lead to cataract (§ 655); in eyes whose lenses transmit light without much diffusion, this injurious effect is exerted on the macula. Accordingly, he finds that senile cataract and central senile chorioiditis do not occur in the same subject.

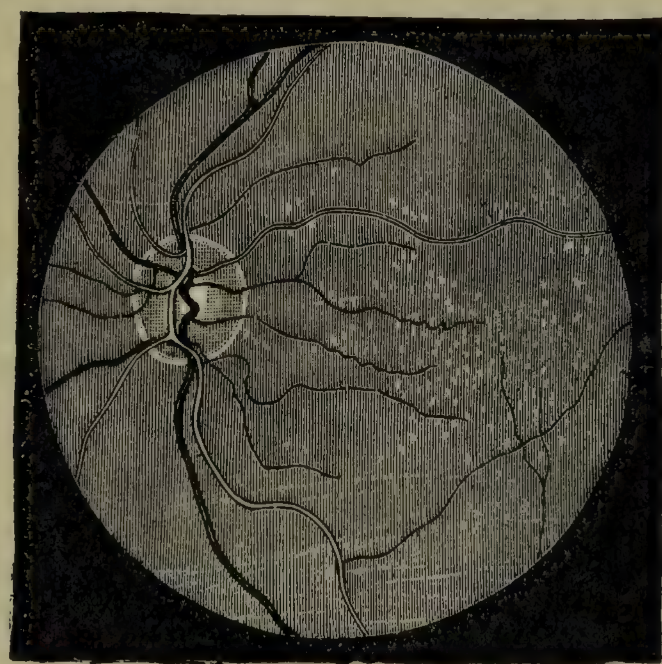


Fig. 329.—Senile Guttate Chorioiditis (Tay and Hutchinson). (After Weeks).

2. **Chorioretinitis Juxtapapillaris** (Jensen's retino-chorioiditis) is a condition marked by the formation of one or more ill-defined, woolly, slightly prominent white exudates, usually situated close to the disk and producing a corresponding sector-like defect in the field of vision extending to the blind spot. Sometimes also there are arcuate or semicircular defects surrounding the fixation point. The exudates may contain small hæmorrhages and may also be associated with string-like opacities in the vitreous with which they are sometimes connected. Later the spots are replaced by ordinary atrophic areas. The optic disk remains normal. The condition shows a tendency to recur. It apparently originates in the nerve-fibre layer of the retina and is due in most cases probably to tuberculosis, in some cases to syphilis (Dimmer).

3. **Chorioiditis Disseminata** is characterized by numerous round or irregular spots which are scattered over the fundus (Fig. 328, 330). This is an eminently chronic variety in which, in the course of time, new spots are being constantly formed. Often this takes place discontinuously; i.e., after a long interval suddenly a number of new foci develop, as if the morbific agent had been carried by the blood-current to different parts of the chorioid at the same time.

In old cases, the chorioid is studded all over with spots, which in many place become confluent, so that the fundus looks spotted for a large part of its extent. Th sight may, nevertheless, still be pretty good, if only the region of the macula lutea remain intact. In the beginning of the disease hyperæmia of the retina and optic nerve i present, but later both become atrophic. The papilla takes on a dirty grayish-red colo and loses its sharp outline (chorioiditic atrophy); the retinal vessels are fewer and ar greatly contracted.

A special form of chorioiditis disseminata is *chorioiditis areolaris* (Förster). I this the first foci develop in the vicinity of the macula, while the subsequent ones mak their appearance at a constantly in creasing distance from the latter. Th behavior of the individual spots is di rectly the reverse of the ordinary cours pursued by chorioiditic patches; th most recent (more peripherally situated spots are entirely black, and afterwar slowly enlarge, and at the same tim become decolorized from the centr toward the edge. They look then like black rings inclosing a whit centre, and at last they get to b almost entirely white.

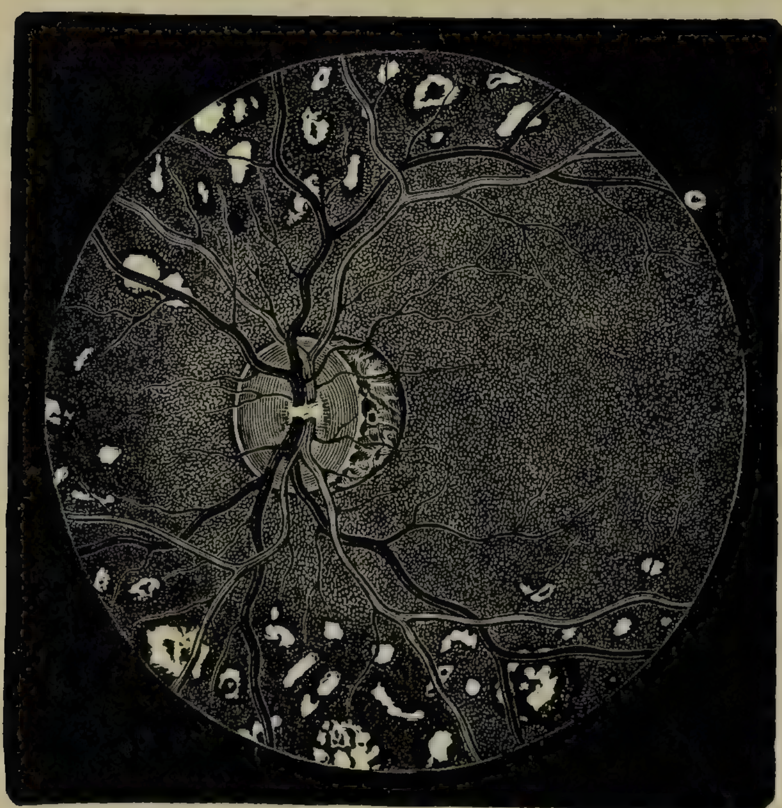


Fig. 330.—Chorioiditis Disseminata. (After De Wecker.)

That the chorioiditis has attacked a myopic eye can be recognized from the atrophic crescent which incloses the papilla upon the temporal side. The crescent is sharply defined by the scleral ring on the side next to the papilla, and by the pigment ring on its temporal side, and shows everywhere remains of the chorioidal vessels and also of the pigment. The chorioiditic foci occupy chiefly the equatorial parts of the fundus. They are white, with a lining of pigment of greater or less width; many also have spots of pigment in their interior. The retinal vessels are perfectly distinct as they pass across the spots and their pigment—i. e., they lie in front of the latter.

Another form is the *chorio-retinitis sympathica* occurring in the early stages of sympathetic ophthalmia (§ 710). The disseminate spots which are sometimes very numerous (like stars in a sky—Caspar) present varying aspects, but especially occur as yellowish or reddish (later whitish) circular spots, having a diameter three or four times that of the larger retinal vessels and surrounded by a mottled brownish fundus (Haab). Sometimes minute red lines are found in the macula, surrounded by brownish mottling. Neither disseminate nor macular changes cause much impairment of vision.

4. **Chorioiditis Anterior** deposits its foci of exudation at the periphery of the chorioid. These foci are therefore readily overlooked if we neglect to examine the most anterior portions of the fundus with the ophthalmoscope. Chorioiditis anterior occurs most frequently in eyes affected with excessive myopia. In young persons, chorioiditis anterior often occurs in consequence of hereditary syphilis; usually the periphery of the fundus is studded with roundish ink-black spots (see § 576). In old people, simple pigmentary changes are frequently found in the anterior portion of the chorioid.

744. Diffuse Chorioiditis.—Chorioiditis which is spread diffusely over the whole chorioid is always combined with a marked retinitis, and is therefore ordinarily known as retino-chorioiditis or chorio-retinitis.

In the typical fashion in which it was first described by Förster this occurs in *syphilis* (Fig. 354, E). In recent cases the retina appears clouded, and, furthermore the entire fundus is veiled by a fine punctate haziness of the vitreous; moreover, circumscribed exudates may also be present in the chorioid and the retina. These occupy

mainly the region of the macula, and usually appear under the guise of large or small irregular patches, which are of a gray or dirty yellow color and are ill defined, so that they are discovered only upon careful examination with the erect image and with the pupil dilated. In the later stages the cloudiness of the retina disappears, to be replaced by atrophy; at the same time a migration of pigment under the form of numerous black spots takes place into the retina, especially at its periphery, so that there is produced a picture very similar to that of retinitis pigmentosa (see § 793). A similar finding is sometimes had in cases of chronic diseases (either inflammations or new growths) of the liver associated with jaundice. There then ensues in conjunction with the symptoms of hemeralopia a chronic inflammation of the chorioid and retina with pigmentation of the latter (*ophthalmia hepatica*).

745. Chorioidal Changes in Myopia.—Myopia is often complicated with changes in the chorioid, it being a rare exception to find the latter normal in high myopia (Figs. 331 and 333). The changes in the chorioid in this case are, to be sure, less those of a chorioiditis proper than of a primary atrophy caused by the stretching which the chorioid necessarily undergoes, when the entire posterior segment of the sclera bulges backward, as is the case in extreme myopia.

The changes consist in an atrophy of the chorioid close by the papilla and in foci of disease at other spots, particularly in the region of the macula lutea.

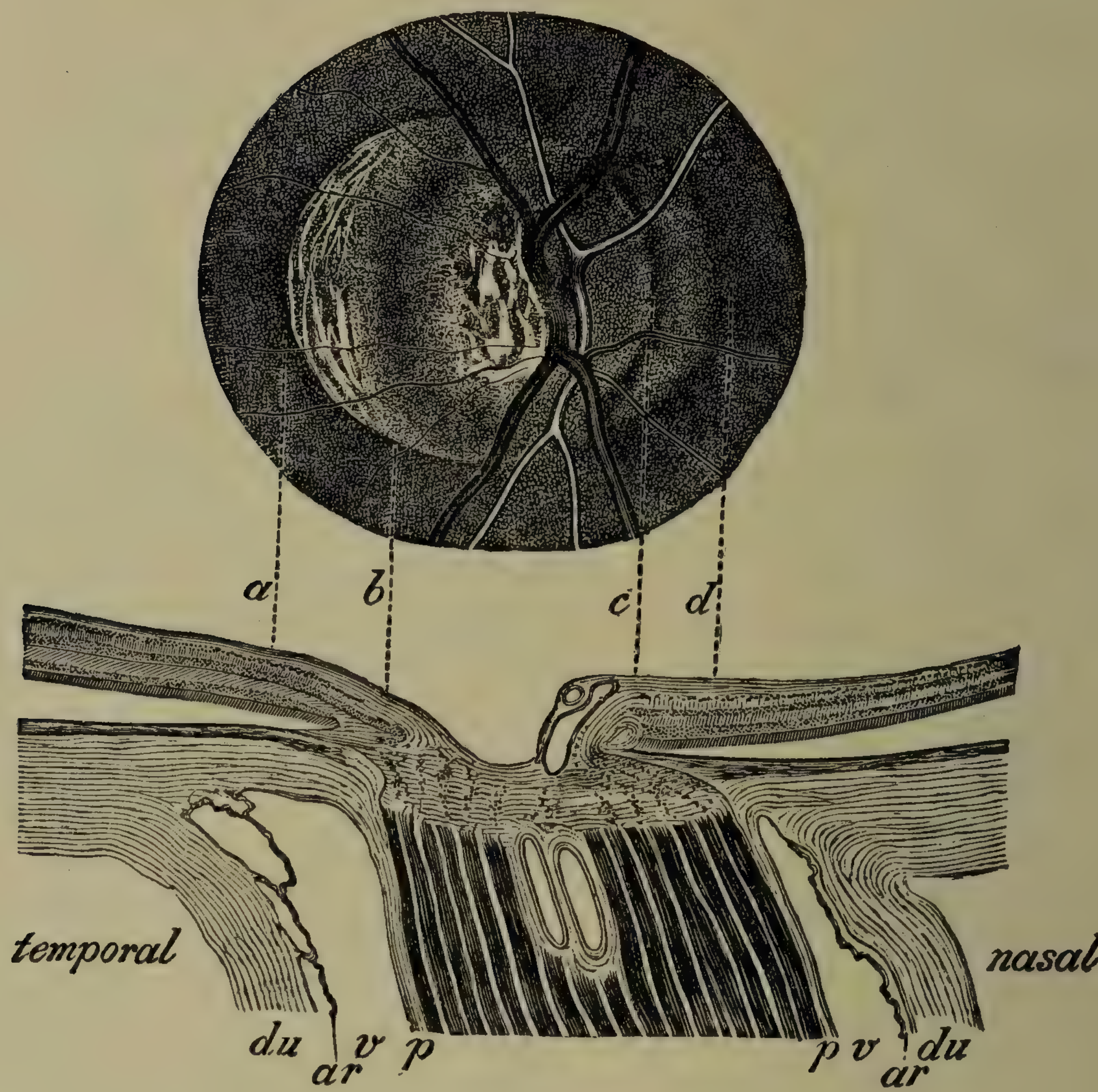
(*a*) Atrophy of the chorioid *at the border of the optic disc* begins at the temporal side of the latter in the form of a narrow light-colored crescent (*distraction crescent*, called conus by Jäger; Fig. 331, *ab*). This is accounted for by Stilling as being due to the displacement of the optic nerve-head toward the temporal side. As such crescents often occur in emmetropic eyes, their mode of development has already been considered in § 404. In near-sighted eyes the displacement of the optic nerve occurs much more regularly and to a much greater extent. As the wall of the sclerotico-chorioidal canal is pulled away from the optic nerve on the temporal side, the sclera and chorioid are pulled up over the nerve to a like degree on the nasal side (Figs. 331, 332, *cd*). At the nasal side the optic nerve shows through the sclera and chorioid, and hence with the ophthalmoscope we see along the nasal border of the papilla a somewhat hazy yellowish crescent (*supertraction crescent*—Weiss). With the displacement of the walls of the orifice for the optic nerve there is afterward associated atrophy of the adjacent chorioid, which now allows the sclera to show white through it. In this way the white surface on the temporal side of the optic nerve grows constantly in circumference and then extends along the upper and lower borders of the nerve to the nasal side of the latter, until finally the papilla is surrounded on all sides by a white zone (*circumpapillary atrophy*; also called annular staphyloma—Fig. 333). The name staphyloma is properly applied to a bulging of the sclera, but is also applied to the atrophy of the chorioid which is the result of the bulging. The circumpapillary atrophy is generally broadest on the outer side, where it started In the atrophic area the fundus is either a pure white, if the chorioid there has completely disappeared and the sclera comes into view; or remains of chorioidal tissue, such as vessels and pigment, are still present in it in varying amount.

The boundary between papilla and circumpapillary atrophy is ill-defined, so that the tyro regards the white disc-shaped area formed by the two simply as an extremely large papilla. He will escape this error if he notes that the papilla proper is red—indeed often so red by contrast with the white area about it that it appears hyperæmic.

When the atrophic areas are large the *disc appears elliptical*, the short axis of the ellipse coinciding with the greatest diameter of the atrophic zone. When, therefore the latter is broadest laterally, as is commonly the case, the disc forms an upright ellipse (Fig. 333). This change of shape must in part be referred to the fact that the disc, being pushed toward one side in consequence of the bulging of the sclera, is seen in perspective foreshortening. But it is also due in part to the fact that the sclera and chorioid,

A

B



FIGS. 331-332.—OPTIC-NERVE ENTRANCE IN MYOPIA.

FIG. 331 (A) OPHTHALMOSCOPIC IMAGE OF THE PAPILLA.—The papilla, *b-c*, is of the shape of a ellipse with its long axis vertical. In its outer half it shows the large physiological excavation, up whose floor are visible the gray stipplings of the lamina cribrosa, while the central vessels ascend on t inner wall of the excavation. Adjoining the outer border of the papilla and not sharply separated fro it is the bright crescent, *a-b* (distraction crescent). This is of a white color, while the papilla itself reddish. The crescent is covered with brownish, elongated markings, representing remains of the stron pigment of the chorioid. The temporal border of the crescent is sharply defined, and the chorioid adjoi ing it is somewhat more pigmented than usual. On the other hand, the chorioid in the vicinity of t nasal border of the papilla shows a somewhat lighter coloration in the space between *c* and *d*, so that yellowish crescent, which, to be sure, is not much more than a suggestion of one, is formed on the nas side of the disc (supertraction crescent).

FIG. 332 (B) LONGITUDINAL SECTION THROUGH THE HEAD OF THE OPTIC NERVE. Magnifie 14 × 1.—The optic-nerve funiculi, wherever they consist of medullated fibres, are colored black b Weigert's hæmatoxylin stain; between them can be seen the septa, which remain unstained, and t longitudinal sections of the central artery and central vein. The black staining ceases abruptly at t lamina cribrosa. In front of the lamina cribrosa the head of the optic nerve presents the physiologic excavation. This is a depression whose floor at its deepest part is formed by the lamina cribrosa. T temporal wall of the excavation slopes down quite gradually from the retina. The nasal wall declin abruptly, and shows the cross section of the central vessels. The trunk of the optic nerve is insert obliquely into the eyeball and also passes obliquely through the coats of the eye; it looks as if the latter had been drawn to the temporal side, and the optic nerve to the nasal side, so that there h occurred a displacement of these two structures with reference to each other—a displacement whic is most marked where the optic nerve passes through the sclera and chorioid—i. e., in the sclerotic chorioidal canal. Consequently, the temporal wall of the latter is turned somewhat forward, an hence, since the overlying retina is transparent, comes into view when looked at from in front (wi the ophthalmoscope), forming a bright crescent extending from *b* to the point *a*, where the pigme epithelium begins. The stroma pigment of the chorioid extends somewhat farther inward than do the pigment epithelium, and is consequently seen under the form of brown spots upon the light-color crescent. The nasal wall of the scleral canal is turned partly backward, so that it has to pass in fro of the most nasally situated portion of the optic nerve, *c-d*. As the displacement affects not on the aperture in the sclera but also in the chorioid, the latter is also drawn up over the nasal border the optic nerve as far as the point, *c*. Since now this nasal portion of the disc, being covered by t sclera and chorioid, is not distinctly visible with the ophthalmoscope, the disc appears contracted in i horizontal diameter. Nevertheless, the portion of the optic nerve that is thus concealed glimme through its covering, so as to be distinguishable under the form of an ill-defined yellowish crescent a the nasal border of the papilla (*c-d* in Fig. 331). The displacement of the optic nerve with referen to the sclera is shared in by the sheaths of the nerve. The dural sheath, *du*, and the adjoining arachno sheath, *ar*, are separated from the nerve, especially at its temporal side, and the intervaginal spac *v*, *v*, is consequently dilated. On the other hand, the pial sheath, *p*, lies in close apposition to the nerv

which on the outer side have receded from the border of the disc, have been drawn up over the border of the latter on its inner side (supertraction crescent—cf. Fig. 332).

The *retinal vessels* that emerge from the papilla are thin and are marked by their straight course—looking as if they had been put on the stretch.

Sometimes two or even three *contrasting zones* are present in the circumpapillary atrophy which differ from each other in their pigmentation, and often also lie at different levels, and which bear witness to the fact that the development and enlargement of the staphyloma have taken place at different periods (Fig. 333). By proliferation of the pigment, brown or even black crescents are formed.

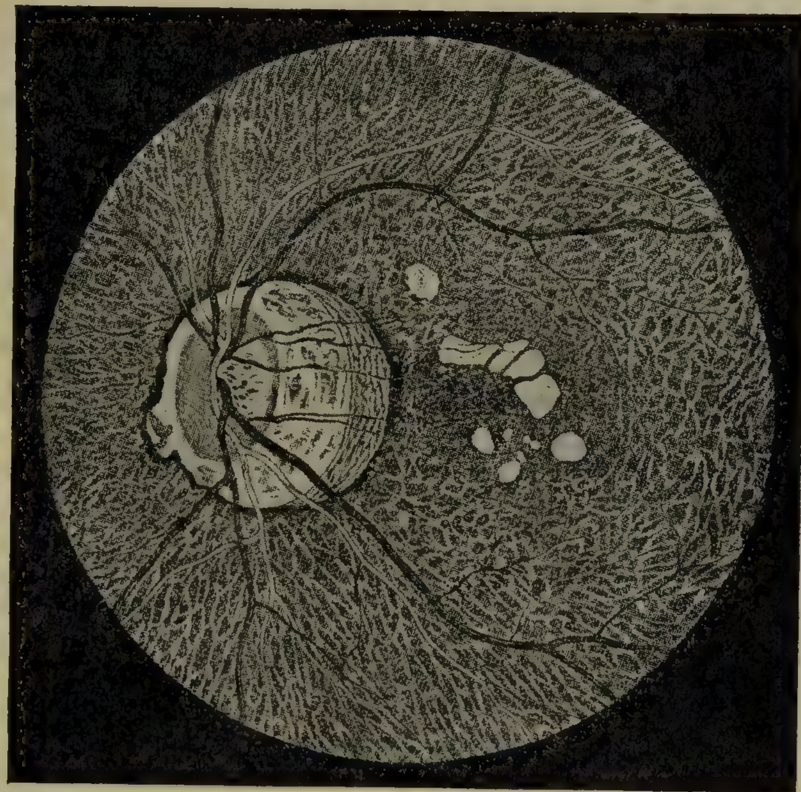


FIG. 333.—FUNDUS IN MYOPIA OF HIGH DEGREE. (In part after De Wecker.)

The papilla is oblong-oval and has a physiological excavation to the outside of the point of entrance of the retinal vessels. It is surrounded by atrophic chorioid, the staphyloma posticum. This is very broad on the temporal side, and consists there of two divisions; the outer, more pigmented one, showing with especial distinctness the remains of the chorioidal vessels. On the nasal side, the staphyloma is narrower, is lined by a rim of pigment, and is of irregular contour. In one of the outlying portions is noticed a posterior ciliary artery which enters the chorioid from the sclera. The rest of the fundus is tessellated in such a way that the vascular network of the chorioid is clearly recognized. The region of the macula lutea is occupied by chorioiditic changes, consisting partly in an overgrowth of pigment (the small black specks), partly in atrophy (the white patches).

The line separating the atrophic zone from the healthy chorioid is often sharply defined, especially if formed by a pigmented margin. In other cases a sharp border line is absent, a circumstance which indicates that the staphyloma is in process of growth so that an advance in the myopia is to be apprehended.

The *size* of the atrophic area is, broadly speaking, in direct proportion to the degree of myopia, but variations from this rule very often exist in the individual cases—e. g., great myopia without atrophy of the chorioid, and vice versa. Not infrequently also white crescents and even annular white zones are found in emmetropic and even in hypermetropic eyes.

What, ophthalmoscopically speaking, we call a staphyloma—i.e., the white ring that surrounds the papilla—is indeed caused by the protrusion of the sclera, but is not quite coincident with the latter, being, in fact, less extensive. In many cases of extreme myopia, however, it is also possible to recognize with the ophthalmoscope the limits of the actual *staphyloma in the sclera* under the form of a broad, dark, curved line, which usually runs in the red fundus along the inner side of the white staphyloma and concentric with the latter. In well-marked cases we can see from the parallactic displacement, or from the bending of the retinal vessels as they pass over the dark curved line, that the latter corresponds to a sudden change of level in the fundus (staphyloma verum).

For the *inferior* crescent see § 404.

With the circumpapillary atrophy of the myopic eye should not be confounded cases of atrophy of the chorioid about the papilla from *other causes.* Under this latter head belong atrophy after chorioiditis, and also the atrophy in cases of glaucoma, the so-called halo glaucomatosus.

(*b*) The changes of the chorioid in the region of the *macula lutea* make their appearance when the myopia has reached a high degree. Sometimes there is simply a slight rarefaction of the chorioid between the disc and the yellow spot, making it appear as if the details of the fundus had been partially erased. When the condition is more advanced both light-colored and pigmented spots are found (Fig. 333), and not

infrequently also white branched lines. The spots gradually enlarge, and finally coales to form pretty large atrophic patches, which may even ultimately become united wi the staphyloma surrounding the papilla. In such cases of extreme myopia, almost t entire posterior section of the interior of the eye is converted into a great white patc Moreover, hæmorrhages occur in myopic eyes, and that, too, preferably at the site the yellow spot. Another, though rare alteration found in myopic eyes, consists in t formation right in the macula of a coal-black, round spot which gradually grows to abo the size of the papilla (Fig. 334). In the subsequent course of this lesion the centre the spot grows continually lighter. It is the changes in the macula lutea that, besid detachment of the retina, represe the greatest danger for the extreme myopic eye. They do not, like d tachment, threaten the eye wi complete blindness, but they rend it unserviceable for any sort of fi work. Besides, they are much mo frequent than detachment of t retina, inasmuch as only a few of t excessively myopic attain any co siderable age without being affecte with these changes.

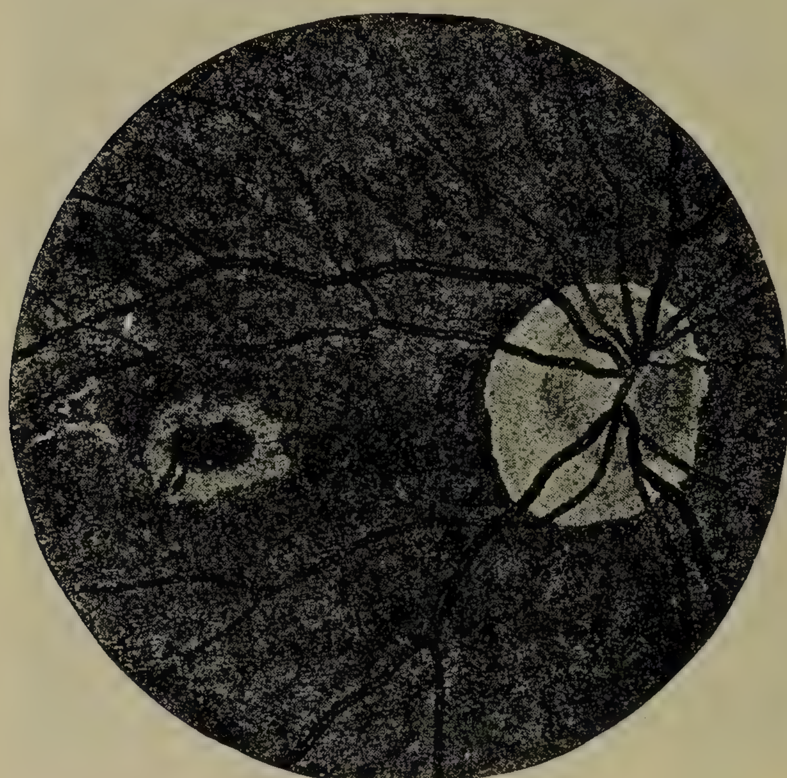


Fig. 334.—The Central Black Spot in Myopia Right eye, erect image.

Fourteen days previously a black spot had appeared before the eye which made reading impossible; the lines of the book seemed bowed. The papilla is surrounded by a zone of atrophic chorioid. At the site of the macula lutea there is a horizontally elliptical spot, the marginal portions of which are deep black and the centre a dark gray. It is sharply defined and is encompassed by an irregular zone of atrophic chorioid in which, to the outer side and below, lie two small streaky hæmorrhages. The eyeground, being but slightly pigmented, enables one to recognize the chorioidal vessels; between the papilla and macula lutea and out beyond the latter the pigmentation of the eyeground is irreguarly maculate. Up and out from the macula lies a group of atrophic white spots, representing the results of stretching of the chorioid.

746. Anatomical Changes i Chorioiditis.—In exudative chorio ditis (Fig. 335) there is first a cellula infiltration, which originates mainl from the innermost layers of th chorioid and spreads less toward th outer layers of the chorioid than i does in the direction of the retina Within these two membranes, th retina and chorioid, the infiltration i found chiefly along the vessels, which indeed may be said to be sheathed i the cells of the exudate. An exudate often of considerable size, is deposited between the chorioid and the retina. This exudate is afterward transformed into a connective-tissue membrane (cicatrix), binding the chorioid fast to the retina; and in this situation these two membranes themselves become atrophic (Fig. 336). From the chorioid the fine vessels and also the chromatophores in great part disappear; and the vessels that are left either have their walls thickened and sclerosed, or are obliterated altogether. The retina is converted into a network of connective tissue and glia, in which can be seen the vessels with their walls greatly altered. Wherever the chorioid is adherent to the retina, the layer of rods and cones is absent, and so is the pigment epithelium, except for a few remnants. On the other hand, the pigment epithelium at the border of the scar proliferates; hence the black lining of the chorioiditic patch visible with the ophthalmoscope. In part the proliferating pigment epithelium migrates into the retina, where it is found particularly in the neighborhood of the vessels. The inner surface of the retina is sunken at the site of the cicatrix, and is frequently adherent to the surface of the vitreous (to the hyaloid membrane).

For the changes in *chorioiditis sympathica* see § 699.

In *irido-chorioiditis chronica* the changes in the chorioid and retina are similar to those just described, with the addition of the evidences of chronic inflammation and afterwards of atrophy in the iris and ciliary body.

The changes in the *chorioid in myopia* consist mainly in atrophy with slight evidences of inflammation. From the stretching of the chorioid results a splitting of the lamina vitrea. This latter gapes and so later on does the underlying tissue of the cho-

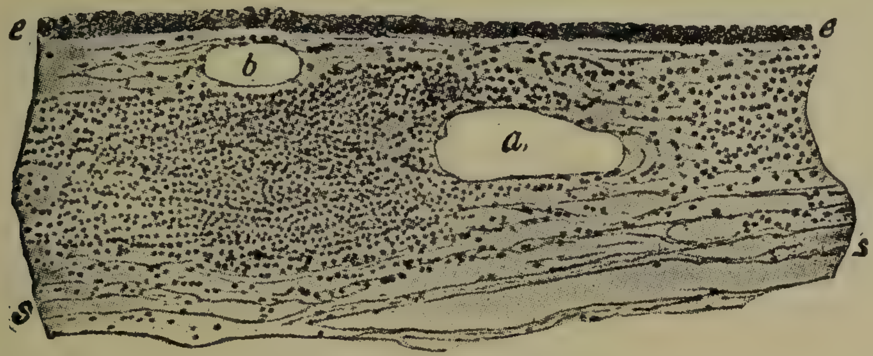


FIG. 335.—RECENT CHORIOIDITIC FOCUS. Magnified 85 x 1.

The cellular infiltration lies mainly in the layer of large vessels to which the vein *a* belongs, while the vein *b* lies in the layer of medium sized vessels. The infiltration extends backward so far as to get between the lamellæ of the suprachorioid *s*. The pigment epithelium, *e*, over the focus is preserved so that the latter under the ophthalmoscope must have been indistinct and, because the leucocytes concealed the red chorioidal vessels, must have appeared under the form of a somewhat light-colored and not sharply defined patch.

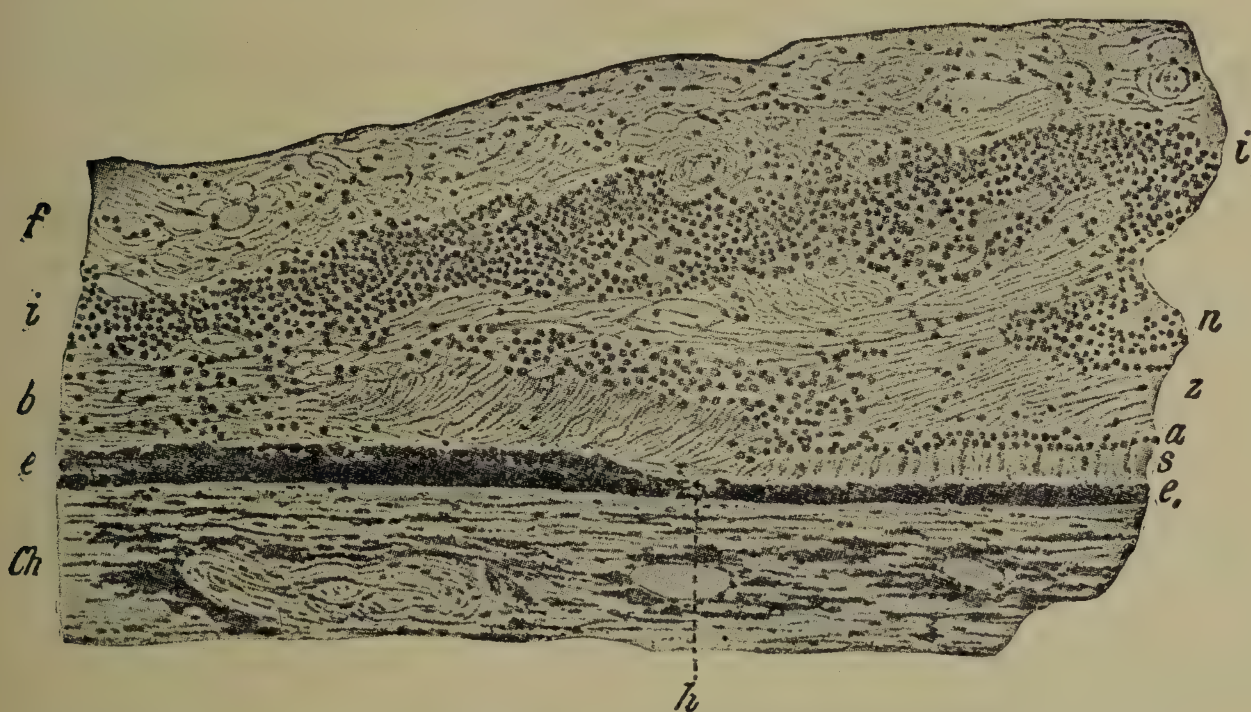


FIG. 336.—OLD FOCUS OF CENTRAL RETINO-CHORIOIDITIS. Magnified 136 × 1.

The inflammation has run its course in the chorioid and the outer layers of the retina in the region of the macula lutea; the figure represents only the edge of the focus which lies to the left of the drawing. In the focus itself the chorioid, *Ch*, is thickened and fibrous, and its vessels are to a large extent obliterated. Upon it lies the pigment epithelium, which at *e* has developed by proliferation into a double layer of cells. This corresponds to a black spot seen with the ophthalmoscope. The retina is adherent to the pigment epithelium and is degenerated. The fibre layer, *f*, is comparatively the best preserved, but still it is filled with holes. The layer of ganglion cells has disappeared, the inner granular layer, *i i*, has been irregularly converted, and the layers of the retina that succeed it exteriorly have been entirely converted into a fibrous structure composed of a new-formed connective tissue, *b*, and of glia fibres, in which there still lie a few scattered exterior granules. The adjacent retina is drawn up by the shrinking of this tissue (cicatrix). This fact can be most distinctly made out from the wrinkling which the inner granular, *i*, has experienced because of the traction, with the result that it shows in the section, at *i* and *n*, a kinking which appears Z-shaped in the section. To a smaller extent also this wrinkling is present in the rarefied external granular layer, *a*. Here the wrinkle is situated at the margin of the focus, at *h*; from this point the outer passes forward to the inner granular layer and becomes amalgamated with it. The atrophic intermediate layer, *z*, consists of delicate oblong fibres (supporting fibres), which come into view because of the atrophy of the nerve tissue. The layer of rods and cones, *s*, and also the pigment epithelium e_1, are fairly well preserved up to the edge of the focus, *h*. Here there is an interruption of the pigment epithelium and, from this point on, the rods and cones are obliquely placed and elongated up to the spot where they finally disappear altogether. Owing to their oblique position there was distorted vision (metamorphopsia) at the border of the blind spot or scotoma, that corresponded to the focus itself in which the visual cells had been entirely destroyed.

rioid. At these points the retina becomes adherent to the chorioid, the rods and cones being at the same time destroyed so that the sight is impaired (Salzmann).

747. Complications.—It is evident that the retina, since it directly adjoins the chorioid, must be sympathetically affected in those spots where the chorioid is diseased. If the implication of the retina is particularly prominent, we speak of retino-chorioiditis, or chorio-retinitis (cf. § 744). Moreover, the exudates from the chorioid pass not only into the superimposed retina, but also through the latter into the vitreous. Opacities of the vitreous are hence an almost constant accompaniment of chorioiditis.

748. Symptoms.—It is the implication of the retina and vitreous which causes *disturbances of vision* of various kinds, and thus directs the patient's attention to the eye. The vision is diminished as a whole on account of the cloudiness of the vitreous and the hyperæmia of the retina. But in those spots in which inflammatory foci exist, sight may be entirely abolished, so that insular defects (scotomata) are present in the field of vision (see § 271). The light sense especially in the diffuse forms is often much reduced. Owing to the fact that the retina over the focus of inflammation is pushed forward and its elements are displaced from their normal situation (Fig. 336), objects whose images fall on the retina may appear distorted (metamorphopsia); straight lines, for instance, appear bent in various directions. Frequently also objects appear smaller than they are (micropsia) or in the atrophic stage look larger than they are (retinal macropsia). (See Fig. 115).

As long as the inflammation is recent, symptoms of irritation of the retina manifest themselves; subjective sensations of light (photopsiæ) exist, such as spots before the eyes, sparks and balls of fire, etc. These phenomena cause the patient annoyance and anxiety, sometimes to an extreme degree. When, after the subsidence of the inflammation, atrophy of the chorioid and of the superimposed layers of the retina has taken place, signs of absence of function—i. e., gaps in the field of vision—take the place of the signs of irritation. The influence that the scotomata exert upon the sight in general depends primarily upon the place they occupy in the fundus. Peripherally situated scotomata cause but little disturbance of vision, even when they are pretty numerous; and if they occur only in one or two places they usually escape the patient's notice altogether. On the other hand, when a scotoma occupies the site of the yellow spot the disturbance of vision is as great as in the preceding case it is trivial; direct vision is then destroyed, and the eye becomes unserviceable for fine work. The first case would be met with in the chorioiditis represented in Figs. 328 and 330, the second in Figs. 333 and 334.

749. Course.—The course of chorioiditis is chronic, it taking many weeks for the foci of exudation to be converted into atrophic spots. The opacities of the vitreous last even longer—often, in fact, permanently. But chorioiditis is chiefly dangerous because of its tendency to recur, in consequence of which new foci of inflammation are constantly developing in the chorioid, so that the latter is finally covered all over with old and recent spots. With this is ultimately associated atrophy of the retina and optic nerve, so that obstinate cases of chorioiditis terminate in partial or total blindness. When the affection of the chorioid is well advanced, clouding of the lens (cataracta complicata) is almost always associated with it.

750. Etiology.—Exudative chorioiditis is a frequent disease, which is observed at all ages. Among its most ordinary causes is syphilis, both acquired and hereditary. As a result of the latter, cases also of congenital chorioiditis have been observed. Chorioiditis may also be caused by general disorders of nutrition of various sorts, such as anæmia, chlorosis, tuberculosis (see § 762), scrofula, gastro-intestinal toxæmia, dental and tonsillar infection, etc. In many cases of chorioiditis the cause remains obscure.

751. Treatment.—The treatment of chorioiditis must regard mainly the *etiological* factor. When, as in syphilis, this is readily amenable to therapy, favorable results are promptly attained—in fact, syphilitic chorioiditis offers the best prognosis, as by energetic antisyphilitic treatment speedy improvement in most cases, and often even an entire cure, can be obtained. If there is evidence that the chorioiditis is tuberculous, tuberculin injections with invigorating general treatment are advisable. Moreover, we must look out for other possible causes of chorioiditis, such as metabolic disorders, remote infections, etc., by a searching examination (cf. § 718), and if these are found must seek to remedy them. To be sure, we are not always able to prevent the frequent recurrences which may still lead ultimately to destruction of sight.

The treatment of the *local* changes should aim to produce rapid resorption of the exudate in the chorioid and the retina and also in the vitreous. Suitable remedies for this purpose are the iodide of potassium or, when necessary, a treatment by inunction, which, even in non-syphilitic cases, can do good service through its absorptive action; furthermore, diaphoresis and subconjunctival injections of salt solution. Care should be taken not to subject the eyes to strain (from reading, etc.), and to protect them from undue light by dark glasses or, if necessary, by rest in a dark room.

B. *Chorioiditis Suppurativa, Endophthalmitis*

752. Origin.—The feature which the diseases included under these names have in common is the purulent exudation into the vitreous. This is the analogue of the accumulation of pus in the anterior chamber, but with the difference that a hypopyon can disappear without entailing any lasting injury, while suppuration in the vitreous leads to permanent and serious changes and generally, in fact, to the destruction of the eye. Like hypopyon, the pus in the vitreous is thrown out by the membranes that line the cavity. The starting point of an inflammation of this sort may be in (1) the *chorioid.* This is the case if pyogenic bacteria have been carried by the blood current into the vessels of the chorioid and lodge there (metastatic inflammation). The inflammation then passes from the purulent foci in the chorioid to the other membranes lining the interior of the eye, and the pus thrown out by these membranes is poured into the vitreous. These cases are rather rare in comparison with the much more frequent instances in which the starting point of the inflammation is (2) the *vitreous* itself. In this case the bacteria are introduced from outside and most frequently by means of a perforating injury. The germs that excite the suppuration then, starting from the vitreous, act, by means of the toxins that they produce, upon the membranes

that line the vitreous cavity—primarily upon the ciliary body and retin and secondarily upon the chorioid. In some cases of perforating injury an also after perforation of a corneal ulcer, the bacteria get into the (3) *aqueou chamber* first and then pass from this into the vitreous; or sometimes it i only their toxins that pass by diffusion from the aqueous into the vitreou chamber. In all these cases, however, whatever the starting point of th inflammation, the ultimate result of it is the same in all, namely a depositio of a purulent exudate in the vitreous due to inflammation of the inner coat of the eye (*endophthalmitis*). We may distinguish three categories o endophthalmitis:

753. (1) That in which the purulent inflammation remains *confined t the posterior segment of the eye.* Since the posterior membranes of the eye i. e., the chorioid and retina, have no sensory nerves, even a purulent inflam mation of them runs its course without pain and without external evidence of inflammatory reaction. Hence, exteriorly the eye appears normal, an we simply see deep in behind the lens the yellow exudate in the vitreous— *abscess of the vitreous* (Figs. 337 and 338). Later on, this is transformed int a membrane, by the shrinking of which the retina is detached and the eye-ball becomes softer (*atrophy of the eyeball*). From the start the sight is ver much reduced by the exudation in the vitreous, and later is as a rule abso-lutely annihilated. It is only in exceptional cases that a portion of th retina remains attached, and that a residue of sight is preserved.

The cases of simple suppurative chorioiditis which run their course without producing an external inflammation are rare.

Cases of simple abscess of the vitreous may be confounded with neoplasms in the eyeball. This is possible because the inflammation in these cases runs so sluggish a course that all external signs of inflammation are wanting. The eye is not discolored, the iris is normal, the aqueous and lens are clear. The lens and iris are pressed forward by the exudation in the vitreous, and the anterior chamber is made shallower. The pupil is dilated, and permits the exudate, or abscess, to be seen deep down in the vitreous. Sometimes the exudate is visible even some distance off as a vivid, light-colored (whitish or yellow) reflex from the pupil (*amaurotic cat's eye*—Beer). Just the same phenomena may be produced by new formations in the vitreous, and particularly by gliomata arising from the retina (see § 804), for which reason many cases of the sort described above have been designated as *pseudo-gliomata.* The most important distinctive mark lies in the tension of the eye. This, in genuine glioma, is normal in the beginning, and afterward is increased; in pseudo-glioma, on the contrary, diminution of tension soon sets in, which is followed by the shrinking of the eye. Then the subsequent course renders the true state of the case perfectly clear, inasmuch as glioma afterward breaks through the sclera and keeps on growing, while in pseudo-glioma the eye keeps constantly growing smaller.[2] But now in glioma it is requisite to remove the eye as early as possible, and it is therefore not right to wait a long time in doubtful cases until the diagnosis can be determined with certainty. In so doing, we should be putting our patient's life in jeopardy. Hence, in doubtful cases, we perform enucleation. Even if it should then turn out that the case was one of pseudo-glioma, the patient has not lost much by the enucleation, since the eye is already blind and would be much shrunken. In any case, the question of a confusion with glioma would come up only in cases of sluggish abscess of the vitreous occurring in children, since it is only in the children that glioma of the retina is found.

[2] In rare cases, however, a glioma or sarcoma may be associated with irido-cyclitis and come to be lodged in a phthisical eye. See remarks under Sarcoma of the Chorioid, § 764.

The causes of pseudo-glioma are most frequently meningitis, also the acute exanthemata, and finally injuries, particularly those attended with the presence of a small foreign body in the interior of the eye. One or two cases of pseudo-glioma have turned out on dissection to be tuberculosis of the chorioid; others are due to exudative retinitis (§ 788) and in some instances a mass of tissue of fetal origin situated behind the lens (see § 666) is the cause of the yellow reflex.

754. (2) The purulent inflammation extends from the back part of the eye to the region of the anterior chamber; and in other cases, as stated

FIG. 337.

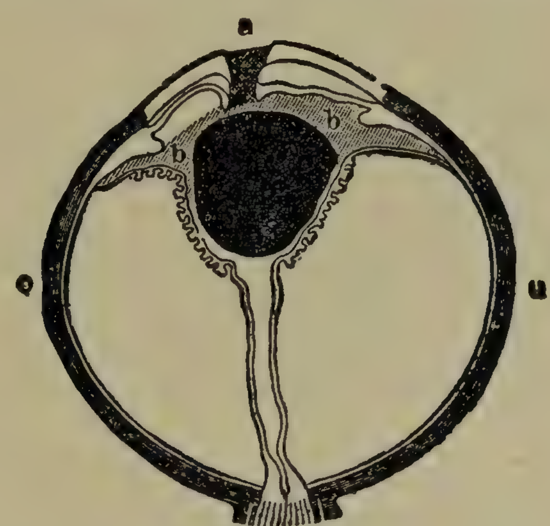


FIG. 338.

FIG. 337.—ABSCESS OF THE VITREOUS. HORIZONTAL SECTION. A perforating injury had been produced by a piece of iron in the upper and inner quadrant of the sclera. When the patient presented himself five days afterward there was irido-cyclitis with hypopyon and a gray reflex behind the lens, and the tension of the eye was somewhat elevated. In the course of the treatment the irido-cyclitis improved, but the deep reflex increased and became yellow. The tension of the eye sank below the normal and the perception of light was abolished. On this account the eye was enucleated five weeks after the injury. The site of the wound in the sclera is not included in the section here represented. As a result of the iritis there is on the temporal side an adhesion of the pupillary margin to the lens capsule, with a hump-like protrusion of the iris. On the nasal side the iris is adherent over its entire posterior surface to the lens capsule and to the exudation membrane which fills the posterior chamber. Hence it does not protrude. A cyclitic membrane, *a*, is also visible on the temporal side (really it runs all the way round, lying on the ciliary body, and on the temporal side it connects the ciliary body with the margin of the lens). By the traction produced by this membrane the ciliary body and in part also the chorioid have been detached from the sclera; on the nasal side, the detachment of the chorioid extends particularly far backwards. There is, furthermore, a detachment of the retina extending from the ora serrata to the papilla. The latter on account of its being markedly swollen extends far into the vitreous cavity. The lens is opaque and has a depression at *b* on its posterior surface, because the lens capsule had been injured in this spot by the foreign body and hence the posterior cortex of the lens had been to a certain extent absorbed in this situation. Close to this spot there lies in the vitreous the inspissated mass of pus, *c*. The latter is separated by a delicate membrane from the adjacent vitreous which is scarcely infiltrated at all. It is this abscess of the vitreous that gave the deep yellow reflex.

FIG. 338.—ABCESS OF THE VITREOUS. VERTICAL SECTION. The patient had suffered a rupture of the cornea from the thrust of a cow's horn. The rupture ran through the middle of the cornea. Inflammation set in and the eye became softer, lost its perception of light, and was, therefore, enucleated one month after the injury. The cornea at *a* has an indrawn depression, corresponding to the scar at the site of the rupture. The scar is continuous behind with an exudation membrane, *b b*. This membrane, which is a product of the inflammation of the ciliary processes, encloses the remains of the lens and forms a tough diaphragm separating the aqueous chamber from the vitreous cavity. At its upper side, *o*, the iris is adherent by its pupillary margin to the exudation membrane but on its posterior surface it is free and hence is driven forward by the accumulated aqueous. Below, at *u*, the iris is adherent by its whole posterior surface to the exudation membrane. Behind the membrane lies a large abscess of the vitreous which is shut in by the membrane in front, by the retina behind. The retina is everywhere detached and where it bounds the abscess is thrown into many folds. The chorioid remains attached everywhere.

above, it starts from the latter. There is then together with the suppuration in the vitreous a purulent iritis (*endophthalmitis septica*). In such cases the suppuration in the vitreous cannot be recognized with the same certainty as in the case of a simple abscess of the vitreous, because exudation into the anterior chamber (a turbidity in the aqueous, a hypopyon, a pupillary membrane) often interferes with a view into the deeper parts. And yet it is in these cases, which externally look like a violent suppurative irido-

cyclitis, that it is particularly important to determine whether the purulent inflammation is present in the posterior segment of the eye also, because upon this point depend both prognosis and treatment. Indeed, in case of suppuration of the vitreous the eye is lost, and if we are dealing with traumatic cases, enucleation is indicated. In cases in which the exudate in the vitreous cannot be seen directly, the diagnosis must be based on a test of the light perception (see §185). If this is nearly or quite abolished, it is a proof that the retina is no longer functioning, which may be taken as evidence that there is a detachment or a purulent inflammation of the latter (cf. § 720).

It must be admitted that this evidence cannot be regarded as absolutely conclusive. Eyes with absent or nearly absent light perception may on enucleation show little change in the retina or optic nerve. Furthermore, in a number of cases of this sort in which enucleation has not been performed the light perception which has been almost lost has been restored. It is conjectured that in these cases the diminution in light perception has been caused by temporary paralysis of the retina and optic nerve through the action of toxins which have entered these parts by diffusion from the exudate in the vitreous.

The outcome of endophthalmitis septica may be gradual shriveling of the eye just as in abscess of the vitreous. In the severest cases, on the other hand, the pus accumulated in the eye breaks through to the outside, and these cases are denoted as—

755. (3) *Panophthalmitis.* The œdema of the lid increases so much that the physician can scarcely separate the lids. The appearance of the eyeball varies. If the suppuration originates in changes in the anterior division of the eyeball—e. g., from corneal ulcers, or from injuries—the symptoms of these conditions appear in the foreground, while those cases in which the inflammation has taken its origin in the deeper parts, show the picture, portrayed above, of a yellow exudate behind the lens. In any case, whatever the appearance of the eye, there is present, as one of the chief symptoms of panophthalmitis, marked protrusion of the eyeball (exophthalmus) with abolition of movement. The pain can scarcely be borne, and not infrequently annoying photopsiæ also are present. There is fever, and vomiting frequently occurs, especially at the beginning of the disease. These symptoms keep on until the purulent exudate in the interior of the eye makes an exit for itself by breaking through the sclera. Perforation takes place in the anterior division of the sclera. The conjunctiva is seen to bulge forward at some spot, showing the yellowish, discolored sclera through it, until finally sclera and conjunctiva are perforated and the purulent contents of the eyeball are slowly extruded. After perforation has occurred the pains soon cease, and the eye becomes softer and ultimately shrinks up to a small stump (*phthisis bulbi*). It takes at least six to eight weeks for this result to be attained and for the eye to become perfectly free from pain.

Panophthalmitis, therefore, is an endophthalmitis which goes on to the formation of an abscess, and is characterized by the development of two additional symptoms—protrusion of the eyeball and purulent perforation of the envelopes of the eye. The protrusion is due to the extension of the inflammation to the tissues behind the eye and above all to Tenon's capsule;

and just as the lids and conjunctiva are œdematous, so also there develops a marked inflammatory œdema behind the eye and in consequence of this a protrusion of the ball.

Most cases of *panophthalmitis* are caused by injury. If the latter is of such a character that the eye is extensively opened, the purulent exudate may be discharged through the wound. This is true of the cases of panophthalmitis after extensive suppuration of the cornea. In all these cases the pus in the interior of the eye is not under any pressure, and hence the disease runs a mild course with but little pain and slight inflammatory symptoms. The total course, too, is comparatively quick, since the condition can proceed without waiting for the sclera to be perforated, which always requires a long time. Yet not infrequently even in these cases, in which the purulent exudate pushes out through the wound, perforation of the sclera by pus is also seen to take place as usual. Panophthalmitis after severe injuries must, from a certain point of view, be regarded as a more favorable outcome than a plastic irido-cyclitis. The former to be sure, causes more violent pain and leads to a greater degree of shrinking of the eye; but when it has run its course the patient has a lasting respite. Plastic irido-cyclitis, on the other hand, often for years produces after-attacks of inflammation, and may also give rise to sympathetic disease of the other eye, if the patient does not submit to enucleation at the proper time.

756. Etiology.—The three categories of purulent inflammation of the interior of the eye are essentially the same process and differ simply in extent (in one instance being confined to the posterior division of the eye, in the other affecting the whole interior of the ball) or else differ in the intensity of the inflammation (termination in one case being in gradual shriveling, in the other in suppuration). Hence the three categories are in no way sharply differentiated, and there are transition forms between them.

So, too, the etiology of all three is alike. It is always a case of an infection of the interior of the eye by pyogenic germs. The infection may originate from the outside or have its source in the body itself.

Infection *from the outside* (*ectogenous infection*) occurs—

(*a*) Most frequently from penetrating injuries of all kinds. In this category belong unsuccessful operations.

(*b*) From the passage of suppuration from without inward, in the case of perforating ulcers of the cornea, and from prolapses of the iris that are covered with pus.

(*c*) As a process starting from a cicatrix of the cornea with or even without incarceration of the iris, when the cicatrix is thinned. Under this head belong the incarcerations of the periphery of the iris, that not infrequently remain after cataract operations. Infection takes place in these cases from the germs penetrating through the thin cicatrix into the tissue of the incarcerated iris, and then traveling in the latter backward into the eye. The starting point for the infection of the old cicatrix may be afforded by inconsiderable lesions of the epithelium covering the cicatrix, or by sudden stretching or bursting open of the latter (see also § 609).

Infection by carriers of infection, which arise from the *organism itself* (*endogenous infection*), takes place—

1. Through embolism, septic substances from a focus of suppuration getting into the circulation and becoming arrested in the vessels of the cho-

rioid or retina. In this way *metastatic ophthalmia* develops. This is one of the symptoms of pyæmia, and most frequently of that form which makes its appearance in the puerperal period as puerperal fever. Moreover, we often find metastatic purulent chorioiditis in meningitis, and particularly in cerebrospinal meningitis. These cases are observed chiefly in children, and are distinguished by their comparatively mild course, so that in rare instances actually some small degree of sight is still retained.

2. By transfer of inflammation from behind forward in phlegmons in the orbit and in thrombo-phlebitis of the orbital veins.

3. By direct transmission of inflammation from the meninges to the eyeball along the sheaths of the optic nerve. This occurs in certain cases of meningitis. But most cases of suppurative chorioiditis due to meningitis are metastatic in origin (see above).

Metastatic ophthalmia may be unilateral or bilateral. Cases of the former kind generally afford a better prognosis as far as the pyæmia is concerned, and particularly so, when no metastases are observable, except the one in the eye. The prognosis of the bilateral cases, on the other hand, is extremely bad, even for life itself.

Many cases of panophthalmitis that develop suddenly, and to all appearances spontaneously, should perhaps be regarded as one of the symptoms of a pyæmia whose point of origin is undiscoverable. In children, suppuration of the umbilical cord, and sometimes also vaccination, may give rise to pyæmia with metastatic ophthalmia. Metastatic ophthalmia may also be caused by gonorrhœa (Von Hippel).

Besides occurring in pyæmia, a purulent chorioiditis, doubtless also of metastatic origin, occurs in rare instances in other acute infectious diseases, such as typhus and typhoid fever, variola, scarlet fever, anthrax, influenza, ulcerative endocarditis, diphtheria, erysipelas, pneumonia, and Weil's disease.

For the exciting germs of endophthalmitis, see §§ 98, 99.

757. Anatomical Changes in Purulent Chorioiditis.—In *ectogenous* inflammation the pyogenic agents act primarily on the surface of the membranes that line the vitreous cavity, i.e., the ciliary body and retina. The superficial layers of the ciliary body are densely infiltrated, while the ciliary muscle which lies deeper is commonly but little changed. An exudate lies on the surface of the ciliary body (Fig. 339, *b b*) and from it extends into the saucer-shaped depression between the lens and vitreous. In the retina, too, it is primarily the inner layers that are infiltrated with emigrated leucocytes, these being particularly aggregated about the retinal veins which they surround with dense envelopes consisting of cells (purulent retinitis). The optic-nerve head is also swollen and infiltrated. From the retina the exudate passes out upon its inner surface, which in severe cases is often covered to a large extent with a layer of pus (Fig. 339, *e e*). Very soon the infiltrated retina wrinkles (in Fig. 339 at a point near the outer margin of the optic nerve) and becomes detached over a progressively larger area. The infiltration of the retina and of the papilla and the detachment of the retina account for the rapid diminution and ultimate extinction of light perception in the cases in which the inflammation gets a foothold in the vitreous cavity.

The chorioid is protected primarily by the retina against the action of the inflammatory agents that are present in the vitreous. If the retina becomes detached early, so that now subretinal liquid separates the chorioid from the inflamed retina, the chorioid may remain exempt from severe inflammation. If, on the other hand, the retina remains for a long time lying on the chorioid, the inflammation passes over to the latter; and the same thing happens when in the case of an especially severe inflammation the retina in places undergoes necrotic disintegration and the inflammatory agents can then act directly on the chorioid. The inflamed chorioid is densely infiltrated with leucocytes, and a free exudate collects both on its inner and on its outer surface. The outer exudate lies in the subchorioidal space, and hence produces a detachment of the chorioid.

In *metastatic ophthalmia* the pyogenic germs start from the blood-vessels, into which they have been carried by embolism, and act upon the surrounding tissue. About the embolic spot in the retina or chorioid a focus of inflammation is formed which rapidly leads to the formation of pus and to necrotic disintegration of the tissue. From this primary abscess the inflammation extends with great rapidity so that in the shortest possible time the whole interior of the eye is involved in the suppuration, and the pus soon breaks through to the outside of the eye (panophthalmitis). In exceptional cases it happens that the inflammation remains confined, the abscess which develops in the retina or chorioid becoming encapsulated, and the anterior division of the eye remaining entirely or in large part exempt from the inflammation (see § 753). The emboli which produce a metastatic inflammation are as a rule of a capillary character, and they affect the vessels of the retina more often than those of the uvea.

Both in atrophy and in phthisis the *optic nerve* becomes subsequently altogether atrophic, so that finally it forms a thin strand consisting simply of connective tissue. This takes place in obedience to the general law that nerve trunks atrophy when their terminal expansions are destroyed (ascending atrophy).

758. Treatment.—Treatment can not save the sight in suppurative chorioiditis. In cases in which the inflammation is not too violent, we confine ourselves to alleviating the pain by means of moist warm compresses and narcotics, until the shriveling eyeball becomes gradually destitute of sensation. If the pain is very severe, it is better to relieve the patient of a long and painful malady by early enucleation, especially when the disease threatens to go on to panophthalmitis. Enucleation is unconditionally indicated in all traumatic cases, since then it is a question not simply of cutting short the disease, but of preventing sympathetic ophthalmia. But when there is a fully developed panophthalmitis, enucleation is regarded as unsafe.

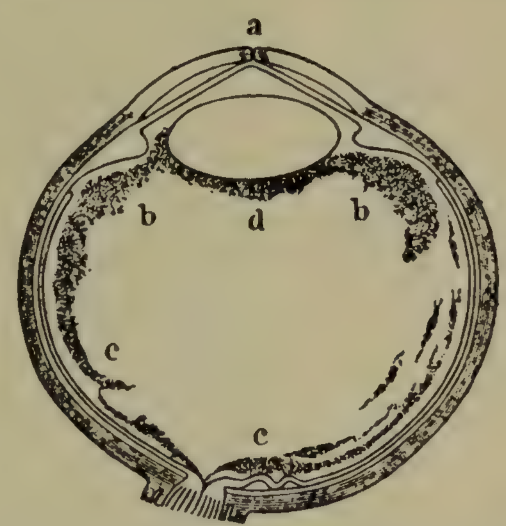


FIG. 339.—PURULENT INFLAMMATION OF THE INNER COATS OF THE EYE. HORIZONTAL SECTION.

Seven days previous to the examination a piece of iron had flown into the eye of an eighteen-year-old boy as he was watching a smith. In the cornea there is a perforating wound, *a*, to which runs the iris. A purulent exudate covers the inner surface of the membranes lining the vitreous cavity, namely, the ciliary body, *b b*, and the retina, *c c*. From the ciliary body the exudate, *d*, extends over the whole posterior surface of the lens. The ciliary body and retina are densely infiltrated, while the chorioid is scarcely changed. Near the entrance of the optic nerve the retina is beginning to detach under the form of two folds.

In these cases enucleation may be followed by a fatal purulent meningitis. It is assumed that by the operation the blood and lymph passages in the orbit are freely opened, and thus made accessible to infection. Whether the scooping out (evisceration) of the suppurating eye is less dangerous is questionable, since cases of death after this operation have also been observed (Schulek). But it must be noted, that fatal meningitis has succeeded a panophthalmitis without any operative interference; and Gradle states that in no reported case of fatal meningitis after enucleation has autopsy shown that the infection took place by continuity, so that here also probably it was a question of metastasis which would have occurred in any event.

Instead of enucleating in panophthalmitis we usually confine ourselves to opening the eye in its anterior portion, either by abscising the cornea or by making a free incision in the sclera. In this way the eyeball, which is tightly distended by the exudation, is freed from tension, the discharge of

the suppurating contents of the eye is accelerated, and thus the pain and th progress of the disease are cut short. When the eye at last becomes shrunke it usually remains quiescent and also admits of an artificial eye being wor over it. Should, however, secondary inflammatory symptoms set in, a they may exceptionally do, in the shrunken eye, its enucleation is indicate

II. Atrophy, Detachment, and Rupture of the Chorioid.

759. **Atrophy.**—Atrophy of the chorioid besides resulting from inflammation ma be due to degeneration. Such atrophy, affecting for the most part the macula or a zo around the disc, occurs as result of myopia (see § 745), glaucoma (halo glaucomatosus or senile changes (circumpapillary atrophy, macular disease—see § 743). Atrophy the chorioid may also be associated with degeneration and atrophy of the retina, as i atrophia gyrata and in Stargardt's familial macular degeneration, which is regarded as form of tapeto-retinal degeneration (see § 795). Occasionally we find a primary atroph of the pigment epithelium and the chorio-capillaris, so that, owing to the exposure of th chorioidal stroma, the red fundus becomes either browner in tint or a lighter-red tha normal, and a circumscribed well-defined patch is formed in which the large chorioida vessels stand out clearly and are accentuated by white lateral streaks which lie upo them and which in advanced cases merge over them, so that the vessels appear creamy white. There is a scotoma in the field of vision corresponding to the defe in the chorioid. Wood calls the condition *primary chorioidal sclerosis.* Syphilis, arteri sclerosis, nephritis, and menstrual disturbances have been assigned as causes, but th etiology is obscure.

760. **Detachment of the Chorioid.**—This is frequently found in enucleated eye In shrunken eyes, the chorioid—and the ciliary body, too—are very frequently found t be detached by the exudates which are present in the vitreous chamber, and whic exert a centripetal *traction* in all directions (*c*, Fig. 311). Then owing to negativ pressure serous liquid collects beneath the chorioid. A detachment of the retina, usuall total, is never wanting in these cases. Since we are dealing with eyes which are alread blind, the detachment of the chorioid has a practical interest only in so far as it produce traction upon the ciliary body; for this may induce irritation in the blinded eye.

Another mode of detachment is the active *propulsion* of the chorioid from th sclera by blood which is poured out beneath the chorioid. This takes place as a resul of rupture of the ciliary arteries when in an eye (usually one affected with old increase c tension or with degenerated blood-vessels) the intra-ocular pressure sinks to zero in con sequence of perforation (due to corneal ulcer, injury, or operation). The hæmorrhag may be so great that the inner coats of the eye are in large part expelled from the eye ball (expulsive hæmorrhage). In this way an eye that still retains sight may be de stroyed. Detachment may also be produced by exudation under the chorioid or by th development of a sarcoma in its outer layers.

It is a rare thing to see with the ophthalmoscope a detachment of the chorioid in a eye which still retains sight and in which the media are transparent. The detache chorioid appears under the guise of a dark prominence projecting into the vitreous. I is distinguished from a detachment of the retina by its dark color and its smoothnes from a tumor by its translucency when transillumination is used. This sort of detachmen is most frequently produced by serous fluid, and, in fact, by aqueous which percolate from the anterior chamber beneath the chorioid through a small rent in the attachmen of the ciliary body. Hence, we find, at the same time, that the anterior chamber is eithe shallower or quite obliterated. According to some the detachment is caused by serou exudation from the chorioidal vessels. It occurs not infrequently immediately after a extraction of cataract or an iridectomy for glaucoma, also quite frequently after Elliot' trephine operation, and still more frequently after Lagrange's sclerectomy (Barkan). I gives a good prognosis, since usually the chorioid becomes reattached to the sclera.

761. **Rupture of the Chorioid.**—This is produced through the action of some blun body upon the eyeball (contusion). Immediately after the injury the blood extrav-

asated into the vitreous usually prevents a clear view of the interior of the eye. After the absorption of the blood the rupture of the chorioid, which ordinarily lies in the neighborhood of the papilla, and most frequently to the outer side of it, is discovered. Sometimes there is only one, sometimes there are several, lacerations. They form long, yellowish-white streaks, as the edges of the laceration in the chorioid gape and allow the white sclera to be seen between them (Fig. 340). The streaks generally have a curved shape, with the concavity toward the papilla; they are broadest in the centre, and taper off to a sharp point at the ends. Their edges have an irregular black coloration, due to proliferation of the pigment. The retinal vessels run without any change over the streaks, a proof that the retina is unruptured.

Fig. 340.—Rupture of the Chorioid. Right Eye. (After De Wecker.)

There are one large and four small ruptures in the lower part of the chorioid. The large rupture appears under the guise of a slightly curved white band with jagged, somewhat pigmented borders. The small ruptures lie concentric with the large one and between it and the papilla. Over all of them the retinal vessels run unaltered.

Ruptures producing very large, irregular white patches in the fundus (so-called *chorio-retinitis proliferans traumatica*) are caused by gunshot injuries of the orbit (hence also called *chorio-retinitis sclopetaria*[3]). These ruptures are *direct*, i.e., produced at the point of application of the contusing force, while the ruptures previously described are usually indirect. The patches represent large lacerations filled or overlaid with new connective tissue, which by its cicatricial contraction causes other changes such as wrinkling and partial detachment of the retina. The patches are often covered with blood which takes a very long time to absorb.

III. Tuberculosis of the Chorioid.

762. Tuberculosis of the chorioid occurs either in small, single or multiple, but discrete foci or in the form of a single large mass.

(*a*). One type of the *discrete* form is *disseminated* or *miliary* tuberculosis of the chorioid which was first described by Jäger. Small ill-defined patches of yellowish or pale-reddish color are seen in the fundus. These even within a short period of examina-

[3] From new Latin *sclopetum*, a gun.

tion—within a few days—grow larger without, however, attaining a size of more than one-third of the optic disc; and at the same time new patches may make their appearance in the fundus. By this rapidity of growth the affection is distinguished from chorioiditic spots, which change very slowly; besides the pigment changes, so frequent in chorioiditis, are wanting in tubercles of the chorioid. The tubercles occupy chiefly the posterior division of the chorioid. Ordinarily only a small number are present, although sometimes as many as twenty or thirty of them can be counted in the eye. Anatomical examination has proved that the spots seen with the ophthalmoscope correspond to nodules of a mean diameter of 1 mm., which possess the typical structure of tubercle nodules (Manz).

Miliary tuberculosis of the chorioid forms one of the symptoms of general miliary tuberculosis (Cohnheim). In a doubtful case of this disease its discovery may help to establish the diagnosis.

In chronic tuberculosis we find not infrequently a single chorioidal focus, usually in the macular region or close to the disc. This may have the size and course of a simple miliary nodule or may form larger patches two or three times as big as the disc and somewhat elevated. These patches become absorbed later leaving an atrophic or pigmented patch in the chorioid. Sometimes the extra-ocular tuberculous lesion that causes the chorioidal focus is not obvious, so that the latter may become of diagnostic value in that it indicates the existence of a latent tuberculosis. Likewise tuberculous, in some cases at least, would seem to be the isolated patches found in chorioiditis juxtapapillaris (see § 743).

In still other cases the discrete foci assume the character of an ordinary disseminate chorioiditis, the tuberculous nature of which is shown by the fact that tuberculin injections cause a focal reaction.

(*b*) *Solitary* or *conglobated* tubercle of the chorioid appears under the guise of a neoplasm. With the ophthalmoscope a rather large, light-colored tumor is seen in the chorioid; it is an argument for its tuberculous nature if smaller light spots (tubercle nodules) are found in the chorioid in its vicinity. The tumor may afterward grow through the sclera to the outside, and there break down. The microscope shows that it consists of a great number of smaller miliary nodules, which have coalesced to form a single, fairly large tumor. In the centre of the latter, caseation takes place. The solitary form of tuberculosis of the chorioid is a very rare disease, pre-eminently affecting young people. It runs a chronic course, and accompanies chronic tuberculosis of the internal organs, especially of the brain. There are, however, cases in which no tuberculous focus other than that in the eye can be clinically demonstrated. The prognosis of solitary tubercle of the chorioid is bad, since the eye in any case is lost, and in most cases also life is endangered through the presence of tuberculous diseases in other parts.

In suspected tuberculosis of the chorioid, we may use injections of tuberculin (§ 140) to establish the diagnosis.

For *treatment* we must first initiate general measures directed against tuberculosis, especially tuberculin injections, with which not infrequently we are successful; but if we see that the tuberculous focus is increasing in spite of these measures and that blindness is setting in, enucleation is indicated.

IV. Tumors of the Chorioid

763. The chief malignant tumor occurring in the chorioid is *sarcoma*, which in most cases is pigmented (melano-sarcoma). In the development of the tumor four stages can be distinguished.

In the *first stage* the tumor is small, and manifests itself only in ophthalmoscopic examination by detachment of the retina at the site of the tumor. The patient notices a disturbance of vision in the shape of a defect in the visual field, corresponding to the site of the tumor. Afterward the detachment of the retina becomes total (Fig. 341, *N*), and thus the eye, which externally still looks normal, becomes completely blind.

Sarcomata of the chorioid consist either of round cells or of spindle cells, or are tumors made up of a mixture of both. They are almost always pigmented (melano-sarcomata); non-pigmented sarcomata (leuco-sarcomata) are among the rarities. Very often they contain many and wide blood-vessels. Sarcomata develop from the external layers of the chorioid (layer of large and of medium-sized vessels) and grow inward toward the vitreous space, pushing the retina before them. In the beginning, the retina lies everywhere in contact with the surface of the tumor, so that with the ophthalmoscope a sharply circumscribed gibbous detachment of the retina is found, rising steeply from the fundus. This, for obvious reasons, does not fluctuate. Through the retina we can often recognize the vessels of the chorioid or of the tumor and also the yellow or brown color ofthe latter. In this case the *diagnosis of sarcoma* is easily made. But afterward, in consequence of the disturbance of circulation in the chorioid produced by the tumor, an accumulation of fluid takes place between the chorioid and the retina. The latter is thus detached over an area greater than that occupied by the tumor, and does not permit the latter to be seen through it any more; ultimately, the detachment becomes total (Fig. 341). In this stage, since the detachment of the retina has lost its characteristic appearance the diagnosis can not for the most part be made with certainty. The tension of the eye affords one diagnostic point, being usually diminished early in simple serous detachment of the retina, while in detachment due to a tumor it is at first normal and afterward increased (Von Graefe). It is an additional argument for the existence of sarcoma if the anterior

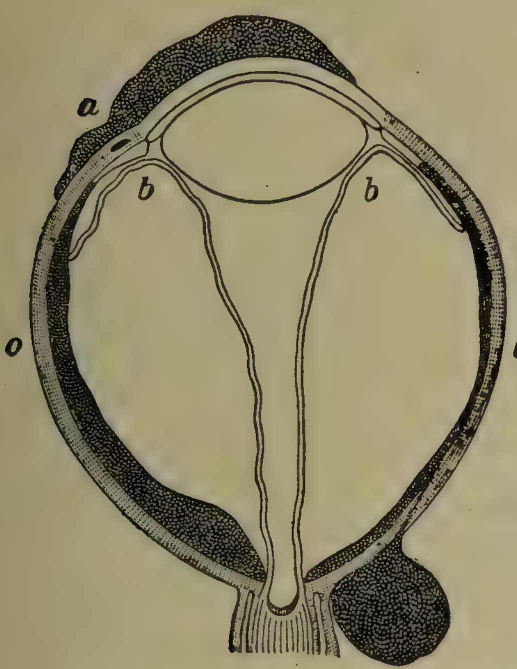


FIG. 342.—SARCOMA CHORIOIDEÆ DIFFUSUM. VERTICAL SECTION THROUGH THE RIGHT EYE.

The patient, who was fifty-four years old, had been blind in this eye for years from an opacity in the cornea. The eye became inflamed from time to time and half a year previous had begun to take on a black color. The eye was enlarged in all its parts and the cornea was covered by a granular brownish-black deposit which only at the nasal and lower side left a narrow marginal portion of the cornea free. In the section made through the eye we see that the latter as a whole is elongated and tapers off into a somewhat oval extremity behind—two signs of high myopia, which was also present in the patient's other eye. The cornea is covered with a flat dark pseudoplasm which, at the upper side, *o*, is continued on to the adjoining sclera, but at the lower side, *u*, still leaves some part of the cornea free. The cornea beneath the tumor is scarcely altered; only in a few spots have the sarcomatous masses destroyed Bowman's membrane, but nowhere have they penetrated into the deeper parts. Near the upper border of the cornea, at *a*, there lies in the thickness of the sclera a little black island of sarcomatous tissue representing the cross section of an anterior ciliary vein. This vein is filled with sarcoma masses and constitutes the way by which the intra-ocular pseudoplasm had grown through to the outside. In the interior of the eye the chorioid and the flat portion of the ciliary body are everywhere uniformly sarcomatous; a coating of black pseudoplasm also covers the floor of the excavation in the optic nerve. Adjoining the lower side of the latter lies a second extra-ocular nodule from which a filament of black sarcoma tissue runs through the sclera obliquely forward and inward as far as the chorioid. At this spot the intra-ocular sarcoma has grown out through the sclera along a ciliary nerve. In the interior of the eye, furthermore, are found the evidences of a long continued and very marked increase of tension—namely pushing of the iris and lens against the cornea, atrophy of the ciliary processes, deep and total excavation of the head of the optic nerve.

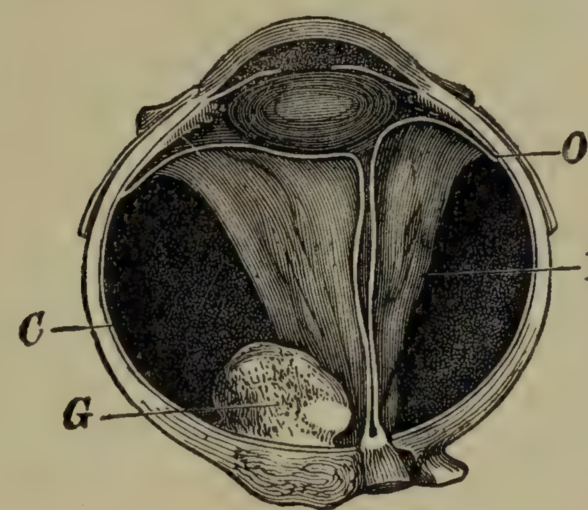


FIG. 341.—SARCOMA OF THE CHORIOID. (After Leber.)

The tumor, *G*, rises from the chorioid, *C*, which everywhere lies in contact with the sclera. The retina, *N*, on the contrary, is detached entirely from its bed under the form of a folded funnel. It retains its connection only with the papilla behind, and with the chorioid along the ora serrata, *O*, in front.

ciliary veins are found markedly dilated upon one side or the other. These dilate veins indicate that the sarcoma is situated in the chorioid, and, in the area affecte prevents the blood in the forepart of the uvea from flowing out through the vasa vorti cosa, so that it has to make its way through the anterior ciliary veins. As the intra ocular pressure later on steadily increases, the detachment of the retina often assumes characteristic appearance. The retinal protrusions are pushed against the posterio surface of the lens and against each other by the increased pressure in the subretina space; they flatten themselves upon each other and often include only a narrow Y shaped furrow between them. There are, it is true, serous detachments which also show this appearance. These are those rare cases in which serous detachment is associate with increase of tension. Such cases are hard to distinguish from tumor of the chorioid To make the right diagnosis in such cases we may call in the aid of transilluminatio (§ 401). Or we may employ puncture. This is made with a fine needle, which i plunged deep in through the sclera at the spot where the tumor is thought to be. If th needle enters a tumor, it is not freely movable, as would be the case if its point lay in th vitreous or in subretinal fluid. If in spite of these diagnostic aids we are still in doubt enucleation of the eye, which would be blind anyhow, is unconditionally indicated.

In the rare *diffuse sarcoma* of the chorioid the new growth does not begin as a cir cumscribed rounded tumor, but as a uniform degeneration, which is diffused ove wide areas (Fig. 342) and in which often the ciliary body and even the iris ar likewise implicated.

764. The *second stage* in the development of sarcoma is marked b increase of tension. Externally the eye presents the appearances of conges tive glaucoma (§ 838). There is marked injection of the eyeball, the corne is dull and clouded, the anterior chamber shallow, the iris discolored, th pupil dilated and immobile, and the tension of the eye is noticeably elevated If the media are sufficiently clear, the gray reflex of the detached retina ca be seen deep down behind the pupil. Later on, the lens becomes clouded so that the clinical picture of glaucoma absolutum with cataracta glaucom atosa is produced. From the time when the symptoms of inflammator glaucoma set in, the patient suffers with pain; very frequently it is by thi that he has his attention first called to his trouble. Since the picture pre sented by the affected eye corresponds completely to the complex of symp toms of congestive glaucoma, the correct diagnosis in this stage is to b made with difficulty, or not at all.

If the detachment of the retina is not already total, it becomes so now, and the ey becomes completely blind. The point of time at which the glaucomatous attack sets i does not depend directly upon the size of the intra-ocular tumor. The increase in ten sion does not arise from the fact that the tumor occupies a certain space in the interio of the eye, for this call for additional space is compensated for by a corresponding de crease of the vitreous. On the contrary, the increase in tension is based upon the con gestion which the tumor produces in the veins of the chorioid, and by which increase transudation of fluid takes place into the interior of the eye. Elevated tension is, there fore, often seen with quite small tumors, while at another time the tumor may hav already filled up a large part of the eye without exciting the symptoms of glaucoma When the glaucomatous attack has set in, the eye looks as if it had been blinded b primary glaucoma, and the diagnosis cannot be made with certainty. The existence o a sarcoma will be suspected if the patient states that the eye was already entirely blin before the outbreak of the inflammation, for in primary glaucoma blindness usually doe not precede the attack, but follows it. Besides, we examine the second eye; if one ey is completely blinded by primary glaucoma, the second eye will rarely be found quit normal.

In rare cases the second (inflammatory) stage of tumor growth does not present the symptoms of glaucoma, but of a severe *irido-cyclitis*, which because the inflammation is especially violent, is often accompanied by chemosis and exophthalmus. Such inflammation is apt to occur when the intra-ocular tumor owing to inadequate nutrition has become largely or wholly necrotic. As a result of the irido-cyclitis, the eye becomes softer and shrinks up so far as the tumor contained in it permits. The growth of the latter is thus retarded for some time—a fact which does not prevent epibulbar tumors and metastases from developing. Sympathetic ophthalmia may also develop (page 684).

765. The *third stage* is that of the growth of the tumor upon the outside. The symptoms are different, according as the tumor breaks through the sclera in its anterior or in its posterior division. In the former case, dark, hard prominences are seen developing in the circumference of the cornea, and the diagnosis can readily be made. If, however, the tumor first grows through the sclera posteriorly, the nodules of the tumor are invisible, and do not give evidence of their existence until later, through the gradually increasing protrusion of the eyeball (exophthalmus). As soon as the tumor has broken through the envelopes of the eyeball to a sufficient extent, the pain usually abates, since the great tension in the eyeball then ceases. But, to make up for this, the extra-ocular masses of tumor, freed from the intra-ocular pressure that constrained them, grow so much the faster. First the orbit is entirely filled by the tumor, afterward the latter projects from the orbit, as big as an apple or as the fist. From the orbit the tumor is continued to the neighboring parts, particularly to the brain. At its exposed portions the tumor ulcerates and gives rise to frequent hæmorrhages.

Growth of the mass exteriorly occurs before the tumor has yet filled the whole interior of the eyeball, and is effected by the gradual growth of the cells of the tumor through the sclera, the cells usually following out preformed passages. We hence find the tumor growing out along the points where the blood-vessels (anterior or posterior ciliary vessels or the vasa vorticosa) and the ciliary nerves penetrate the sclera (Fig. 342), or more rarely, along the optic nerve or its sheaths.

766. The *fourth stage* is that of the generalization of the tumor by the development of metastatic nodules in the internal organs, most frequently in the liver.

Years usually pass before the sarcoma has run through the four stages just pictured. The first and second stages last a long time, while afterward the growth of the tumor becomes continually more rapid. The patient dies either from exhaustion, in consequence of the suppuration and hæmorrhage from the tumor, or succumbs to the extension of the latter into the brain or to the metastases in the internal organs.

The *metastases* in remote organs arise through embolism. The blood current detaches cells from the tumor and carries them into other parts of the body, where they develop into independent tumors. Local recurrences are scarcely to be apprehended if the growth was confined to the eyeball at the time of the operation. On the other hand, metastases occur even in cases in which enucleation was performed very early. Such metastases remain unobserved at the outset; and it may be five or ten years before they cause the patient's death.

767. The *prognosis* of sarcoma of the chorioid is absolutely unfavorable for the life of the patient if the eye is not removed early. But even then the prognosis is by no means to be regarded as perfectly favorable. Both local recurrences in the orbit and also metastases may develop after the removal of the eye, the germs for their development having been already scattered abroad, although, at the time when the eye was removed, they were too small to be demonstrable. Sarcoma of the chorioid is hence to be regarded as one of the most malignant of diseases—one which, in very many cases, ends in death. Sarcomata of the iris and ciliary body behave, in respect to their course and ultimate outcome, like sarcomata of the chorioid.

Sarcoma of the chorioid is a rare disease. It is found most frequently between the fortieth and sixtieth year: in childhood it is extremely rare. This gives a means of distinguishing it from the gliomata which spring from the retina, and which in part present symptoms like those of sarcomata, but which occur in childhood exclusively. A malignant neoplasm developing in the eyeball will, therefore, have to be regarded in all probability as a glioma in a child and as a sarcoma in an adult.

The *treatment*, as long as the neoplasm is still confined to the eyeball, consists in enucleation, which should be performed as early as possible. In doing it, we cut off the optic nerve as far back as possible, to meet the contingency that the degeneration has already passed over upon it. If the neoplasm has already grown out of the eyeball, everything diseased must be removed according to surgical rules. The surest method in this case is exenteration of the orbit—i. e., the removal of the entire contents of the orbit, together with the periosteum (§ 999).

Radium may be applied as a prophylactic after enucleation; and it may possibly help in case of local recurrence (Janeway).

768. Other Tumors of the Chorioid.—Cavernous angioma, endothelioma, perithelioma and melanoma have been known to occur as primary tumors of the chorioid in a few cases. *Melanomata* are benign tumors which greatly resemble small sarcomata. They can be distinguished from the latter by their uniform slaty color; their definite, somewhat feathered, outline; the absence of abnormities in the appearance of the overlying or adjacent retina; and the absence of pigmentary disturbances adjoining the tumor (Moore).

Carcinomata and also adenomata have been observed as a great rarity in the chorioid, but only as secondary tumors, as metastases from tumors in other organs (especially in the mamma or in the thoracic glands). Carcinomata are whitish, flat, disc-shaped growths. As they are always secondary, the prognosis is hopeless and the treatment palliative only. To distinguish them from aberrant forms of sarcoma which would require enucleation, we should carefully examine the chest for evidences of solidification indicating the presence of the primary tumor. If such is found, enucleation is contra-indicated (Greenwood).

V. Congenital Anomalies of the Chorioid

769. Coloboma of the Chorioid.—In this affection the ophthalmoscope shows a brilliant white area in the red fundus below the optic-nerve entrance (Fig. 343). This represents a circumscribed defect in the chorioid and usually also in the retina, in the confines of which the sclera lies exposed, and is hence visible as a white surface. Coloboma of the chorioid is frequently

found along with coloboma of the iris, and also with other congenital anomalies of the eye. Such eyes are often smaller than usual (microphthalmus).

Corresponding to the coloboma there is usually a defect in the visual field, and, moreover, central vision is often defective because the eye as a whole is not properly developed.

Coloboma of the chorioid is in a marked degree transmissible by inheritance, and that, too, not infrequently in conjunction with other congenital malformations of the body.

FIG. 343.—COLOBOMA OF THE OPTIC NERVE AND CHORIOID. FROM THE RIGHT EYE OF A FOURTEEN-YEAR-OLD GIRL. ERECT IMAGE. (After Casper and Krüger.)

The disc appears about nine times as large as in the normal state, and lies considerably below the level of the adjoining retina. An upper (yellowish) and a lower (gray-colored) portion can be made out in it. From the former rise the central vessels, which are abnormal in sending most of their branches upward. The lower (gray) portion of the disc shows several light-colored, ridge-like projections and but a few blood-vessels, although numerous blood-vessels emerge at its overhanging border and run out into the retina, evading the coloboma. The enlarged papilla is bordered above by a narrow atrophic crescent. The coloboma of the chorioid lies below the disc and somewhat to its temporal side. Its lower (anterior) border is not represented in the drawing. The coloboma is of brilliant white hue, is sharply defined and is placed somewhat deeper than the adjoining portions of the fundus. It shows a few blood-vessels and in spots a fine granular pigmentation.

Coloboma of the chorioid has the shape either of an oval, whose long axis corresponds nearly to the vertical meridian, or of an obtuse-angled triangle whose apex is directed toward the papilla. The peripheral border of the coloboma not infrequently presents a tapering prolongation extending toward the ciliary body. Even the smallest colobomata are much larger than the optic disc, and the large colobomata are so extensive that their anterior border can no longer be seen with the ophthalmoscope, because it lies too far forward. So, too, they may extend so far backward that they involve the papilla. The latter, in that case, is generally changed in shape and appearance—sometimes so much so that we can scarcely tell where it lies except for the intimation of its existence afforded by the place of origin of the retinal vessels. (See page 728).

The edge of the coloboma is sharply defined and commonly bordered by pigment. The coloboma itself is of a pure white or bluish-white hue, and displays here and there pigment spots and also vessels. The vessels are in part those that arise from the adjoining retina and chorioid, in part belong to the sclera, which lies exposed within the area

of the coloboma, and in part are seen to originate from the coloboma itself. The latter set must be regarded as posterior ciliary vessels. In eyes of this sort the retinal vessels often display an irregular course; not infrequently it looks as if they were trying to evade the coloboma, since they run along its borders instead of passing over it.

The surface of the coloboma lies deeper than the rest of the fundus, and often presents channeled depressions or prominent ridges, as can be inferred both from the way in which the vessels bend and also from the parallactic displacement.

Cataract, generally of a complicated, inoperable character, frequently develops in eyes affected with coloboma of the chorioid. In one case it seemed to be due to detachment of the retina, the latter being adherent to the margin of the coloboma and thus subjected to traction (Fuchs).

In rare cases large, white, depressed areas have been observed not below, but to the outside of, the optic nerve, in the region of the yellow spot. These have likewise been looked upon as congenital malformations—*colobomata of the macula.* In exceedingly rare cases coloboma may also occur *above* the disc (Gifford).

Fig. 344.—Lower Half of an Eye with Congenital Coloboma of the Iris, Ciliary Body, and Chorioid.

In the iris, whose posterior surface is seen in the figure, is recognized the prolongation of the pupil, running out in a sharp point to the lower ciliary margin. In the corresponding portion of the ciliary body, the ciliary processes are wanting; the processes immediately adjoining this gap are higher and longer than the rest, and, as they diverge backward inclose a triangular, very darkly pigmented area. Still farther back there is found in the wall of the eyeball a deep excavation of oval form, whose edges are sharply defined and in part overhanging. Upon the floor of the excavation is seen the sclera, covered simply by a very thin, transparent pellicle, in which run several vessels. The posterior pole of the oval that is formed by the coloboma of the chorioid is directed toward the fovea centralis, *f.*

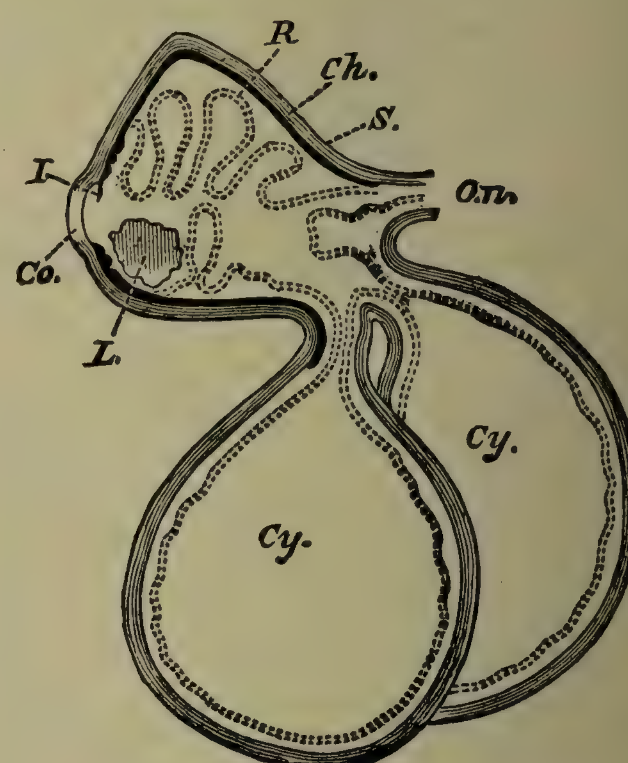


Fig. 345.—Diagrammatic Representation of a Microphthalmic Eye with Two Cysts Attached. (After Lang and Collins in Norris and Oliver.)

Co, cornea; *L,* lens displaced and shrunken; *I,* iris; *S,* sclerotic; *Ch,* chorioid; *R,* retina much folded; *O.n.,* optic nerve; *Cy,* cysts lined by retina.

Colobomata of the chorioid may be associated with a complete *coloboma of the retina*—the retinal vessels in this case being cut short off at the margin of the affected area. In other cases functional tests show that the retina is at least in part preserved over the colobomatous chorioid.

Colobomata may occur also in the *optic nerve.* Either a deep depression is found in the lower part of the latter or the entire optic-nerve entrance is enlarged to several times its usual size, and the vessels coming out from it are, as it were, forced apart (Fig. 343).

Anatomical examination of the eye affected with coloboma of the chorioid shows even upon an external view a protrusion of the sclera, situated below the optic nerve. This is the scleral protrusion first described by Ammon (see § 632). Corresponding to this, in the inner membrane of the eye, is the coloboma visible with the ophthalmoscope (Fig. 344). Within this, microscopical examination reveals, for the most part, only a thin pellicle composed of connective tissue, the remains of the fused chorioid and retina.

In some cases a coloboma produces a more considerable deformity, so that a sac-like protrusion develops from the lower side of the eyeball containing retinal tissue (Von Hippel,). In the more marked varieties of this anomaly of development, the eyeball itself remains quite small while the pocket develops into a pretty large sac. In this way develop the cases of *microphthalmus*, or of apparent *anophthalmus*, with the simultaneous presence of quite a large cyst situated in the lower lid, and glimmering with a bluish lustre through the skin of the latter. The cyst is filled with a serous liquid, is lined with rudimentary retina, and is connected by a process with the dwarfed eyeball (Fig. 345).

In some cases a microphthalmic eye is covered by unbroken skin instead of the normal lids (cryptophthalmus).

According to the views that long prevailed, a coloboma is *produced* by non-closure of the fetal ocular cleft and the interposition of a plug of mesoblastic tissue between the lips of the latter (Von Hippel). But there are a number of objections to this view nor has any other theory obtained universal acceptance.

770. Congenital Anomalies of Pigmentation.—Albinism consists in the absence of the physiological pigment. Albinos have yellowish-white flaxen hair and also white eyebrows and lashes. The iris is light gray, and appears reddish by transmitted light, while the pupil has a vivid red lustre. With the ophthalmoscope the blood-vessels of the retina and chorioid are seen with perfect distinctness running upon the almost white fundus, to which the papilla by its dark, grayish-red color offers a striking contrast (Figs. 194 A, and 196). Albinotic eyes are photophobic, and hence see better in the dusk; their visual acuity is always reduced; and nystagmus, frequently combined with quite a high degree of myopia or with strabismus, is constantly present. Albinism is congenital and often inherited. In albinotic eyes the pigmentiferous cells of the uvea and the retina are present just the same as in the normal eye, only they contain little or no pigment. All sorts of transition forms exist between complete albinism and normal pigmentation. In infants large areas of the fundus may be albinotic. Such areas may alternate with quite sharply defined islands of normally pigmented chorioid. The albinotic areas seem to occur especially in the periphery of the fundus, and this tendency to semi-albinism in the periphery may persist even in adult life.

In **Melanosis Oculi** both uveal and retinal pigment are excessively developed. The iris is brownish black and has a warty look; the fundus is slate colored; there are pigment spots on the sclera at the points of exit of the anterior ciliary vessels and sometimes at the limbus, on the posterior surface of the cornea, and in the optic nerve. The condition is usually unilateral.

771. Posterior Vortex Veins (Venæ chorio-vaginales).—These occur only in myopic eyes with a conus. From the margins of the latter extends a short branch which sends out finger-like branches terminating abruptly in a rounded end. These veins enter the sclera and pass into the dural sheath of the optic nerve (Salzmann).

CHAPTER XXVII

DISEASES OF THE RETINA

I. Vascular Disturbances of the Retina

772. Hyperæmia.—Hyperæmia of the retina may be either arterial or venous. The former accompanies all inflammations of the retina and the neighboring tissues, particularly the uvea, and is characterized by a pronounced distention and tortuosity of the arteries. Venous hyperæmia manifests itself by dilatation and great tortuosity of the veins, while the arteries are often thinner than usual. It is produced most often by plugging of the veins (see § 774) or by their compression. The compression is mostly located in the papilla, as in glaucoma, where the increased intraocular tension squeezes the veins down into the excavated papilla, or in optic neuritis, in which the swelling of the papilla compresses the veins. In orbital tumors it is the trunk of the optic nerve that is compressed. Venous hyperæmia also occurs as a symptom of a general venous congestion, particularly in heart disease or arteriosclerosis (see below), juvenile recurrent hæmorrhages (§ 667), and polycythæmia.

Beginners often fall into the mistake of considering a very vividly red eyeground hyperæmic. The aggregate tint of the eyeground, however, depends on its pigment content, and the background of slightly pigmented eyes is always much redder than that of the darkly pigmented ones. Moreover, great dilatation of the retinal vessels is in itself no certain proof of hyperæmia of the retina, for it often occurs physiologically, especially in the young. Exceptionally, too, cases of very marked congenital tortuosity of the retinal vessels occur in healthy eyes. (Cf. Pseudoneuritis, § 406). But in all these cases the condition of the retinal vessels in the two eyes is alike. Dilatation and tortuosity of the retinal vessels, therefore, can be regarded as certainly pathological only if unilateral or if confined to certain districts of the fundus, or if there is a faint cloudiness of the retina near the papilla, indicating permeation with serum, or, and more especially, if hæmorrhages can be made out.

Very rarely the retinal arteries and veins show sac-like dilatations (*aneurysms*) or varicosities. Miliary aneurysms of the retinal arteries occur, especially in old persons and most often in conjunction with extensive arteriosclerosis and atheroma (less often with heart disease and nephritis). There is a peculiar, usually very chronic and progressive condition, in which multiple aneurysms are associated with extensive infiltration of the retina—exudative retinitis (see § 788) (Leber).

Conditions of *arteriosclerosis* and high vascular tension are often accompanied by visible changes in the retinal vessels. As summarized by De Schweinitz these consist of: (1) Corkscrew appearance of arterial twigs, while the arteries from which they spring may be normal. (2) Flattening of veins, where crossed by arteries. Later on, this flat-

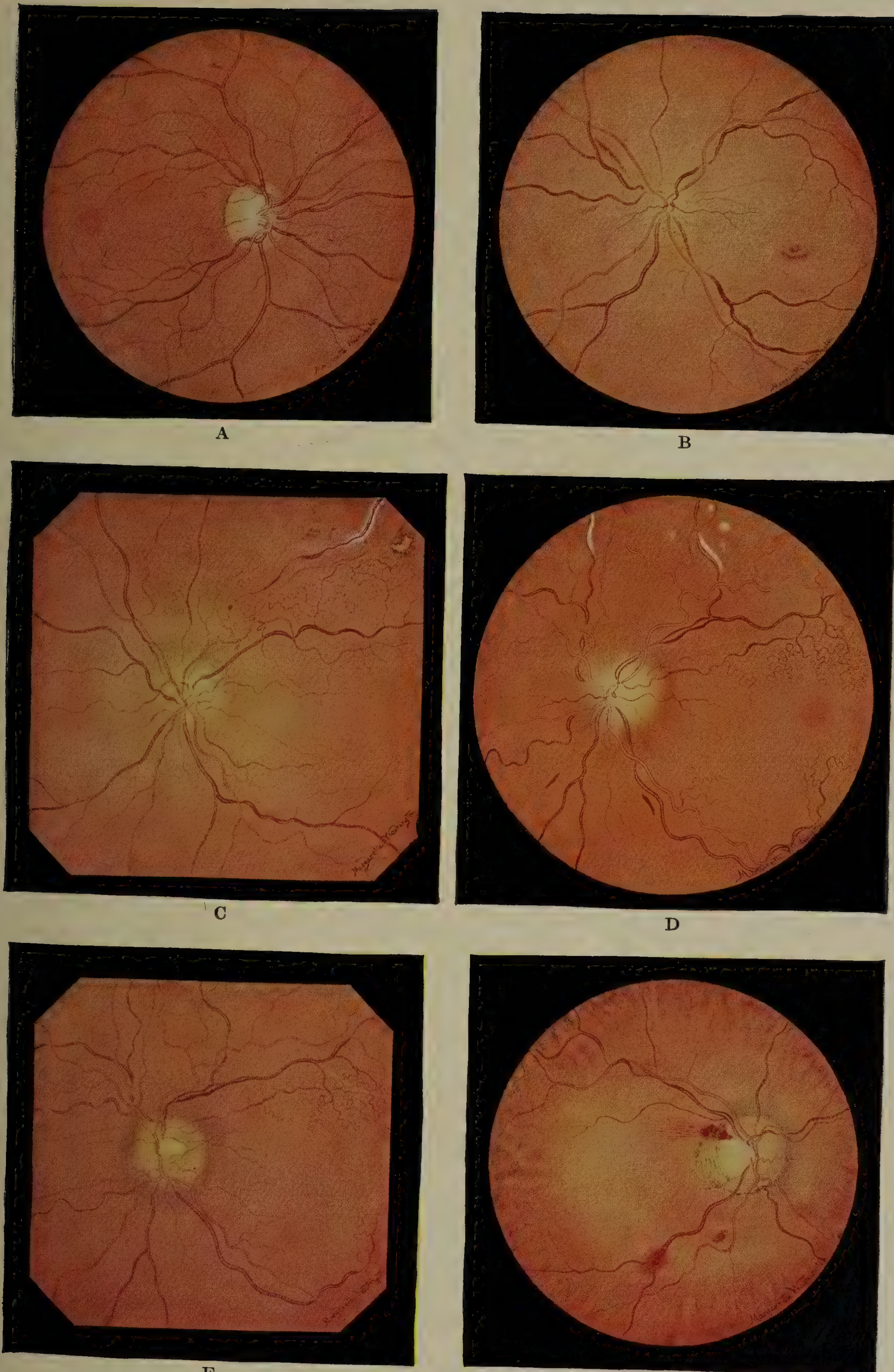


Fig. 346.—Changes in Arteriosclerosis. (After De Schweinitz.)

A, Normal fundus. B to F, successive changes occurring in arteriosclerosis including pallid arteries (B), later assuming a silver-wire appearance (C); indented veins (B, C) afterward showing ampulliform enlargements (D, E); corkscrew capillaries (C, D); corkscrew arteries and veins (D, E); perivasculitis (C, D); sclerosis of vessels (F); œdema of disc (B, C, D, E), hæmorrhages (C, F).

tening becomes an indentation and the veins beyond the point of compression become ampulliform. (3) A dull red congestion or actual œdema of the optic disc. (4) In the later stages the formation of white streaks along arteries and veins (perivasculitis) and hæmorrhages. These changes (see Fig. 346) are important because of their effect on the sight, because of their liability to give rise to retinal hæmorrhages, and particularly because they form a frequent and early sign of general arteriosclerosis, especially of the brain and kidney. Ocular arteriosclerosis may be associated with persistent asthenopia which is relieved only by general treatment (De Schweinitz).

Phlebitis and periphlebitis of the retinal vessels, with retinal hæmorrhage occurs in conjunction with *tuberculosis.*

Cyanosis of the retina may occur in congenital heart disease (patent foramen of Botallo), in polycythæmia, and from the action of certain poisons (dinitrobenzol).

773. Anæmia.—Anæmia of the retina may be of sudden or gradual development. The latter form is much more common. This sort of **Chronic Anæmia** occurs as a result of retinal atrophy and less often in consequence of profound general anæmia.

The retinal vessels then either become simply attenuated (Fig. 357) or surrounded by white bands, the result of thickening of the vessel walls, the blood column being simultaneously narrowed (*perivasculitis retinæ*). (See C and D, Fig. 346 and D and F Fig., 349). Ultimately the vessels may vanish altogether from the retina or be transformed into white bloodless strands. In *general anæmia*, especially the aplastic anæmia of childhood, the vessels are attenuated, and paler, the distinction between arteries and veins is less marked, the fundus assumes a yellowish-brown hue. The grave forms of anæmia in adults are marked not only by retinal anæmia, but often also by retinal hæmorrhages.

Acute anæmia of the retina is caused by sudden stoppage of circulation in the central artery or any of its main branches. Such stoppage may be due to embolism, thrombosis, spasm, compression, or breach of continuity in the artery.

Embolism of the central artery was first observed by Von Graefe. The patient is made aware of his disorder by sudden and complete blindness, which appears at once upon the obliteration of the artery. If immediately afterward an examination is made with the ophthalmoscope, the signs of an extreme arterial anæmia of the retina are found (Fig. 347; Fig. 349, A and B). The larger arteries are narrowed down to thin filaments, the smaller ones become invisible. The veins, on the other hand, are not markedly contracted except upon the papilla itself; the latter appears paler. Within a short time, often within a few hours, the retina, as it dies, loses its transparency. It becomes of an opaque milky white, most markedly so in the vicinity of the papilla and of the fovea centralis. Owing to this opacity the outlines of the papilla are obscured, and, on the other hand, fine ramifications of the retinal vessels, such as ordinarily are not visible with the ophthalmoscope, come into view in the vicinity of the fovea, where they stand out with great distinctness upon the white background.

At the centre of the fovea a vivid red spot stands out on the cloudy white background, because the inner layers of the retina and hence also the retinal cloudiness are lacking at this spot, and consequently the fundus appears of its normal red color, which looks even more pronounced from

contrast with the turbid white area surrounding it. In this region al
small retinal hæmorrhages sometimes occur.

After some days the retinal vessels fill up again. Then a peculiar phenomenon
sometimes observed. In some of the vessels (particularly the veins) the blood colun
appears broken up into short sections separated by clear interspaces, and the whc
column makes jerking movements, sometimes in the sense of the normal blood curren
sometimes in the reverse direction. In the following weeks the cloudiness of the reti

Fig. 347.—Embolism of the Central Artery, Developing Eight Days Previous to the Dati of Observation in a Woman Affected with Aortic Aneurysm.

The whitish haze over the retina obscures the outlines of the disc and the initial portions of tl vessels arising from it. The arteries are already better filled than they were, although they still a below the normal in this respect. The veins have a very uneven calibre, the latter in general increa ing toward the periphery. In the large veins, running upward and outward and downward and outwar the blood column is seen to be broken up into short separate sections. The vicinity of the fovea ce tralis is covered with a dense whitish haze, upon which the finest ramifications of the vessels star out distinctly, although the connection between them and the main vessels is in places concealed b the haze. The mid point of the fovea centralis is occupied by a spot which is dark red with a ligl centre, and which represents the chorioid showing through the haze.

vanishes; the retina regains its transparency, but becomes perfectly atrophic. The opti
disc is now white and sharply outlined, the blood-vessels both upon the disc and in th
retina are scanty, thin, filamentous, and often bordered by white lines; many of the fine
vascular twigs become completely invisible. The blindness persists and is permanent
Very rarely indeed the condition is found in both eyes.

Instead of affecting the central artery, the embolism may affect only a *branch* of it
In that case the visible alterations are limited to that portion of the retina which draw
its blood supply from the obliterated vessel. The blindness, too, corresponds then simpl
to the part of the retina that is diseased—that is, appears under the form of a defect in th
visual field, one half of the latter or a sector of it being destroyed. Moreover, even i
embolism of the central artery itself a small portion of the retina may retain its functiona
power. This is the case when cilio-retinal vessels are distributed to the retina (see § 38)
In ophthalmoscopic examination such vessels can be recognized from the fact that the
arise by a hook-shaped extremity from the border of the disc (Fig. 35). These vessels
which receive their blood from the short posterior ciliary arteries, are of course unaffecte
by embolism of the central artery, and hence the region of the retina which is supplied b

them, and lies between the papilla and macula, retains its functional power. (See Fig. 349 B). In rare cases a cilio-retinal vessel itself may be blocked by an embolus (Meller).

Embolism occurs in all disorders which give rise to the entrance of clots into the circulation, and above all in affections of the heart. A cure can be effected only in very recent cases, before the death of the retina has taken place. The retina might then regain its function if we could succeed in restoring the circulation in it. The only way in which this can be accomplished is for us to drive the plug lodged in the central artery on into the smaller branches, where it will do less harm. With this object in view, we draw off the aqueous by paracentesis of the cornea. In consequence of the sudden diminution of intra-ocular tension thus produced, the blood tends to flow into the eye in increased quantity, and may thus push the embolus forward if it is not too tightly fixed in place. To effect the same object, we massage the eye (see § 123), and dilate the retinal arteries by giving inhalations of nitrite of amyl. In these ways it has been possible to restore the circulation in the retina, and with it the sight in some few cases, in which the lesion was still very recent. Spontaneous recovery took place in one case in which embolism was produced by an air bubble (Hirsch).

What is known clinically under the name of embolism of the retinal arteries corresponds to those cases in which there are no infected emboli in question; hence, no inflammation occurs, but only the results of the mechanical cutting off of the blood supply. The retina being no longer nourished simply dies. It does not indeed become necrotic, because it still obtains a supply of nourishment from the adjacent choriocapillaris of the chorioid, although this does not suffice to maintain the function of the retina. But *infectious emboli* also may get into the retinal arteries, as sometimes happens in pyemia. Then a suppurative retinitis develops, the suppuration from which soon extends to the other structures of the eyeball, so that the clinical picture of panophthalmitis is produced (metastatic ophthalmia; see §§ 755, 756, 783).

Spasm and other Conditions Simulating Embolism.—The ophthalmoscopic picture of embolism is the expression of changes which set in when the supply of arterial blood is cut off from the retina. It is hence not confined to embolism of the central artery, but is found in sudden occlusion of it due to any cause. Such occlusion may be produced by endarteritis, by thrombosis of the artery, and also by spasm of it, in case it lasts long enough. Evidence of extreme spasm with occlusion can sometimes be furnished by the ophthalmoscope. We see then a thread-like bloodless vessel which as we look at it dilates (perhaps under the influence of amyl nitrite) to full size once more. Some would ascribe to spasm many of the cases regarded as embolism; particularly the cases in which the complete occlusion has been preceded by repeated attacks of transient obscuration. Spasm is also produced by acute quinine poisoning (see § 819). The ophthalmoscopic picture of embolism may also be caused by compression of the artery within the trunk of the optic nerve consequent upon hæmorrhage or inflammatory infiltration in the latter; and by a solution of the continuity of the artery occurring when the optic nerve is cut or torn through in front of the point where the central vessels enter it (§ 822).

774. Thrombosis of Central Vein.—Thrombosis, which was first anatomically demonstrated by Michel, is characterized by an enormous distention of all the retinal veins with blood, while the arteries are so attenuated as to be scarcely discoverable (Fig. 348, also Fig. 349 C and D). The blood escapes from the turgid veins at many spots so that the entire fundus is covered with hæmorrhages. These keep recurring again and again, and with their recurrence the sight which from the start was greatly reduced is at length utterly destroyed.

Thrombosis may be confined to a *branch* of the central vein, in which case these changes in the fundus are present in that portion only of the retina which represented the area of distribution of the occluded vein. The prognosis then is more favorable than in occlusion of the main trunk. The disturbance of the circulation may be com-

pensated for by the capillary vessels of collateral channels—capillary vessels in this ca dilating to form larger vessels which can be seen with the ophthalmoscope.

In some cases of thrombosis a white star figure like that present in albuminur retinitis is found in the macula (cf. Fig. 348).

The marked venous stasis, which must occur as a result of a venous thrombosi is without doubt the cause of the *increase of tension* frequently found in these cases. Th so-called hæmorrhagic glaucoma gives a bad prognosis, since it is generally incurable, eve by iridectomy.

Thrombosis of the central vein occurs for the most part in elderly persons sufferin from *arteriosclerosis*. *Inflammation in the orbit* may also lead to thrombosis of the centr

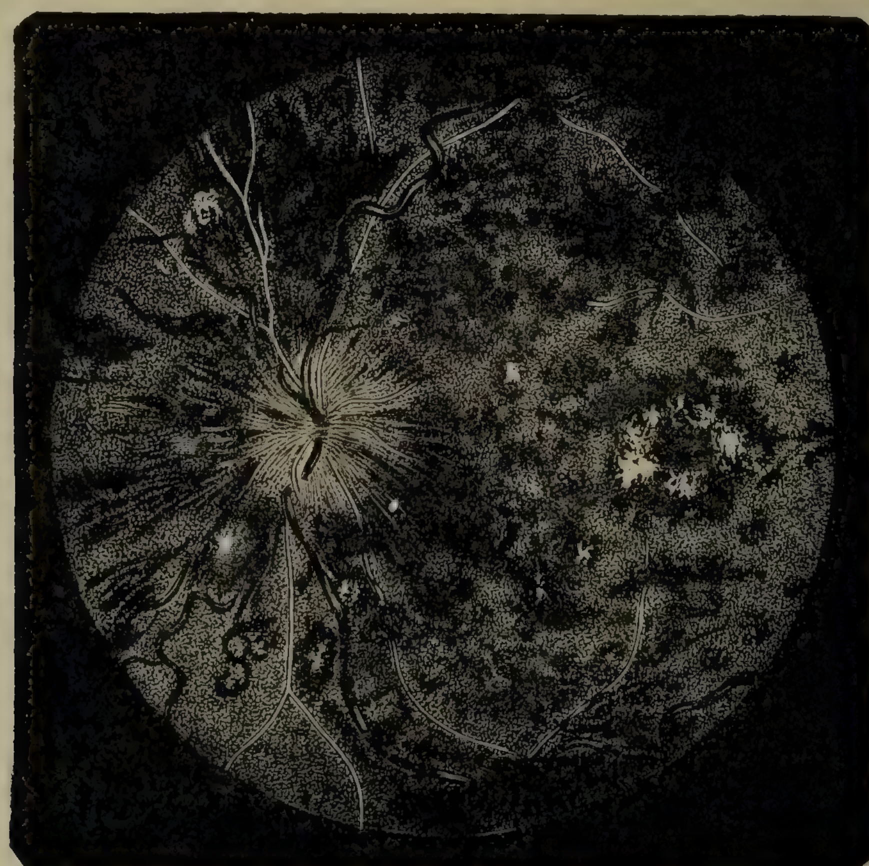


Fig. 348.—Thrombosis of the Central Vein. Present for Fourteen Days in a Man Fift two Years of Age.

The outlines of the papilla are concealed partly by a gray striate opacity, partly by radially dis posed hæmorrhages. The retinal arteries are attenuated; the retinal veins are unusually broad anc tortuous, and are filled with blackish blood. In many places the vessels are covered by extravasation of blood, and hence appear interrupted. The hæmorrhages are extremely numerous, have a dark-red almost black, color, and are either radiately striate or irregularly rounded in shape. A few of th hæmorrhages have taken on a brilliant-white hue in their centre. This is particularly visible in th large patches of blood lying outside of the macula lutea. The retina, wherever it is not occupied b hæmorrhages, is clouded and grayish.

vein, probably through the development in the orbital veins of thromboses, which subsequently extend into the central vein. In this way cases of blindness are sometimes produced in facial *erysipelas*. The latter by extension may set up an orbital cellulitis which in turn may produce a thrombosis of the central vein (Knapp). In such cases we find, after the erysipelas has run its course and the swelling of the lids has abated, that the eye is blind. The ophthalmoscope shows atrophy of the optic nerve with extreme attenuation of the blood-vessels. This blindness due to erysipelas may affect both eyes. Thrombosis running a comparatively favorable course occurs after *influenza* (Coats, Jackson). The field of vision is commonly preserved.

775. Retinal Hæmorrhage.—Extravasations of blood into the retina are frequent and occur in all sorts of sizes and shapes. They form dark-red patches contrasting with the brighter red of the fundus.

If in the fibre layer of the retina they have striate or flame-like shapes, because the exuded blood spreads along the fibres (Figs. 351 and 353). Hæmorrhages in the

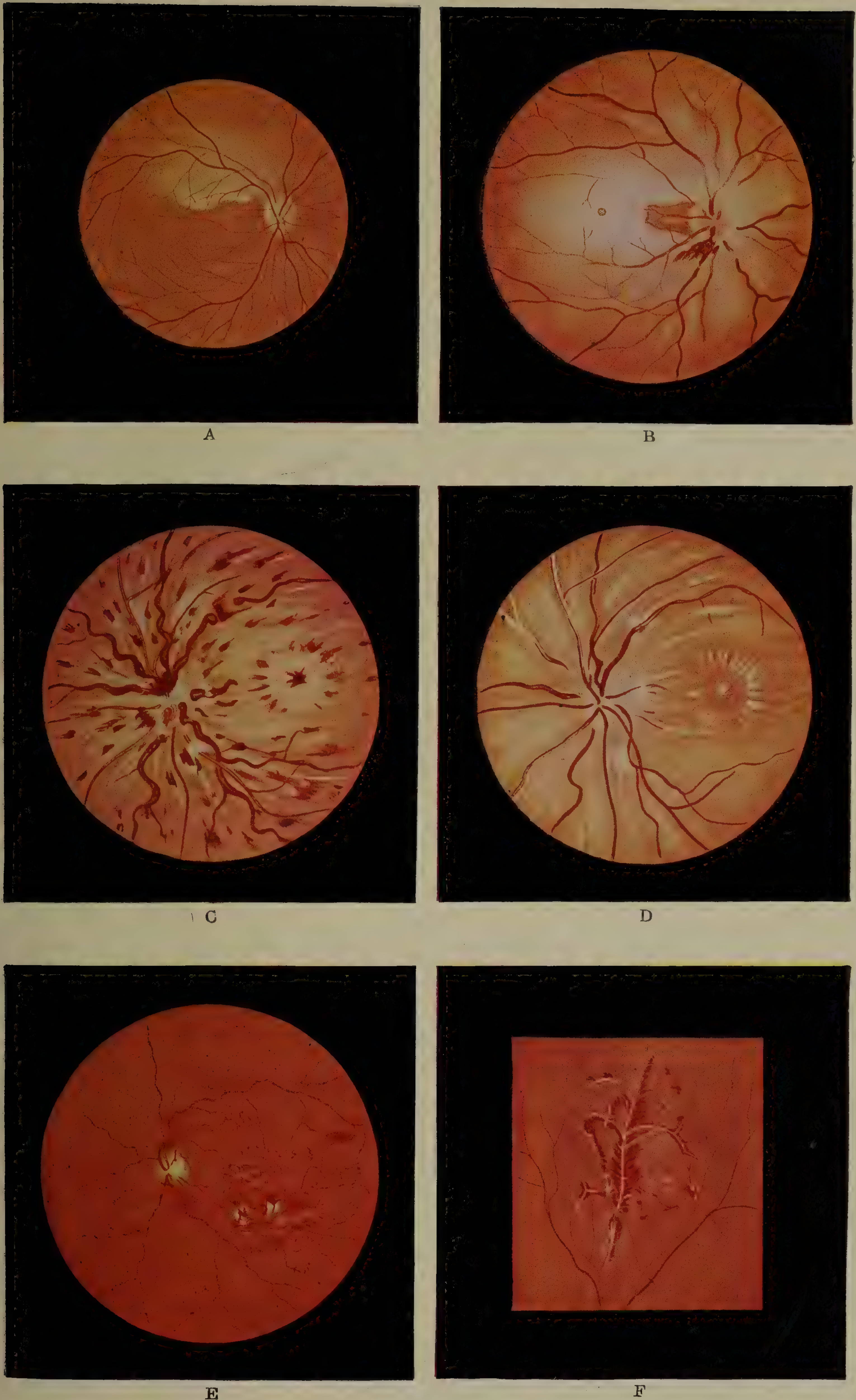


Fig. 349.—Changes in Retinal Vessels. (After Würdemann in Posey and Spiller.)

A, Embolism central artery; partial, affecting only inferior branch (Haab). B, Embolism central artery; total within nerve; a cilio-retinal vessel supplies a small area of retina in which function is preserved (Würdemann). C, Thrombosis of central vessels from mumps (Würdemann). D, same case six months later, showing sclerosis and atrophy (Würdemann). E, Hæmorrhages from retinal vessels Magnus). F, Perivasculitis luetica (Magnus).

deeper layers of the retina or between it and the chorioid are of rounded or irregular shape (Fig. 352). In the region of the macula large disc-shaped extravasations sometimes occur situated not in the retina, but between it and the vitreous (preretinal or subhyaloid hæmorrhage, Fig. 350). The retinal tissue then is not injured by the hæmorrhage, so that, after resorption of the blood, vision becomes normal again. Sometimes, however, preretinal hæmorrhages are not absorbed but form large membranes (retinitis proliferans, see § 784).

The extravasations are most frequently located in the neighborhood of the larger vascular trunks. The causes of retinal hæmorrhages are—

1. Brittleness of the retinal or chorioidal vessels due either to general disease (arteriosclerosis, syphilis, nephritis etc.) or to local changes. Under the latter head

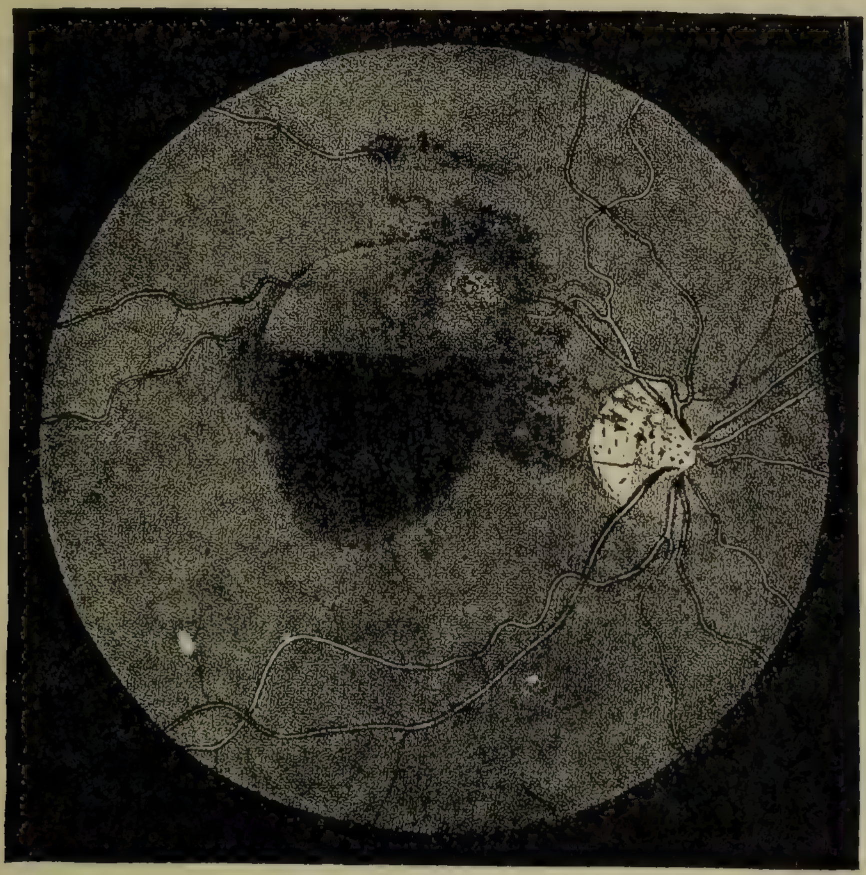


Fig. 350.—Preretinal Haemorrhage.

This represents the right eye of an elderly woman. The eye, in consonance with its myopia, shows a broad white crescent at the temporal border of its disc; and the latter also has a pretty large physiological excavation. The retinal arteries are very tortuous, the veins normal. The middle of the fundus is occupied by a large hæmorrhage, covering the region of the macula lutea, and extending upward as far as the superior temporal vessels, which are partially concealed by it. The lower part of the hæmorrhage is dark red, and is separated by a sharp horizontal line from the upper, pale-red portion. This division into two parts is caused by the settling of the blood corpuscles to the bottom of the still fluid blood. In the vicinity of the large hæmorrhage, especially at its upper and inner borders, lie numerous small spots of blood. These extend up to and upon the white crescent adjoining the optic nerve and up to the superior temporal vessels. From these vessels is derived the extravasated blood, which, after breaking through the membrana limitans interna of the retina, gets between the latter and the vitreous, and sinks down to the region of the macula lutea, where even in the normal eye the connection between the retina and the vitreous is the least.

must be reckoned those hæmorrhages which occur so frequently in excessively myopic eyes in the region of the macula.

2. High blood pressure especially when combined with the vascular changes above mentioned.

3. Over-distention of the blood-vessels by circulatory disturbances, such as active and passive hyperæmia of the retina and occlusion of the central artery and vein or their branches. In new-born infants retinal hæmorrhages are often found as a result of the disturbances of circulation occurring within a child's skull during birth. They are present in from 21 to 32 per cent of the new-born, occurring especially in protracted labors, and in cases of instrumental delivery and constriction of the umbilical cord. They absorb very rapidly (Leber). Many cases of so-called congenital amblyopia (§ 240) may possibly be explained by them; i.e., the extravasated blood is absorbed and leaves

no trace, but the lacerated retinal tissue never again becomes entirely normal. T retinal hæmorrhages which very often occur in glaucomatous eyes as a result of iride tomy are the result of sudden reduction of the intra-ocular tension, so that an unwont amount of blood pours into the retinal vessels and distends them unduly. Moreov the retinal hæmorrhages adduced under the next head may in part be caused by blocki of the smaller vessels—e.g., the hæmorrhages in sepsis by emboli composed of mass of fungi.

4. Altered composition of the blood affecting the vessel walls. Here belo retinal hæmorrhages in extreme anæmia, particularly pernicious anæmia, in leucæm scurvy, purpura, sepsis, albuminuria, diabetes, oxaluria, intermittent fever, relapsi fever, influenza, jaundice, phosphorus poisoning, extensive burns of the skin, e Tuberculosis is often the cause of the recurrent retinal hæmorrhages which, togeth with recurrent hæmorrhages into the vitreous, sometimes occur in young persons (§ 66 Often, however, these happen without assignable cause.

5. Rupture of the blood-vessels due to trauma.

Retinal hæmorrhages absorb very slowly, requiring for this weeks and month during which they are often seen to take on a white color (Fig. 348). They ultimate disappear, leaving either no trace of their presence or decolorized whitish, rarely pi mented spots in the fundus. When large they may become organized into plaques masses of connective tissue. They cause a scotoma which, when relative, may appe greenish because the patient is seeing through a thin layer of blood. Whether t scotoma remains permanently or not, depends on the degree to which the hæmorrhage h lacerated the retinal tissue.

For the treatment of retinal hæmorrhages see remarks under treatment of vitreo hæmorrhages (§ 668).

II. Inflammation of the Retina

776. Symptoms.—Inflammation of the retina (retinitis) is characte ized first of all by a diffused cloudiness of the organ. The cloudiness vari very greatly in intensity, although in general it is greatest in the vicinity the papilla, because here the retina is thickest. Consequently, the outline of the papilla become indistinct and the vessels in the retina hazy. In addi tion, circumscribed exudates occur in the retina, usually under the form brilliant-white, sharply defined patches. Retinitis is always associated wit hyperæmia of the retina, evidenced by distention and tortuosity of th vessels, and often also by extravasations of blood. Owing to the passage the exudate from the retina into the vitreous, opacities of the vitreous ar produced.

The function of the retina is impaired in proportion to the intensit and extent of the inflammation. In the lightest cases vision may be normal so that the patients complain simply of the presence of a light-colored clou before their eyes. But for the most part vision is very considerably reduced both because of the changes in the retina itself and because of the accom panying opacities in the vitreous. Circumscribed exudates cause fixed sco tomata in the field of vision. The light sense is often much reduced Retinitis affecting the region of the macula lutea is marked by a centra scotoma and by retinal metamorphopsia (micropsia, macropsia—see § 23 and cf. § 748).

The course of the retinitis is always pretty sluggish. Only in the light est cases does the inflammation abate completely in a few weeks, and the the sight may again become perfectly normal. But for the most part i

takes several months for all inflammatory symptoms to disappear from the retina, and the sight remains permanently impaired. Severe and, more particularly, recurrent inflammations of the retina lead to atrophy of it (§ 791), pigmentation frequently occurring at the same time (through migration of pigment from the pigment epithelium). When atrophy of the retina has once set in the sight is always abolished, either completely or all but a remnant, and cannot be restored.

A *new formation of blood-vessels* may occur in retinitis proliferans and also whenever a retinitis, particularly a syphilitic retinitis, has lasted a long time. The vessels in this case project from the retina into the vitreous under the form of slender and, often also, of repeatedly convoluted coils.

For the *pathological changes* occurring in retinitis, see § 798.

777. Etiology.—Retinitis is rarely a local lesion; in most cases it is due to some internal or general disease, to the discovery of which we are often led just by finding the retinitis. Among such general diseases are, above all, albuminuria, diabetes, leucæmia, syphilis, gout, and diseases of the vascular system. In these cases, in which a general disease underlies the retinitis, the latter is usually bilateral.

It is very rare indeed for retinitis to remain confined strictly to the retina, the fact being that it usually implicates the head of the optic nerve as well. If this implication is present to a marked degree we call the case one of *neuro-retinitis*. We use the same term when an inflammation that starts from the head of the optic nerve extends out into the retina. With regard to its etiology, therefore, neuro-retinitis is at one with retinitis on the one hand and with neuritis on the other. A similar relation exists between retinitis and chorioiditis. A chorioiditis cannot exist without the portions of the retina immediately adjacent participating in the process. From an anatomical standpoint every chorioiditis is a *retino-chorioiditis* (or *chorio-retinitis*), although we generally do not use this term unless ophthalmoscopically visible signs of inflammation can be demonstrated to exist both in the retina and in the chorioid. This occurs when the retinitis predominantly affects the outer layers of the retina or the neuro-epithelium (*retinitis externa*). This form, which is usually described under chorioiditis, is marked by atrophy or proliferation of the pigment epithelium, but not usually by changes in the retinal vessels or (at least in the beginning) in the optic nerve but by superficial white spots. The subsequent atrophy of the disc has the appearance of a retinitic atrophy. *Retinitis interna*, which affects the inner (cerebral) layers of the retina, is marked by changes in the retinal vessels, hæmorrhages, diffuse cloudiness due to retinal œdema, white patches which may overlie the retinal vessels, and often by haziness or actual inflammation of the optic nerve. The subsequent atrophy of the disc has the appearance of a neuritic atrophy (Dimmer). Retinitis affecting all the layers also occurs.

It must be observed that in some of the conditions noted below as retinitis the changes are more those of œdema and degeneration than of true inflammation.

778. Varieties.—The main varieties of retinitis are as follows:

I. Retinitis Interna.

Retinitis Albuminurica.—This of all inflammations of the retina is the most characteristic. In addition to the general signs of retinitis, such as haziness of the retina and of the outline of the papilla, distention of the retinal arteries, and striate hæmorrhages, it is particularly distinguished by the white patches in the fundus (Fig. 351, Fig. 354, C and D). The white spots are found chiefly in two places—in a certain area

surrounding the papilla and in the macula lutea. In the former situation we find rathe large white patches, which may be so numerous as to form a speckled zone, which usually interrupted at a point corresponding to the macula lutea. The latter, on th other hand, is occupied by small white stipplings radially disposed, so that a prett radiating crown is formed with the fovea centralis at its centre. Very often the crow is incomplete, the rays being fully formed only on certain sides of it. Later the spot and hæmorrhages may be absorbed or they may be followed by atrophy of the retin and optic nerve. In some cases detachment of the retina, usually bilateral, occurs.

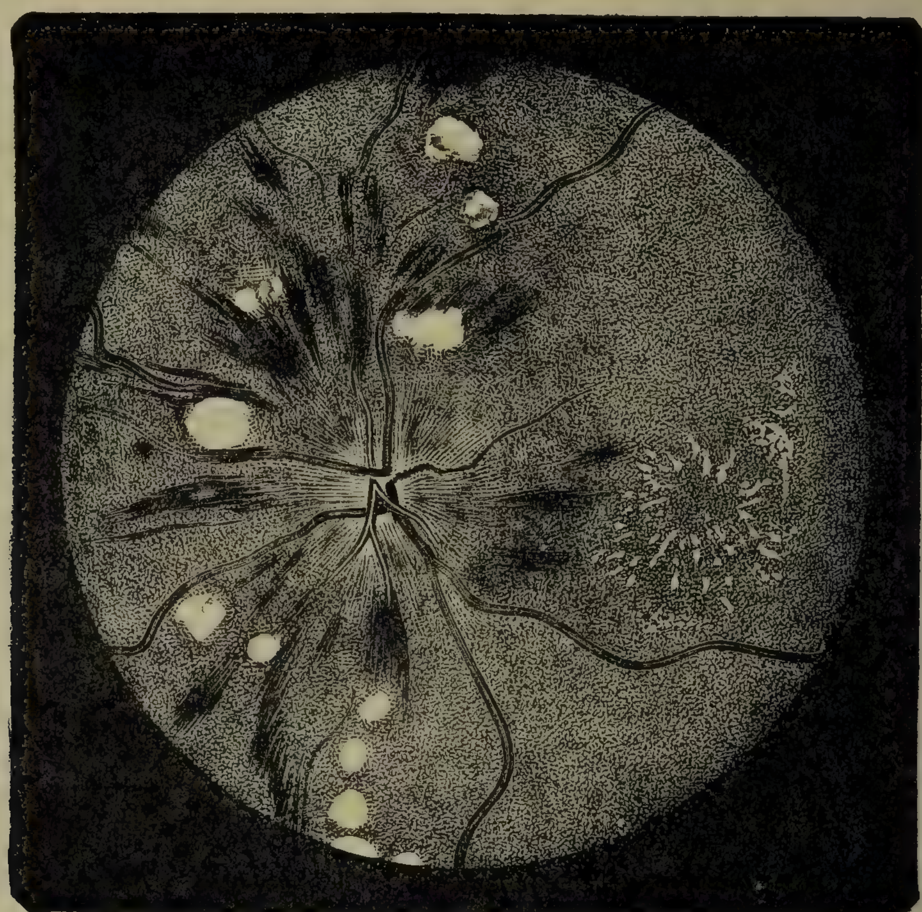


Fig. 351.—Retinitis Albuminurica.

The clouding of the retina is most pronounced in the region of the disc, where it exhibits a fine radial striation, and completely veils the margin of the nerve. Furthermore, even at a considerable distance from the disc, the retinal clouding covers isolated portions of the vessels and especially the distended veins, with a delicate haze, so that the vessels in these places look lighter. Surrounding the papilla are found rounded, brilliantly white spots of exudation and numerous dark-red, radially striate hæmorrhages. The latter lie mainly in the neighborhood of the larger retinal vessels, and in part cover them. From this fact and from their striate appearance, it can be inferred that they belong to the most anterior layer of the retina—the nerve-fibre layer. In the region of the macula lutea is seen a group of small white specks, which combine to form the stellate figure characteristic of retinitis albuminurica. In the present case this is not very regularly formed, and above it is a somewhat larger spot, produced by the coalescence of several small dots.

The affections of the fundus occurring in albuminuria do not always present this characteristic appearance. Thus we find simple retinal hæmorrhages, retinitis hæmorrhagica (Fig. 353), retinitis cachecticorum (§ 783), and neuritis (including even choked disc). On the other hand a picture very similar to that of typical retinitis albuminurica may be produced by other causes, (see Retinitis Stellata, § 779). Hence an examination of the patient's urine should be made in every case of retinitis.

All forms of kidney disease may be complicated with retinitis, but the one most frequently thus complicated is the atrophic kidney. The severity of the retinitis bears no fixed proportion to the intensity of the kidney disease nor to the amount of albumin in the urine. The like is true of the subsequent course; the retinitis may improve while the kidney lesion grows worse, or vice versa. Nevertheless, retinitis albuminurica has, on the whole, an evil prognosis. Even if it does occur sometimes with benign kidney lesions (e.g., with scarlatinal nephritis and the renal affection of pregnancy), yet it far more frequently is associated with the serious chronic cases, and it is a matter of experience that most patients suffering with typical retinitis albuminurica succumb from their renal disorders within a year or two. This bad prognosis does not apply so fully to

some of the atypical forms especially retinitis cachecticorum. (For the connection between the retinitis and the renal disorder, see § 91).

Albuminuric retinitis is to be distinguished from *uræmic amaurosis* (§ 242), which also occurs in renal disease but in which instead of a gradual and incomplete loss of vision such as occurs in retinitis, there is suddenly developing and complete but transient blindness with retention of the pupillary light reaction and without lesion discoverable by the ophthalmoscope. In making this distinction we must remember that uræmic amaurosis may occur in a patient already suffering from albuminuric retinitis.

779. Retinitis Stellata.—This is also called *pseudo-nephritic retinitis,* because it resembles albuminuric retinitis in having the characteristic star figure at the macula, but

FIG. 352.—DIABETIC RETINITIS IN A MAN SIXTY-NINE YEARS OF AGE.

When the diabetes was discovered eleven years previous 6 per cent of sugar was present, while for some years past, under the influence of suitable treatment, the sugar has almost disappeared. The disturbance of sight has lasted for nine months, and is caused mainly by a central scotoma for blue—a scotoma which corresponds to the exudate in the macula. In keeping with the myopia of the eye there is an atrophic crescent, about half the width of the disc adjoining the optic nerve. In its posterior section the retina shows numerous punctate hæmorrhages, which are generally disposed in groups, and frequently coalesce to form rather large patches. In addition, small, brilliantly white dots are present of irregular shape and sharp outline. These are disposed quite irregularly in a large circle surrounding the macula. In the macula itself is found quite a large exudate having a structure like that of the small dots.

is not due to nephritis. One form occurs as a unilateral or bilateral affection, especially in young people and without discoverable cause. It may or may not be associated with a slight papillitis. The visual disturbance is often moderate, and after a long time complete recovery may take place. Other cases are more acute in onset, being preceded by violent pain in the eyes and head and accompanied by marked neuro-retinitis with great narrowing of the arteries. These cases are helped by salicylic acid. Other cases occur with optic neuritis due to influenza, syphilis, or chlorosis; and a star figure in the macula also accompanies some cases of choked disc due to brain tumor, meningitis, or hydrocephalus. Other cases, marked by sudden blindness or sector-like limitations of the field are caused by circulatory disturbances (probably embolism) in the retina. Finally somewhat similar changes occur in severe injuries of the skull, produced by blunt force (Purtscher's *angiopathia retinæ traumatica*) and in ruptures of the liver (Tietze).

780. Retinitis Diabetica.—This is characterized in many cases by the presence of small, brilliantly white spots in the retina, which chiefly occupy the region of the macula lutea and its vicinity, without, however, presenting a stellate arrangement, as in album-

inuric retinitis (Fig. 352). Sometimes by the confluence of small dots one or two large patches are formed, which show by their crenated border that they are composed of smaller spots. Between the white patches lie punctate extravasations of blood. The rest of the retina is transparent, and the papilla, too, is unaltered.

In other cases this characteristic picture is not present; in fact, diabetic retinitis may actually appear under the guise typical of albuiminuric retinitis or closely resembling a retinitis circinata (see § 787). Very rarely in young diabetics, especially when nearing death, *retinal lipœmia* is found, i.e., a condition in which on account of the large amount of fat in the blood, the retinal vessels appear reddish-white or pure white.

781. Retinitis Leucæmica.—In this, superadded to the general symptoms of inflammation of the retina (namely, retinal cloudiness and hæmorrhages), there is found, as a

Fig. 353.—Hæmorrhagic Retinitis in a Woman of Forty-eight, who Suffered from Chronic Nephritis and Cardiac Hypertrophy.

The outlines of the grayish-red papilla are obscured, the arteries of the retina are somewhat contracted, and in places are concealed by the cloudiness of the retina. The veins are very tortuous, and from the character of their reflex streaks it can be seen that not all their convolutions lie in the same plane. The retina, as a whole, shows a faint, striate opacity, and is filled with very numerous, partly striate and partly rounded hæmorrhages. To the left and below, between the two branches of a vein, is a white spot which has originated from a hæmorrhage.

characteristic feature, a light hue of the blood in the retinal vessels, which ordinarily are greatly dilated. Furthermore, since in leucæmia the blood that flows in the chorioidal vessels is of lighter color than normal, the entire fundus, even where there is no retinitis present, is of a much lighter red than usual, and shows a yellowish tinge. Likewise characteristic of leucæmic retinitis are white patches with a red rim (consisting of white corpuscles surrounded by red ones); but such patches are present in only a few cases of retinitis leucæmica. (See Fig. 354, B).

782. Retinitis Hæmorrhagica.—This is diagnosticated when, along with the presence of numerous hæmorrhages in the retina, the latter itself is hazy and the papilla is obscured (Fig. 353). Hæmorrhagic retinitis is due for the most part to diseases of the retinal vessels. Many of these cases are probably identical with those of thrombosis of the central vein, others occur in nephritis, diabetes, leucæmia, arteriosclerosis, gout,

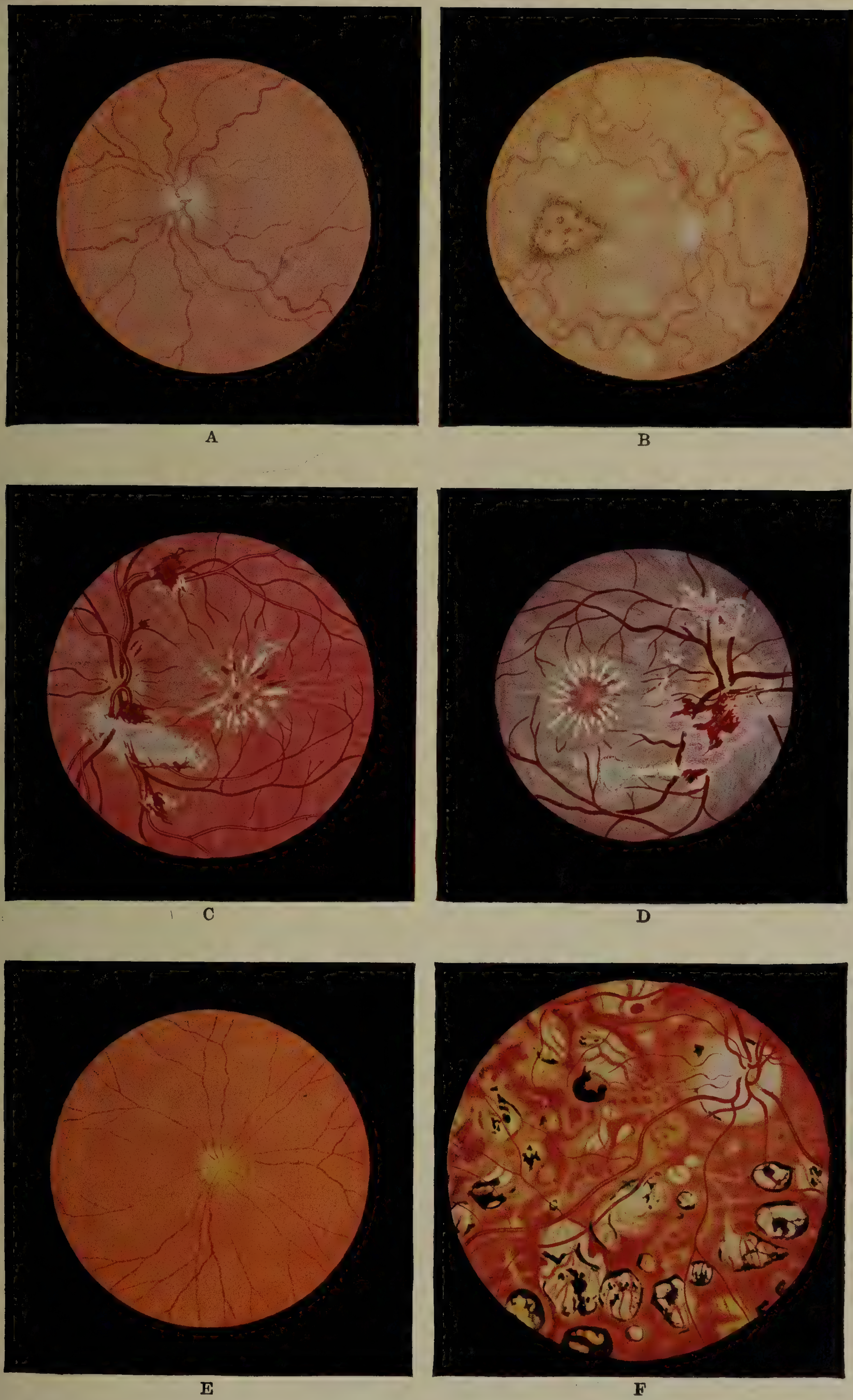


Fig. 354.—Inflammations of the Retina. (After Würdemann in Posey and Spiller.)

A, Œdema in pernicious anæmia (Oliver). B, Leucæmic retinitis (Oliver.) C, Albuminuric retinitis and neuritis of pregnancy (Würdemann). D, Albuminuric retinitis in the negro (Würdemann). E, Syphilitic retinitis (Haab). F, Atrophy of retina, chorioid, and nerve following chorio-retinitis luetica (Oeller).

oxaluria, and syphilis. The syphilitic (and also the non-syphilitic) varieties may be associated with disseminated chorioiditis (Dimmer).

783. **Retinitis Septica.**—This is a form of retinitis found in septic conditions, both grave and mild, in pernicious anæmia, and in anæmia due to hæmorrhages caused by intestinal parasites. It is marked by the presence of striate hæmorrhages and small white patches often surrounded by a rim of hæmorrhage. Very similar appearances occur in the retinitis produced by metastasis (*retinitis metastatica*), when as sometimes happens this is confined to the retina. If on the contrary the metastasis goes on to produce metastatic ophthalmia (§ 756) we see, if the retina is visible at all, large often very prominent white masses, retinal hæmorrhages, optic neuritis, and vitreous opacities. *Retinitis cachecticorum*, occurring in the cachexia of cancer, nephritis, tuberculosis, liver disease, gangrene of the lungs, etc., shows changes like those of retinitis septica but the white spots are more numerous (Leber, Dimmer).

784. **Retinitis Proliferans.**—Manz described under this name an affection in which dense masses of connective tissue extend out from the retina into the vitreous and cover

Fig. 355.—Retinitis Proliferans.

In a man thirty-five years of age, who suffered frequently from palpitation of the heart and nose bleed, sudden obscuration of the sight occurred in both eyes five years ago. This obscuration has recurred repeatedly since, being due to recurrent hæmorrhages into the vitreous. In the vitreous of the left eye close to the papilla lies a shining white mass having a structure partly fibrous, partly granular. From this mass pass strands in different directions, some extending as far as the periphery of the fundus. No vessels are recognizable in the white mass, which covers the upper half of the papilla. The other half of the papilla and the rest of the fundus are somewhat hazy (owing to the presence of a faint opacity of the vitreous), and besides appear indistinct, because the ophthalmoscope is adjusted not for them, but for the connective-tissue mass, which is situated farther forward. Indistinctly visible are indications of the retinal vessels (above and to the outer side) and indications of a tessellation of the fundus (below and to the inner side).

a portion of the fundus—in fact, even the disc itself (Fig. 355). Into these masses run new-formed vessels from the retina. For a number of these cases it is probable that these masses of connective tissue have been preceded by hæmorrhages, which were poured out from the retina into the vitreous and afterward became organized (see § 775). The most common cause of these are the spontaneous hæmorrhages occurring in the retina and vitreous in young persons particularly as a result of tuberculosis (see §§ 667, 775). Other causes alleged are anæmia, chlorosis, menstrual congestion, syphilis, nephritis, diabetes, arteriosclerosis, oxaluria, etc. Other cases are traumatic. Such

are those in which we find dense white masses encapsulating a foreign body or an entozoön or dense cicatricial masses traversing the vitreous as the result of a penetrating or non-penetrating injury (especially that inflicted by some projectile) (Leber). Some of these cases retain surprisingly good vision.

II. Inflammation Affecting the Middle and External Layers

785. Retinitis Syphilitica.—Acquired syphilis is one of the most frequent causes of retinitis. Syphilitic retinitis is usually associated with disease of the uvea, and above all of the chorioid; often also of the iris, which then exhibits the picture of syphilitic iritis. It generally affects the middle and external layers of the retina and is commonly seen in the *diffuse* form. In this the entire retina is clouded and faintly gray (see Fig. 354 E); here and there, especially in the macular region, more densely gray spots may be found. Later on in the disease, in proportion as the cloudiness of the retina abates, changes in the pigment epithelium set in, and ultimately migration of pigment from the latter may take place into the retina, so that a picture is formed resembling that of

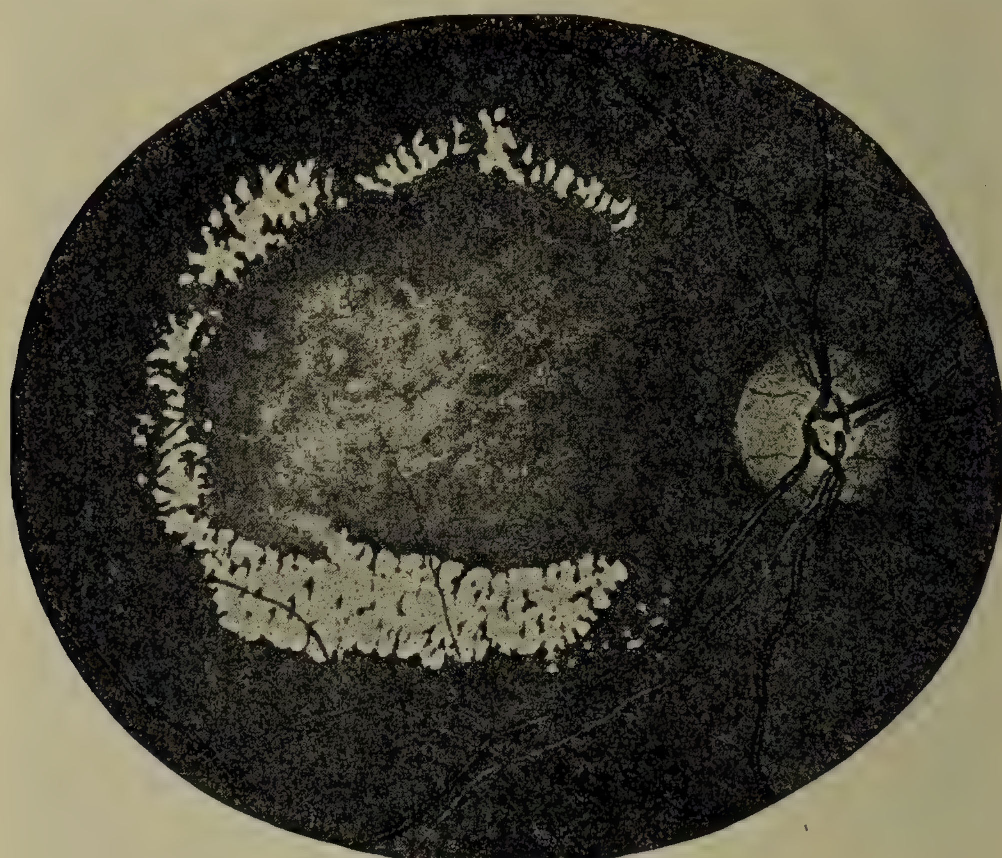


Fig. 356.—Retinitis Circinata.

Right eye of a woman seventy years of age. Erect image. The papilla and the retinal vessels are normal. The macula lutea and its vicinity are transformed into a yellowish-gray, not sharply limited area which is enclosed by a zone composed of white spots. The spots run together, to form map-like figures. On the side toward the papilla the spots are absent, so that the ring here is open.

retinitis pigmentosa. This form of syphilitic retinitis consequently coincides in part with the syphilitic chorioiditis described by Förster (§ 744).

In the rarer *circumscribed* form a bulky white exudate is found either in the region of the macula lutea or more frequently close to one of the larger retinal vessels. In the latter case it is often possible with the ophthalmoscope to recognize that an affection of the wall of the vessel is the cause of the circumscribed exudation. Later on, the exudate is transformed into bluish-white scar tissue, which by shrinking may give rise to detachment of the retina.

In syphilis we may also find multiple white spots lying along the retinal vessels, hæmorrhagic retinitis (§ 782), and *recurrent central retinitis*. In the latter, except for faint cloudiness and a few whitish spots in the macular region there are hardly any ophthalmoscopic changes, but there are recurrent attacks of suddenly developing obscuration of sight with central or annular scotomata (Von Graefe).

Hereditary syphilis, too, leads to retinitis, which may be observed in children or may even be congenital. Ordinarily we see only the evidences of inflammation after it has

run its course; such evidences occurring either under the form of numerous small, light or black spots, or under the form of an old, bulky exudate, that has been transformed into connective tissue.

786. Retinitis in Irido-Cyclitis.—A diffuse retinitis occurs in cases of plastic irido-cyclitis. The form occurring in sympathetic irido-cyclitis resembles diffuse syphilitic retinitis.

In *leprosy* retinitis of varying form occurs; particularly characteristic being white nodules found in the region of the ora serrata (Trantas and Valetta), which are always accompanied by leprous changes in the cornea or iris.

787. Retinitis Circinata (Fuchs).—This rather rare, very chronic condition is characterized by an elliptical ring of white patches, often agglomerated into lobulated masses, surrounding the macula, which itself is generally occupied by an area of gray or yellowish opacity. The disease occurs particularly in old women. Its cause is unknown.

788 Retinis Exudativa.—This is so called because it is characterized by particularly massive exudation into the retina (Coats). In the latter there are numerous large and small white spots, which are often arranged in groups, and later may become confluent, or there are extensive white areas, usually somewhat elevated above the level of the retina. They may, indeed, project like a veritable tumor. Frequently the blood-vessels take a prominent part in the process either under the form of a general dilatation or a localized aneurysmal widening or the formation of numerous new vessels. These cases are described under the name of *Hippel's retinitis* or *angiomatosis retinæ*. This is often classed as a distinct disease—angiomatous degeneration or primary angioma. The arteries and veins are greatly distended, and there is a rounded prominence into which one of the distended vessels empties (Leber). Both conditions are very chronic, and in both detachment of the retina usually sets in later, followed by increase of tension or irido-cyclitis and complete blindness. Exudative retinitis is rare and its cause unknown. It usually attacks young persons, and more frequently males, and commonly affects but one eye. Coats, who ascribed it to hæmorrhages in the outer layers of the retina, called it *retinitis hæmorrhagica externa.*

789. Photogenous and Actinic Retinitis.—Inflammatory or degenerative changes may be produced in the retina by exposure to intense light or other forms of radiant energy. Here belong the slight pigment changes in the macula produced by looking at the sun or a powerful artificial light (*solar* and *electric retinitis*). These changes are associated with a permanent central scotoma, which while not in many cases altering greatly the visual power for distance, impairs the ability to read. This is often combined with metamorphopsia (cf. § 86). The seat of the trouble is evidently in the outer layers of the retina.

Exposure to *X-ray* may produce a retinitis marked by yellowish-white plaques near the disc and diffuse pigmentation of the fundus (Ehnhuus). The translator has seen a case in which a large pre-retinal hæmorrhage was produced by two applications of *radium.*

790. Treatment.—The treatment must be directed both against the casual lesion and against the local affection of the retina. The first indication is most readily fulfilled in cases of syphilitic retinitis, where energetic mercurial treatment in most cases results in rapid improvement. In albuminuric retinitis due to pregnancy artificial interruption of pregnancy may have to be considered. The symptomatic treatment consists in fully safeguarding the eye by forbidding all work and by protecting the eye from glaring light, either by means of dark glasses or in severe cases by confinement in a darkened room. To combat the inflammation, and also to cause

resorption of the exudate and restoration of the transparency of the vitreous, mercury, potassium iodide (both remedies being used in non-syphilitic as well as in syphilitic cases), saline purgatives, diaphoretic treatment, and subconjunctival injections of salt or mercury oxycyanide are employed.

III. Atrophy of the Retina

791. Atrophy of the retina is the result of its protracted inflammation, or is the final outcome of an embolism or a thrombosis of the retinal vessels.

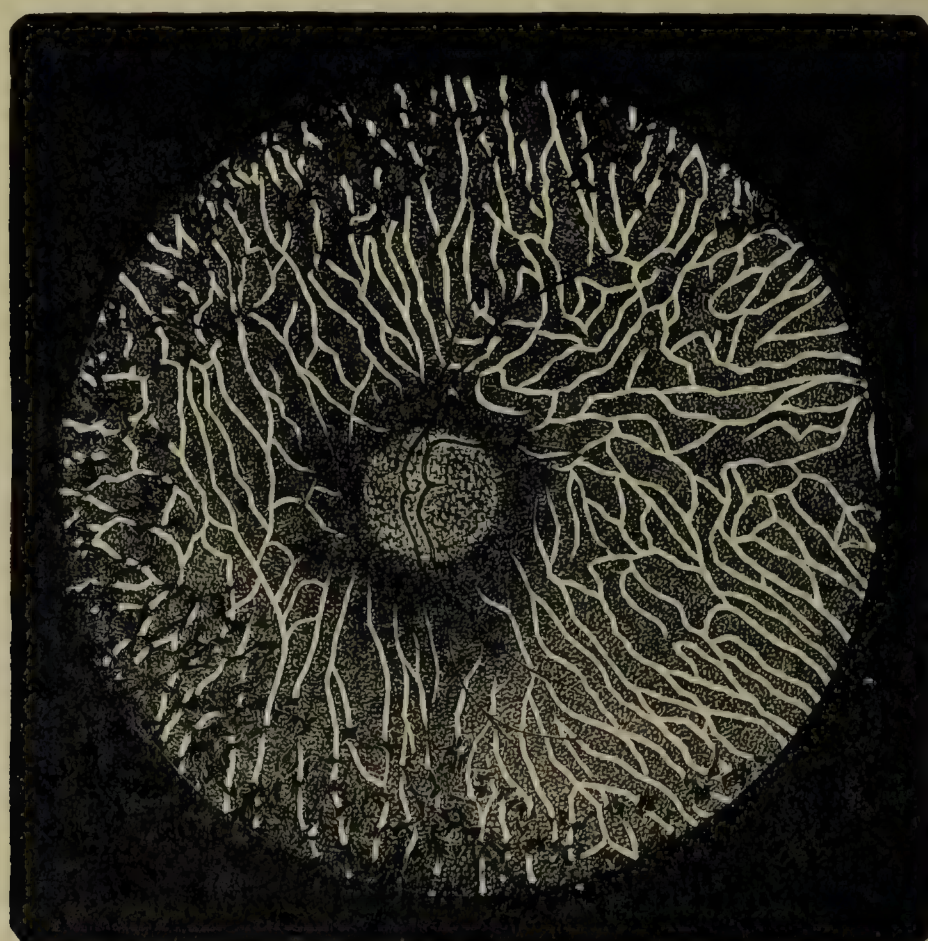
nasal  *temporal*

Fig. 357.—Retinitis Pigmentosa. (In part after Jäger.)

Owing to the disappearance of the pigment epithelium, the stroma of the chorioid is exposed, so that the bright-red chorioidal vessels with the darkly pigmented intervascular spaces are everywhere visible. For the same reason numerous branched, interconnected pigment spots are found in the periphery of the retina. This pigmented zone extends in a circle, although it comes closer to the papilla on the nasal than on the temporal side, where in fact, it lies so far to the periphery that it is not represented in the drawing at all. The papilla is of a dirty grayish-yellow color and ill defined. Of the retinal vessels, only the main trunks are visible, and these, especially the arteries, are greatly contracted.

Ophthalmoscopically, atrophy is characterized principally by the stenosis of the retinal vessels (Figs. 349D, 357), which in severe cases may amount to complete obliteration, so that the vessels are either transformed into white strands or have become altogether invisible. The retina may look otherwise unchanged and transparent, or it may bear traces of the antecedent inflammation. In every case the signs of a secondary atrophy can be made out upon the disc as well; its outline is indistinct and it is of a pale, dirty-gray color.

792. Retinitis Pigmentosa.—A special variety of atrophy which runs a very chronic course is the *pigmentary degeneration of the retina* (also called *retinitis pigmentosa*). This is marked by such characteristic subjective symptoms that the diagnosis can be made almost from them alone. The

persons affected with this disease, even when still young, complain that they see worse whenever the illumination is reduced, and particularly at night (hemeralopia). This state of things increases with the age, so that finally the patients are no longer able to go about alone at night, while in the daytime they still see quite well. The cause of this phenomenon is disclosed by the examination of the field of vision.

In the beginning of the disease the field, when taken with good illumination, shows a broad blind zone between the centre and the periphery, i. e., an annular scotoma (see § 271). Central vision is good, and because the periphery of the retina functions, orientation is good too, so that the ring-shaped defect in the field is not noticed. The peripheral portion of the retina, however, is undersensitive, and when the illumination is diminished it no longer functions, and then the periphery of the field disappears just like the portion that corresponds to the ring scotoma, so that the patient has left only the small central portion lying inside of the latter. In feeble illumination, therefore, orientation is no longer possible for him. And even the central portion of the retina is damaged, because the light sense in general (§ 258) is reduced, and hence the liminal stimulus is increased, i. e., the minimum quantity of light required to stimulate the retina is greater than in a normal eye. As the disease advances, the periphery of the retina becomes entirely blind, so that even in good illumination nothing but a small central portion of the visual field is left, and the patient can scarcely guide himself alone even in daytime. At the same time direct vision may still be so good that the patient is able to do fine work. Finally, central vision too is lost, so that complete blindness supervenes. This ordinarily is not the case until late in life (in the sixth decade or later).

In some cases of retinitis pigmentosa the main disturbance of sight, especially for reading, is caused by a cortical cataract. The densest part of this lenticular opacity lies right in the centre of the pupillary area, and hence these patients see worst when the pupil is contracted, i.e., in a strong light. They therefore form an exception to the rule that persons with retinitis pigmentosa see worst when the illumination is reduced. These cases can be recognized by the fact that dilatation of the pupil with atropine or homatropine enhances considerably their ability to read.

793. Ophthalmoscopic examination shows, as the most prominent symptom of the disease, the presence of small black spots in the retina (Fig. 357). These are branched, so that they have been compared to bone corpuscles or spiders; they are connected with one another by their processes, and are found especially along the veins. In the beginning of the disease they occupy only the most anterior portion (periphery) of the visible fundus corresponding to the equatorial region of the eyeball, but as time goes on, new spots keep forming farther and farther back, until at length they reach the macula lutea and the papilla. As fast as the retina becomes pigmented, the pigment epithelium becomes decolorized, so that the chorioidal vessels get to be more and more visible. With the increasing pigmentation of the retina the signs of atrophy of the retina and the papilla

become more and more prominent. Accordingly, what takes place is a gradual degeneration of the retina associated with a migration of pigment from the pigment epithelium into the retina (Fig. 358). The degeneration begins in the equatorial region and thence advances toward the periphery and toward the centre. In the same way, too, the retina gradually loses its function; the affected portions of it at first are simply less sensitive than before, being still stimulated to action by a pretty strong light; later on, they become completely insensitive.

The black spots in pigmentary degeneration of the retina are not always like bone corpuscles, but sometimes are also rounded or irregular, like the black spots in chorioiditis. Their characteristic distinguishing mark lies not so much in their shape as in their situation, which must be assigned to the retina. This is recognized from the fact that the retinal vessels, wherever they run by the black spots, are covered by them; hence, the spots must lie in front of the vessels—i.e., in the inner layers of the retina. (In the case of pigment spots in the chorioid we can distinctly follow the retinal vessels in their course over the spots.) Spots of pigment in the retina are not, however, limited to pigmentary degeneration of the latter; on the contrary, the migration of pigment into the retina may take place ultimately in any case of retino-chorioiditis. This is particularly true of syphilitic retino-chorioiditis, in which the pigment in the retina can, moreover, assume the bone-corpuscle shape, so that a picture quite similar to that of pigmentary degeneration may be produced (Förster). In chorioiditis, however, there are also usually present atrophic changes (white spots) in the chorioid, which are wanting in retinitis pigmentosa, (see Fig. 354, F). Nevertheless there are cases in which the diagnosis is very difficult and can only be made with the aid of the previous history and the careful testing of the function. Compare also remarks on Ophthalmia Hepatica, § 744.

FIG. 358.—RETINITIS PIGMENTOSA. SECTION THROUGH THE RETINA. Magnified 170 × 1.

The retina is bounded on its anterior surface by the membrana limitans interna, *i*, upon its posterior surface by the membrana limitans externa, *e*; the layer of rods and cones that succeeds the latter has disappeared altogether as a result of atrophy. Succeeding the limitans interna is a coarse-meshed network, *a*, which has originated from the sustentacular framework of the retina. The nerve fibres and ganglion cells which normally are inclosed in this framework have been completely destroyed. The vessels however, can still be seen, and are inclosed in pigment. One of them, *g* (very greatly contracted) has been divided longitudinally; another, g_1 transversely by the section. The succeeding layers of the retina—namely, the inner plexiform layer *r*, the inner granular layer, *k*, the outer plexiform layer, r_1, and the outer granular layer k_1,—are altered, and here and there contain pigmented cells.

Not only is pigmentation of the retina not confined to pigmentary degeneration of this part, but, on the other hand, such degeneration is not necessarily associated with the presence of pigment. There are cases of what are called *retinitis pigmentosa sine pigmento,* in which the same gradual attenuation of the retinal vessels, progressive atrophy of the optic nerve, hemeralopia, and ultimate blindness are observed as in retinitis pigmentosa, and only the migration of pigment into the retina is absent. These cases resemble *congenital hemeralopia,* in so far as in this also there is hemeralopia without any pigmentation of the retina being present. A further point of resemblance is that congenital hemeralopia frequently occurs in several members of the same family. The distinction between these cases and retinitis pigmentosa sine pigmento is that in the former the fundus presents no signs of atrophy of the retina or optic nerve, and vision remains good throughout life. The condition, therefore, is a stationary one as opposed to the slowly but surely progressing retinitis pigmentosa.

794. Retinitis pigmentosa attacks both eyes. It develops in childhood, and would seem to be congenital in many cases, although it usually is not discovered until some time after birth. Inheritance plays a great part in its production; retinitis pigmentosa occurs frequently in brothers and sisters, and also in several successive generations. The female members of the family are less frequently affected than the males. It is often found at the same time with other congenital anomalies, like deafness, mental weakness, harelip, or supernumerary fingers or toes, or with malformation of the eye, such as persistent hyaloid artery, posterior polar cataract, etc. After it has lasted a pretty long time, posterior cortical cataract usually develops. In almost a fourth of the cases the disease occurs in individuals descended from consanguineous parents. Herein apparently lies the explanation of the fact that pigmentary degeneration of the retina is so frequently associated with other congenital anomalies, since these latter also occur as a result of the consanguinity of the parents.

Treatment is powerless against pigmentary degeneration of the retina and the prognosis, therefore, is bad, since complete blindness inevitably supervenes—though not, to be sure, until after the lapse of many years.

For treatment we may as a satisfaction to the patient try mercury, potassium iodide, hypodermic injections of strychnine, the constant current, diaphoresis, and the like. Considering the extreme slowness with which the disease progresses, a hopeless prognosis should not be given in these cases, to the patients, who can often get adequate vision for a good many years by properly selected glasses, changed when required. If a cortical cataract develops, causing impairment of sight, this can be removed, sometimes with quite satisfactory results.

795. Other Forms of Retinal Degeneration.—Allied to retinitis pigmentosa is **Retinitis Punctata Albescens** (Gayet, Nettleship). This in all the rest of its symptoms agrees with retinitis pigmentosa, but instead of the pigmentation of the retina, shows hundreds of small white dots, which are distributed pretty uniformly over the whole fundus. These dots resemble druses (see page 704 and Fig. 329), with which, indeed, Leber believes they are identical. Other allied conditions are **Central** and **Diffuse Tapeto-retinal Degeneration** (Leber). In the former, which is nearly always bilateral and usually occurs in several members of the same family, white spots (druses) and varying pigmentary changes occupy the maculo-papillary region, and in the latter there are fine diffuse pigmentary changes. The central form develops usually after the age of ten, but sometimes not till after middle life or later, and causes progressive diminution and ultimate loss of central vision. The diffuse form causes total or nearly total blindness, which may begin at or about birth or not till later in childhood. It often occurs in several members of the same family and is frequently associated with cerebral degeneration producing idiocy (**Amaurotic Family Dementia**—a condition not to be confounded with the amaurotic family idiocy, described below, in which the site and nature of the lesions are different). It is a not infrequent cause of congenital blindness.

Another form of degeneration is characterized by a very extensive atrophy of the pigment epithelium and the chorioid, so that the normal red of the fundus is replaced by a great glistening white area. This may occupy nearly the whole fundus, a small portion round the macula and nerve being alone exempt (**Atrophia Chorioideæ et Retinæ Totalis, Chorioideremia**); or may form a peripherally situated, lobulated white ring, surrounding a more or less intact central red area (**Atrophia Gyrata Chorioideæ et Retinæ** of Fuchs). Pigmentation usually occurs as in retinitis pigmentosa, and, as in the latter, the condition often is found in several members of the same family (Leber).

Amaurotic Family Idiocy is a very rare affection developing in the first months of life with the following symptoms: The region of the macula lutea is occupied by a

grayish-white patch of the size of the papilla, having in its centre a small vividly red spot like that found in embolism of the central artery. The rest of the fundus is normal, although the disc becomes paler and paler and finally altogether atrophic. The changes are always found in both eyes, and are alike in both. The children, who are apathetic and show paralytic weakness of the muscles, gradually go blind. The apathy and paralysis increase, and in about two years the children die. Autopsy shows changes in the cerebral cortex and in the ganglion cells of the retina (Holden) and descending degeneration of the cord. The cause of the disease, which sometimes affects several members of the same family and is found especially among Jews, is unknown.

Cystoid Degeneration of the retina, perhaps due to œdema of the latter, occurs as a senile change near the ora serrata and in younger subjects in other parts of the retina, in eyes affected with a variety of diseases. Sometimes it occupies the macula, producing a hole there (§ 796). Rarely, if ever, is cystoid degeneration made out with the ophthalmoscope, being discovered only with the microscope in enucleated eyes (Leber).

796. Macular Atrophies; Hole in the Macula and Elsewhere.—The macula lutea has the most delicate anatomical structure of any of the tissues of the eye, and is hence also specially vulnerable. We find it therefore diseased particularly often, e.g., in myopia where it participates in the affection of the subjacent chorioid (§ 745). But the macula is also often injured in cases in which it is not in any way directly affected by the agent causing the injury. Thus a circumscribed affection of the macula often develops after contusion of the eyeball, or in the case of foreign bodies in the vitreous or in compression of the eye by orbital tumors. From rarefaction of tissue at this spot, sometimes ascribed to inflammation (*retinitis atrophicans centralis*) but more probably due to cystoid degeneration, an actual *hole* may be sometimes produced in the retina. This, too, may result from severe contusion of the eye and also from various diseases, inflammatory or degenerative, of the chorioid and retina. Under the ophthalmoscope, the hole looks like a deep red disc at the site of the macula (Knapp, Noyes, Kuhnt). This is usually one-third to one-half a disc diameter in width, and 0.3 to 0.5 mm. deep. The vision is very much reduced (Leber). Holes may also be produced in other parts of the retina either by trauma or retino-chorioiditis (Lister). They sometimes assume a horse-shoe shape.

Chance describes a form of retinal degeneration occurring in a brother and sister, marked by a sharply defined, red, crater shaped area in the macular region of each eye and by an area of diffuse pigmentation between the macula and disc.

797. White and Dark Striæ in the Retina.—We sometimes find in the retina *white*, irregular striæ, over which the retinal vessels pass either undeflected or with a slight bend. This condition, improperly called *retinitis striata*, seems to be due generally, if not always to reattachment of a detached retina.

Dark striæ occur under the following forms (Dimmer): (1) *Angioid streaks* (Doyne, Knapp), dark, wavy, branched or anastomosing striæ lying beneath the retinal vessels. These often form about the disc a ring from which striæ radiate to the periphery. The angioid streaks are sometimes red or may be accompanied by white striæ. (2). Black beaded striæ running in a radial or parallel direction from the disc (Lindner). These and the angioid streaks are probably due to hæmorrhage. (3) Yellowish or brownish (sometimes white), cobweb-like striæ, probably caused by folds in the retina (Lindner). (4). Black striæ, variously disposed, occurring after detachment of the chorioid (Schur and Lindner).

798. Anatomical Changes in Inflammation and Atrophy.—In inflammation the signs of inflammatory œdema exist, or those of a cellular infiltration due to extravasated white blood corpuscles; also extravasations of blood. The changes observed in the tissue elements of the retina itself are: 1. Fatty degeneration both of the nervous elements and of the supporting framework of the retina. 2. Thickening (sclerosis), especially in the nerve fibres of the fibre layer. 3. Free exudate under the form of homogeneous masses interposed between the tissue elements. The three changes just mentioned constitute the principal cause of the brilliant white spots occurring in many cases of retinitis (particularly in retinitis albuminurica). 4. Hypertrophy of the supporting tissue, which becomes the more prominent in proportion as the inflammation passes over

into atrophy. 5. Thickening of the walls (sclerosis) of the blood-vessels, causing contraction of their lumen or even their obliteration. 6. The migration of pigment cells into the retina. These are ordinarily regarded as derivatives of the pigment epithelium from which they emigrate into the retina, where they may undergo spontaneous multiplication. But according to Krückmann they are glia cells of the retina itself, which have simply taken up pigment from the cells of the pigment epithelium (Fig. 358).

When, after protracted inflammation, the retina has become perfectly atrophic, it consists of a reticulum which is derived from the supporting framework and contains pigment cells, but from which the nervous elements have absolutely disappeared. The blood-vessels are in great part obliterated and converted into solid strands of connective tissue.

IV. Detachment of the Retina

799. Objective Signs.—In detachment of the retina (*ablatio* sive *amotio retinæ*) we see with the ophthalmoscope, a delicate gray membrane that rises above the level of the normal fundus and projects forward into the vitreous (Fig. 359). Exteriorly the eye looks normal, only the interior chamber is often strikingly deep and the tension is also diminished.

The detached portions of the retina, being pushed forward, exhibit a lower degree of refraction than the rest of the fundus; they are, in fact, generally very *hypermetropic*. On account of this difference of refraction one cannot, using the erect image, see the detached and the adjoining portions of the retina distinctly at the same time; it being possible to do this only by means of the indirect method and then only by a certain effort of the accommodation, since the inverted images of the detached and the undetached retina are at different distance from the observer's eye. The examination in this case is much facilitated and also a wider field of view is obtained if we use a strong object glass (one of 2 inches or less focal distance—Dimmer). With the direct method we can determine the actual height of the various parts of the detachment above the surrounding retina (§ 411). If the retina is pushed very far forward it can actually be seen by oblique illumination when the pupil is dilated; a gray membrane, with the characteristic retinal vessels, being recognized deep down in the eye.

The *ophthalmoscopic appearance* of a detachment differs according as the case is one of serous detachment or of detachment due to a tumor or a cysticercus.

In *serous* detachment of the retina from the chorioid, the pigment epithelium remains in its place upon the latter. The retina, therefore, is at first transparent, but very quickly becomes cloudy, because it is separated from the chorioid, which in great part provides for its nutrition. The detached retina, accordingly, has a light, rather transparent gray color and a dull lustre. If some blood is mixed with the subretinal serum, the detachment acquires a greenish tinge. The retina lies in folds of greater or less size, whose tops show a whitish sheen; and it shakes all over when the eye is moved. The gray hue and dull lustre, the folds, and the tremulousness of the detached retina justify the comparison made between it and a gray silk or satin fabric.

Especially characteristic is the appearance presented by the *blood-vessels* that run over the detachment. Inasmuch as these follow the folds of the retina, they are very tortuous, and some of the bends are entirely concealed between the folds. The blood-vessels are dark red, indeed almost black, as though the blood circulating in them had been altered in character. This, however, is not the case, the dark color being really due to the fact that the blood-vessels are in part at least seen by transmitted light, since some light always passes through the detached retina and is then reflected from the more posteriorly placed chorioid. The blood-vessels, consequently, look dark for the same reason that opacities in the media appear black.

The sides of the detachment may merge by a gradual slope into the surrounding retina, or they may be baggy and overhanging. In extensive detachments the disc is partly or wholly concealed by the overhanging retina. Very flat detachments are

rather hard to diagnosticate. In this case the red hue of the fundus shows a slight gray cloudiness in the parts affected, and is traversed by low folds of a rather lighter gray but the main thing that enables the diagnosis of detachment to be made is the unusual tortuosity and the dark color of the vessels. When there is a flat detachment in the region of the macula, we can sometimes see at a point corresponding to the macula a pale-red spot in the detached retina.

The detached retina sometimes exhibits white patches, extravasations of blood, or pigmented spots. Particularly often there is found a rent in it (*ruptura retinæ*). This rent lies generally in the periphery of the fundus, and most frequently in its upper part. The edges of the tear gape and are frequently everted so that we can see between them for quite a long distance the more posteriorly placed chorioid. The latter owing to its

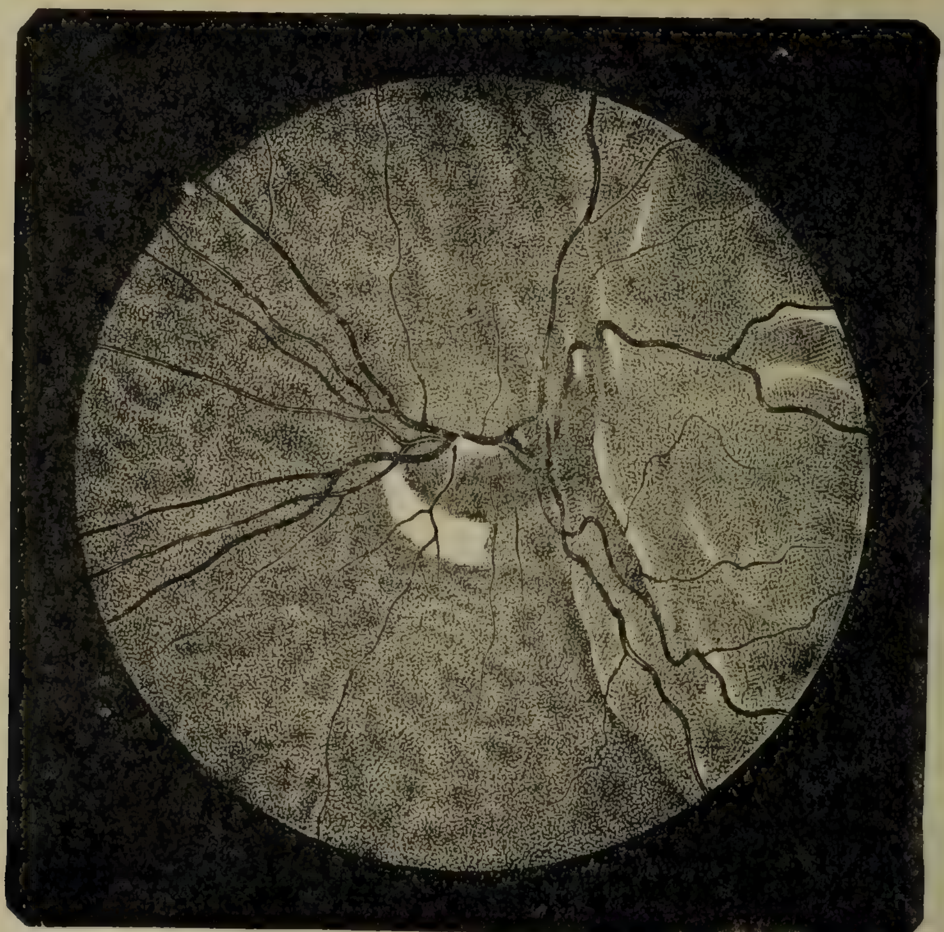


FIG. 359.—SEROUS DETACHMENT OF THE RETINA IN MYOPIA.

A woman of sixty-two, having previously been very myopic, had for four years suffered from a cataract in the right eye. After the removal of the cataract by operation, the lower half of the retina proved to be detached, thrown into folds, and tremulous. The upper border of the detached retina lay upon the lower border of the papilla, and concealed it. To the outer side the detachment is sharply demarcated from the normal fundus, while to the inner side it spreads out quite gradually into two or three flat folds. On the crests of the folds the detached retina looks lighter than in the depressions between them. The retinal vessels running downward from the papilla soon after they start disappear behind the overhanging edge of the detached portion of the retina, and are apparently interrupted at this spot. In their subsequent course they are distinguished by the remarkably sharp bends they make and which follow the folds of the detached retina. The outer side of the optic disc is bordered by a white atrophic crescent, which is about half the width of the papilla, and is attributable to the myopia pre-existing in the eye. The outlines both of this papilla and the crescent are hazy. The rest of the fundus is tessellated—i. e., displays the chorioidal vessels and the dark intervascular spaces.

vivid red color forms a marked contrast with the gray of the detached retina. According to Leber, rupture of the retina plays an important part in producing detachment in myopic eyes. For in myopic eyes delicate membranes develop on the interior surface of the retina specially at its anterior portion and by the subsequent shrinking of these membranes traction is made on the retina. This throws the retina into folds, and may even, because of the delicate structure of this membrane, rupture it, whereupon liquefied vitreous passes through the rupture and beneath the retina. This accounts not only for the detachment itself, but also for its sudden development.

A detachment due to *tumor*, is not tremulous, not thrown into waves, often rises abruptly from the surrounding fundus, and is sometimes nodular. At times vessels which are not retinal and belong to the tumor itself can be made out. Transillumination

gives a shadow, while in serous detachment it gives a reddish reflex. (Cf. §§ 763 and 804). A detachment due to a *massive exudation or hæmorrhage* has like characters.

In *cysticercus subretinalis* a rounded, rather sharply circumscribed detachment is found, beneath which may be recognized the bluish-gray cysticercus bladder with its lighter colored margin (Fig. 360). The detached retina is not tremulous, but spontaneous movements may be made out through it taking place in the bladder (cf. § 670). *Other entozoa,* found very rarely in the retina, are the echinococcus, filaria, and larvæ of diptera (Leber).

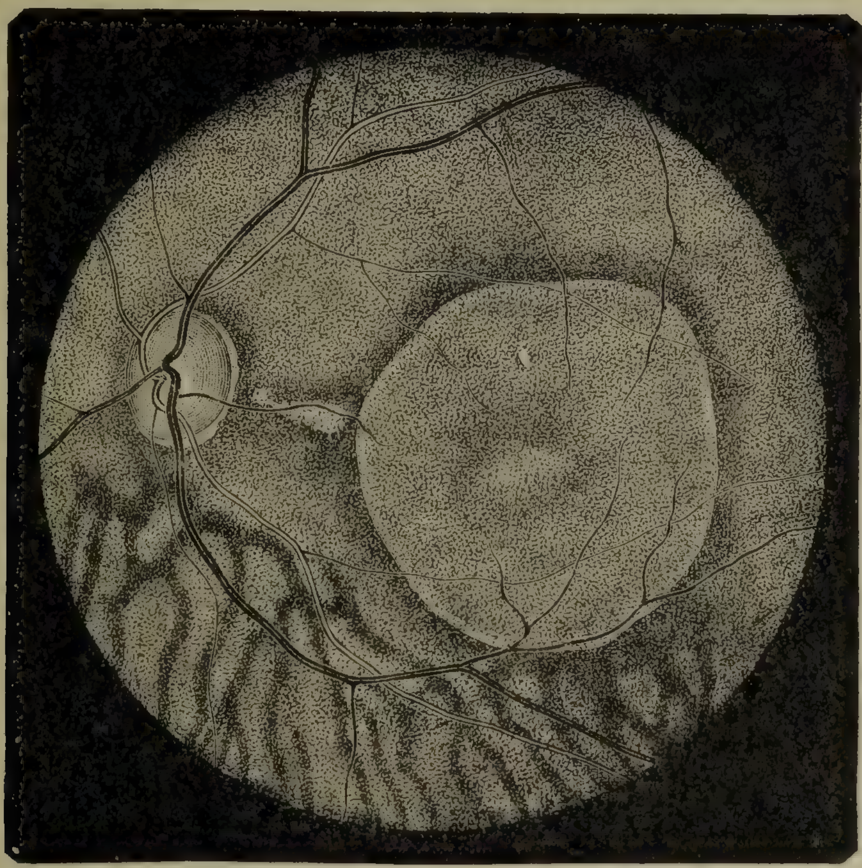


FIG. 360.—CYSTICERCUS SUBRETINALIS IN A WOMAN TWENTY-SIX YEARS OF AGE, WHO HAD NOTICED A SUDDEN DIMINUTION IN HER SIGHT TWO MONTHS PREVIOUS.

The papilla is encompassed on its outer and lower side by an irregular crescent. The region of the posterior pole of the eye is occupied by a bladder-like detachment of the retina. This detachment has a faint-gray hue, and allows the red of the fundus to appear through it, although dully. The edge of the bladder shows a light-gray, silky lustre, while in the centre of the bladder a bright yellowish-white speck is visible, which corresponds to the animal's head. The retinal vessels run up upon the bladder without showing any marked bend as they pass its edges. The bladder displays active spontaneous movements, in which the central white speck changes its position, shape, and size. Between the papilla and the inner edge of the bladder is an irregular, light-colored speck in the retina lying beneath a small retinal vessel. The upper part of the fundus is of a uniform red, the lower is somewhat albinotic, so that the dark chorioidal vessels stand out distinctly upon the bright-red background.

800. Symptoms.—The detached retina is wholly or partially insensitive to light. The visual field is correspondingly limited, and very frequently there develops, often quite suddenly, a positive scotoma, i. e., a black cloud or curtain which lies over objects in the portion of the field corresponding to the detachment. The examination of the field is hence of great importance for the diagnosis (see also page 752). Direct vision is preserved as long as the detachment has not extended to the macula lutea.

In the beginning of a detachment, objects frequently appear crooked (*metamorphopsia*), in consequence of the oblique position of the percipient retinal elements (see § 238). *Photopsia,* too, is caused by the traction upon the retina and often gives the first warning of the advent of the detachment. *Monocular diplopia* may occur if both undetached retina and an overlying detachment receive an image of the same object (Leber). Even the detached retina partially retains its sensitivity, its *light sense* is deficient, so that when the illumination is diminished, the defect in the field of vision is greater than in a bright light. The *color sense,* especially the appreciation of blue, is also defective (Salzmann).

801. Course.—The detachment is at first partial—i.e., is confined to one portion of the retina. It may develop at any spot whatever of the retina but most often at its upper part and usually (in case it is caused by fluid) changes its place afterward, for, as the subretinal fluid sinks on account of its weight, it depresses the detachment gradually to the lower part of the eye; hence detachments are most frequently found low down, although their original situation very often was at some other spot in the fundus.

Every detachment of the retina has a tendency to enlarge and finally becomes total. In the latter case we find the retina pushed forward en masse and connected with its bed at two points only—at the papilla and at the ora serrata. Then the detached retina forms a plaited funnel, beginning at the papilla and opening out in front, a shape which Arlt has well compared to that of the flower of a convolvulus (see Figs. 311, 341, 342).

In its later stages a detachment, whatever its origin, is frequently made inaccessible to observation with the ophthalmoscope, owing to turbidity of the media, especially of the lens and vitreous, and hence the diagnosis is rendered difficult or impossible. In such cases the diagnosis must be based upon two factors—upon the *field of vision* and the intra-ocular tension. If on account of the turbidity of the media, only light perception is left, the field of vision must be tested with the candle flame in a darkened room (see page 253). In detachment of the retina a corresponding limitation of the field will then be found. The *intra-ocular tension* in detachment is generally reduced, because the volume of vitreous is diminished; for the same reason the anterior chamber is often found to be deepened, because the lens has sunk backward. When in advanced cases of irido-cyclitis, irido-chorioiditis, or complicated cataract we find limitation of the field and reduction of the tension, we are warned that complete blindness due to total detachment of the retina and subsequent atrophy of the eyeball is imminent.

In that form of detachment which is produced by active propulsion of the retina away from the chorioid (see § 802) the tension is not diminished, but rather is increased. Hence, *increase of tension* with detachment of the retina is in doubtful cases an argument that the detachment is due to an intra-ocular tumor (Von Graefe).

The retina, when but recently separated, retains for some time its sensitiveness to light, and, if it soon becomes re-attached, may resume its function perfectly. Thus there is a possibility afforded of a *cure of the detachment* in respect to function as well as in other regards. Moreover, it sometimes happens that the sight improves very much, although the detachment remains and is not diminished in size. Such an apparent cure is brought about, whenever the detachment, after occupying the site of the macula lutea, afterward settles lower down, so that the macula resumes its function, and nothing is left but a peripheral contraction of the visual field, that causes but little disturbance. But a real spontaneous cure of retinal detachment by *re-attachment* of the retina does occur. This, to be sure, is very rare. Moreover it is imperfect in this regard; first, that the attachment is ordinarily not complete and, second, that the attached portion of the retina is so greatly damaged as to have very little functional power. Re-attachment is more likely to take place in the comparatively infrequent cases due to active propulsion by exudation or hæmorrhage (see § 802). A cure in the sense of re-attachment occurs in about 8.5 per cent of the cases, but restoration of even moderately useful vision occurs in from 3 to 6 per cent only (Leber). As a rule, a re-attachment can readily be made out with the ophthalmoscope. The fundus in the region concerned is spotted, as in old chorioiditis, owing to partial disappearance of the pigment epithelium, and is quite frequently traversed by rectilinear striæ, which are either pure white or lined with pigment, and which lie back of the retinal vessels (see § 797). The whole affected area is demarcated from the remaining, normal fundus by a sharp, usually curved line, which is yellowish or gray and is encompassed by pigment.

After a detachment has lasted some time, the retina becomes entirely *atrophic.* It has then lost its sensitiveness to light, and, moreover, becomes again transparent. At the same time the recognition of the detachment by means of the ophthalmoscope becomes more difficult, being now made mainly from the anomalous characters presented by the vessels.

802. Etiology.—The retina simply lies upon the chorioid without being connected with it anywhere except at the papilla and the ora serrata. In the dissected eye it can be lifted from its bed with the greatest ease. In the living eye the retina is kept pressed against the chorioid by the vitreous. A detachment of the retina, therefore, is possible only when either the pressure exerted by the vitreous ceases to act, or when the retina is pushed from its bed by a force greater than this pressure.

(*a*) The former variety of detachment occurs when through *disease of the vitreous* the pressure exerted by it is diminished or becomes absolutely negative—i. e., is converted into a traction. This occurs: 1. When quite a large quantity of vitreous has escaped, in the case of injuries or of operations. 2. When shrinking of the vitreous is produced by some disease of it. The most frequent cases of this sort are those in which the formation of exudates has taken place in the vitreous in irido-cyclitis or irido-chorioiditis. When these exudates become organized and shrink they draw the retina, to whose surface they are attached in places, away from the chorioid. This form of detachment, it is true, cannot be seen with the ophthalmoscope, since the media are too cloudy, but can be readily diagnosticated by the softening of the eyeball and the contraction of the field of vision.

When the retina is detached from the chorioid by alterations taking place in the vitreous, fluid transuded from the chorioidal vessels collects between the retina and the chorioid, owing to the negative pressure set up beneath the retina by reason of its detachment. This subretinal fluid is a quite albuminous, usually rather yellow serum, and hence detachments of the retina of this sort are called *serous.*

In high myopia a serous detachment of the retina, which can be seen with the ophthalmoscope (Fig. 359), often occurs without there having been any antecedent inflammation. It constitutes by far the greatest of the dangers that threaten very near-sighted eyes.

The way in which the detachment is produced has not yet been altogether cleared up, although most probably it is due to changes in the vitreous. Such changes, consisting of floating opacities and liquefaction, (or membranes—see page 750), do actually exist in the myopic eye. The same is conjecturally true of the so-called senile detachment which sometimes occurs in elderly people without any special cause and which should probably be attributed to senile changes taking place in the vitreous.

(*b*) Much less frequent are those cases in which the separation takes place in consequence of an *active propulsion* of the retina away from the chorioid. The causes of such a propulsion are: 1. An acute process of exudation from the chorioid, as occurs in purulent chorioiditis, in retinitis exudativa, and in phlegmons in the orbit. 2. Hæmorrhage from the chorioidal vessels, whether spontaneous or due to injury. 3. Tumors

of the chorioid or of the retina, and also a cysticercus developing benea the retina.

Among the causes of detachment of the retina must be mentioned *scars* that r main after perforating wounds in the region of the sclera. Such scars may be produc by accidental traumatism or by operation (cf. § 859). They attach the retina to t chorioid and the sclera, and by their subsequent contraction cause a pull upon t retina that leads to its detachment from its bed.

803. Treatment.—In serous detachment of the retina we try to secu the absorption of the subretinal fluid. This may be accomplished by pure medicinal means, i. e., by diaphoresis, by the use of purgatives or prepar tions of iodine, and by subconjunctival injections of salt solution, mercu oxycyanide, or dionin (see § 135), also, in case it is tolerated by the eye, b a pressure bandage applied with moderate firmness. Of operative proce ures, may be mentioned the superficial cauterization of the sclera with th galvanocautery, after the sclera at the site of the detachment has bee exposed by dissecting up the conjunctiva; also puncture and trephining the sclera (see §§ 976, 977) for withdrawing the subretinal fluid directly, resection of the sclera by Müller's method (§ 977). We undertake thes operations when the other measures have failed us, or when at the start w are dealing with a baggy detachment produced by a large amount of flui Puncture or trephining is made at the spot where the detachment is mos pronounced, for which purpose the site and extent of the detachmen must have been precisely determined beforehand with the aid of th ophthalmoscope.

Deutschmann has reported remarkably good results from division of the vitreou bands by a double-edged knife, or, when this fails, by the injection of an artificial vit reous. Birch-Hirschfeld withdraws the subretinal fluid and injects this (pure or mixe with salt solution) into the vitreous; but this operation has caused serious reaction.

In every form of treatment the patient should keep to his bed for sev eral weeks, since the detachment is increased in size by movements of th body. Hence it is that patients with detachment of the retina generall state that they see best in the morning after prolonged rest in bed, and tha their sight gets worse again as the day passes. Occasionally rest in bed alone, will effect a cure. It is most serviceable in recent cases when th detachment is still in the upper part of the retina. Later, when the detach ment has settled, it is useless (Leber).

By these methods of treatment it is generally possible in recent an not too excessive cases of separation of the retina to obtain an improvemen of the sight by partial attachment of the retina, and in especially favorabl cases even to cause the detachment to disappear completely. Unfortun ately, it is only in the rarest cases that these good results are lasting.

As a rule, after some time the separation develops anew, and ultimately in spite o all our therapeutic endeavors becomes total, so that the prognosis of retinal detachmen in general must be characterized as very unfavorable. The cause of the recurrences lie

in the fact that no treatment is able to do away with the lesion which usually lies at the bottom of the trouble—namely, the altered state of the vitreous. In inveterate cases or in total detachment of the retina, we had better abstain from any form of treatment. In total detachment of the retina, cataract usually develops later on, the eye becomes soft, and a slight degree of atrophy of the eyeball supervenes. Moreover, iritis is not infrequent in eyes with detachment of the retina.

Patients in whom a one-sided detachment has already occurred or in whom, because of their high myopia, detachment may occur at any time, should be warned to abstain from near work or straining eye work in general and to avoid excessive physical exertion.

When the retina is detached by means of a neoplasm, enucleation of the eye must be performed. A cysticercus occurring beneath the retina, may be extracted by an incision into the sclera, and the eye may thus be preserved in a condition serviceable for vision (§ 976).

V. Tumors of the Retina

804. Glioma.—Glioma is almost the only neoplasm which occurs in the retina. It is found only in children. In a case of it the parents notice that a bright, whitish, or golden-yellow reflex emanates from the pupil, which sometimes even is noticeable at a distance.

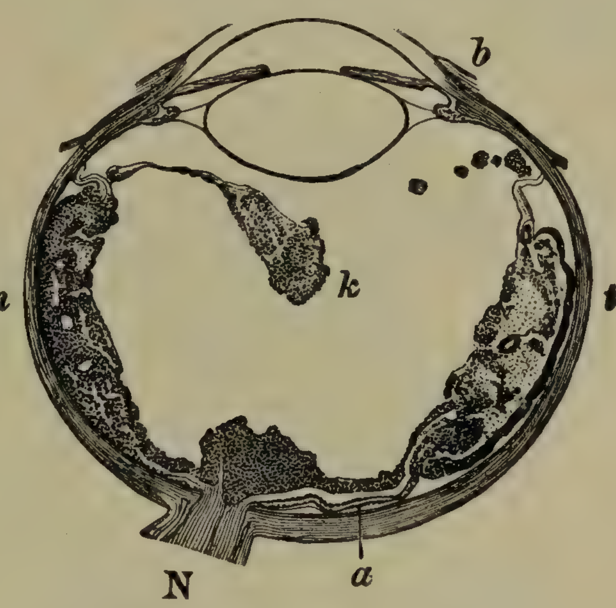


Fig. 361.—Glioma of the Retina. Magnified 2 × 1.

The glioma has spread over the entire extent of the retina. The latter on its nasal side, *n*, has been completely consumed in the formation of the new growth, while on the temporal side *t*, the external retinal layers are still in places preserved (at *a*). The pseudoplasm also covers the optic papilla, *N*, into the excavation of which it penetrates. In the anterior segment of the vitreous lie isolated nodules, the largest of which, *k*, is connected with the ora serrata. The anterior segment of the eyeball shows the effects of the increase of tension; that is, shows on the nasal side the apposition of the root of the iris to the sclero-corneal junction, while at the point corresponding to this on the temporal side the precursor of a staphyloma intercalare can be perceived under the form of an excavation, *b*.

By oblique illumination, or with the ophthalmoscope, we recognize as the cause of the reflex a light-colored nodular mass (the degenerated retina) situated behind the lens and covered over with minute vessels.

Glioma was called amaurotic cat's eye by Beer; amaurotic, because the eye is blind; and cat's eye, because like a cat's eye it shines in the dark. The same appearance may be produced by exudate into the vitreous and other conditions grouped collectively as *pseudo-glioma* (see § 753).

The *subsequent course* of the glioma shows the same stages that we have learned to recognize in the case of chorioidal tumors (§ 763). In the first stage, inflammatory symptoms are absent; the disease manifests itself only by the light colored reflex and the blinding of the eye. The second stage is characterized by the development of increase of tension. The eye becomes irritated and painful, and the child begins to suffer. Occasionally in this stage, the eye instead of becoming glaucomatous, shrinks and takes on temporarily the characters of phthisis bulbi (cf. § 764). Afterward, in the third stage, the tumor grows out from the eye, first of all along the optic nerve, then in other places as well, particularly through the cornea or

in its vicinity. The eye at length is transformed into a large, ulcerated painful, and readily bleeding mass (exophthalmia fungosa) which fills the whole orbit and projects out between the lids. In the fourth stage the tumor spreads to remote parts.

Metastases are uncommon, so long as the glioma remains confined to the eye, the disease in this respect contrasting favorably with sarcoma, in which early metastases are frequent (see § 766). On the other hand, extension along the optic nerve to the brain, is common and may occur early, a thing which rarely happens in sarcoma. The late metastases usually occur in the base of the skull or in the face (Leber).

The children finally die either from exhaustion or from the spread of the neoplasm to vital organs, especially the brain. The course of the disease from its very outset to its fatal termination usually extends over several years.

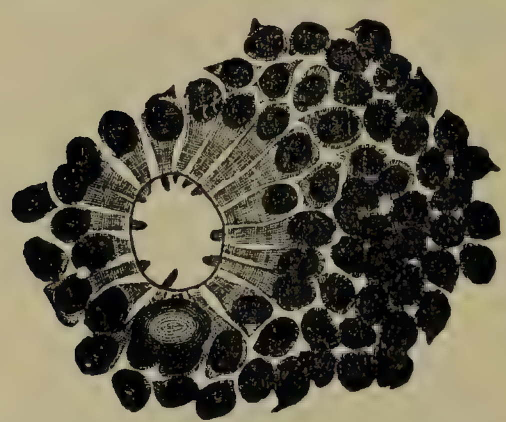


Fig. 362.—Glioma Retinæ. (After Wintersteiner.) Magnified 500 × 1.

The tumor consists of cells which have a single nucleus, and surrounding the latter a very thin, often scarcely visible, protoplasmic body. The left half of the drawing is occupied by a structure which resembles the cross section of a tubular gland. Surrounding its lumen are long cylindrical cells, whose nuclei lie at their peripheral extremities (rod and cone granules). The centrally directed extremities of the cells are bounded by a clear-cut line, the membrana limitans externa. From this latter short conical processes of protoplasm (rudimentary rods and cones) project into the lumen of the gland-like structure. Right below the latter lies a rather large, elliptical, very lustrous body—one of those hyaline concretions that sometimes occur in gliomata.

A few cases of spontaneous recovery by permanent shrivelling have been reported including one in which the process had invaded the orbit.

Glioma is a rare disease which is found in children only, and mostly before the fifth year of life.

Most cases develop before the age of three and probably over half before the age of two. Less than 2 per cent have been observed after the age of ten, and the oldest authentic case known was between fifteen and sixteen. Often it is observed at such an early age that its beginning must be dated back to fetal life. This, as well as the fact that several children in the same family are one after another affected with glioma, would argue that the cause of it is in many cases to be looked for in a congenital vice of development.

In almost 20 per cent of the cases of glioma both eyes are affected (Hirschberg, Wintersteiner, Lawford and Collins). In the familial forms bilateral involvement seems even more frequent.

Morbid Anatomy.—Glioma retinæ (Virchow), in contradistinction to intra-ocular sarcomata, is never pigmented. It probably develops from remains of the embryonic retina, that have not been differentiated into distinct layers. The tumor is composed of small cells and a very soft basement substance (Fig. 362). The cells consist of a nucleus surrounded by a very scanty amount of protoplasm which in many spots possesses minute processes. In many cases we find cells having the shape of cylindrical epithelial cells, arranged about an open space, so that the whole is like the cross section of a gland tubule (rosettes of Wintersteiner; Fig. 362). The multiplication of the tumor cells takes place mainly in the immediate vicinity of the numerous wide blood-vessels. Here, therefore, are situated the youngest cells, which keep pushing the older ones further and further away from the nutrient vessels. The older cell-layers consequently undergo necrosis. Thus in many cases the structure of the tumor becomes tubular, the vessels being surrounded by envelopes of living cells and lying with these cells in the midst of a necrotic mass. The process of overgrowth of the retina leads to its irregular thickening, and consequently to folding and detachment of it; but in many cases, as Fig. 361 shows, the detachment may for a long time remain confined to a small circum-

scribed spot. Neoplasm germs pass from the degenerated retina both into the chorioid and into the vitreous, where they develop into small independent nodules (*k*, Fig. 361).

Treatment consists in the promptest possible removal of the neoplasm. So long as the growth is still confined to the eyeball, it is suffcient to enucleate the latter, in doing which we take care to divide the optic nerve as far back as possible. Nearly half the cases thus operated on early are cured. When the tumor has perforated the eyeball and is growing outside of it, but is still confined to the orbit, complete removal of the neoplasm can still be attained through exenteration of the orbit (see § 999). But in this case rapid recurrences both in loco and in the neighboring lymphatic glands seldom fail to occur. Nevertheless, even in such cases the operation is indicated, because by the removal of the local focus of disease the child is spared much suffering. In inoperable cases x-ray or radium may check

Fig. 363.—Medullated Nerve Fibres. (After Jäger.)

The disc shows in its centre a whitish coloration, representing the physiological excavation. The temporal border of the disc is surrounded by an irregular chorioidal ring, while the upper and lower borders are concealed by the white fibrous masses that arise from them. These in places cover the retinal vessels, and especially the two arteries running outward and downward. At their peripheral borders the white masses break up into fibres.

local growth or relieve pain, and may be applied with advantage after enucleation to prevent recurrence. It should not, however, replace operation, when this is at all admissible.

Other Tumors of the Retina.—Carcinoma and sarcoma (primary and secondary) have been described as occurring, though very rarely, in the retina. Tumor-like products of inflammation (cysts and granulomata) also occur. For the so-called angioma of the retina, see § 788.

805. Injuries of the Retina.—*Ruptures* of the retina occur in consequence of contusions of the eyeball, even without perforation of the remaining tunics of the eyeball; but these cases of isolated laceration of the retina are extremely rare. The retina is much harder to tear than the chorioid, since in ruptures of the latter the retina is generally found

to be uninjured. Much more frequent are the spontaneous ruptures of the retina in detachment of the latter.

A transient alteration of the retina after contusion of the eyeball is the **Commotio Retinæ,** described by Berlin. This is characterized by a milk-white cloudiness of the retina, which occupies either the vicinity of the papilla or that part of the retina which corresponds to the point at which the effect of the blow was felt. In many cases also the spot diametrically opposite is found to be clouded. At the same time a moderate reduction of central vision and often also a contraction of the field of vision are present. The clouding of the retina disappears after some days, and with it also disappears the disturbance of vision that is produced. The condition, according to Berlin, is an œdema of the retina; according to Leber, it is a stretching of the retina with minute lacerations.

For hole in the macula produced by contusions see § 796 and for the large atrophic areas produced especially by gunshot injuries, see § 761.

806. Early Acquired and Congenital Anomalies of the Retina.—An early acquired anomaly is constituted by the *medullated nerve fibres.* At birth the fibres of the optic nerve in its orbital section are non-medullated. Within the first few months after birth they become medullated as far as the lamina cribrosa (Fig. 37), but within this, on the disc or in the retina, regularly remain without medullary sheath. In not a few cases however, some of the intra-ocular fibres, too, become medullated. We then see with the ophthalmoscope a brilliant-white spot adjoining the edge of the disc and splitting at its periphery into white fibres so as to have a flame-like look. Such spots are found most often at the upper and lower borders of the disc (Fig. 363), but may surround the disc completely, in which case the latter, by contrast, appears of a strikingly dark red. In rare cases white spots, formed of medullated fibres, lie within the disc itself or conversely far from it in the transparent retina. The retinal vessels are in places covered by the masses of white fibres. The vision may be normal or somewhat reduced, and the blind spot may be enlarged, though by no means always in proportion to the extent of the white patches seen in the fundus (Leber, Gradle). It is thus evident that the medullated fibres are not absolutely opaque.

Among the strictly *congenital anomalies* of the retina are absence of the retinal vessels (with aplasia of the optic disc), observed in anencephalia and microphthalmus and as a very rare anomaly in eyes that exteriorly appear normal· sac-like protrusions of the retina at the macula; coloboma of the retina (see § 769).

CHAPTER XXVIII

DISEASES OF THE OPTIC NERVE

I. Hyperæmia of the Optic Nerve

807. Simple hyperæmia of the optic nerve is marked by increased redness of the nerve and by haziness of its outlines, so that it contrasts but little with the red fundus surrounding it, which shows a radiate striation representing the course of the nerve fibres in the retina. To this are added dilatation and tortuosity of the retinal vessels.

A wrong diagnosis of hyperæmia of the papilla is often made if one forgets that in dark pigmented eyes the papilla looks redder and does so, too, when in the case of a faint uniform opacity of the vitreous it is seen through a turbid medium. Moreover, it must be remembered that the normal disc varies greatly in color. In doubtful cases a comparison with the other eye may decide, since if the disc was naturally redder than ordinary we should expect the unusual redness to be present in both eyes. Cf. Pseudo-neuritis (§ 406).

Hyperæmia of the disc is a frequent occurrence. It is not only a constant accompaniment of all inflammations of the retina and chorioid, but is found in connection with violent inflammations of the anterior division of the eye—e.g., in irido-cyclitis and even in corneal ulceration. It also occurs in connection with conjunctival congestion, refractive errors, and arteriosclerosis (see § 772).

II. Inflammation of the Optic Nerve

808. Inflammation of the optic nerve (neuritis optica), may develop at any spot whatever of the nerve. Of course, it is directly visible in the living eye only when the optic papilla, which is accessible to ophthalmoscopic examination, is involved. Such cases we call neuritis intra-ocularis, From them are to be distinguished those cases in which the inflammation is located in a portion of the optic nerve situated farther back (neuritis retrobulbaris). Since in this case the focus of inflammation cannot be seen, its existence must be inferred from the other symptoms.

If an inflammation of the retina is associated with pretty marked involvement of the optic nerve, or if, conversely, an inflammation of the optic papilla has extended so as to occupy quite a large area of the retina, the picture of *neuro-retinitis* or papillo-retinitis is produced. Almost all forms of retinitis, as well as of neuritis described in these pages, may appear under the form of neuro-retinitis.

(*a*) *Neuritis Intra-ocularis*

809. Symptoms and Course.—Neuritis of the optic papilla manifests itself externally by no sign except that the pupils are dilated, to correspond with the diminution or absolute loss of sight. With the ophthalmoscope we find the disc altered in color, being either white, gray or reddish, enlarged laterally so as to appear bigger than normal, and more or less prominent. The retinal arteries (Fig. 364, *a a*) are thinner, while the veins (*vv*) are distended, owing to compression of the vessels by the swollen optic

nerve, and are exceedingly tortuous, especially where they pass down upon the retina over the edge of the swollen papilla. Retinal hæmorrhages are often present near the disc and not infrequently also in the peripheral fundus.

Two forms of neuritis are distinguished: (a) *True or exudative neuritis, papillitis* (Fig. 364). In this the protrusion of the disc is slight so that there is no marked bending of the vessels at its border. The latter is ill-defined; the cloudiness and reddish or grayish-red discoloration of the disc, indicating the presence of exudation in it, are marked. The whole disc is affected,

Fig. 364.—Ophthalmoscopic Picture of Inflammatory Papillitis.

The disc is grayish-white, clouded, and appears considerably larger than it really is because it cannot be demarcated, from the surrounding retina which is likewise clouded and gray. To a diminishing extent the cloudiness and the radial striation extend out into the retina beyond. The retinal arteries, *a*, *a*, are contracted, the retinal veins, *v*, *v*, are exceedingly dilated and tortuous, and both are obscured in places. In the retina, adjoining the papilla, are found radially disposed, striate, red spots (hæmorrhages), *h*.

although not always to the same extent; it is often radially striate and may be diffusely clouded or mottled with white spots and hæmorrhages (Fig. 364, *h*). The vascular funnel is filled up, and the markings of the lamina cribrosa are invisible. The exudation, which in places may veil or completely cover the vessels, extends into the surrounding retina. Often, indeed, the picture of a neuro-retinitis is produced. (b) *Congestive neuritis.* This is really not so much a true inflammation as an inflammatory œdema due to constriction, (see § 810), and hence, is also called *choked disc* or *papillœdema* (Fig. 365). The outstanding features of choked disc are the swelling of the nerve, which even in the incipient stages has an overhanging edge (mushroom shape), and the great vascular engorgement. Hence, we see the disc protruding far into the vitreous, the veins very greatly distended, the arteries much contracted, and the vessels kinked or actually interrupted at the edge of the disc. On the other hand, the disc is fairly

defined, and both it and the surrounding retina are free from exudation. The swelling, which is gray and translucent, begins at a circumscribed portion of the disc, usually at its upper or lower margin, and gradually extends to the parts adjacent (Von Hippel), affecting the temporal side last (Kearney). At the outset and sometimes for a long time the vascular funnel remains exempt, and the markings of the lamina cribrosa are distinctly visible.

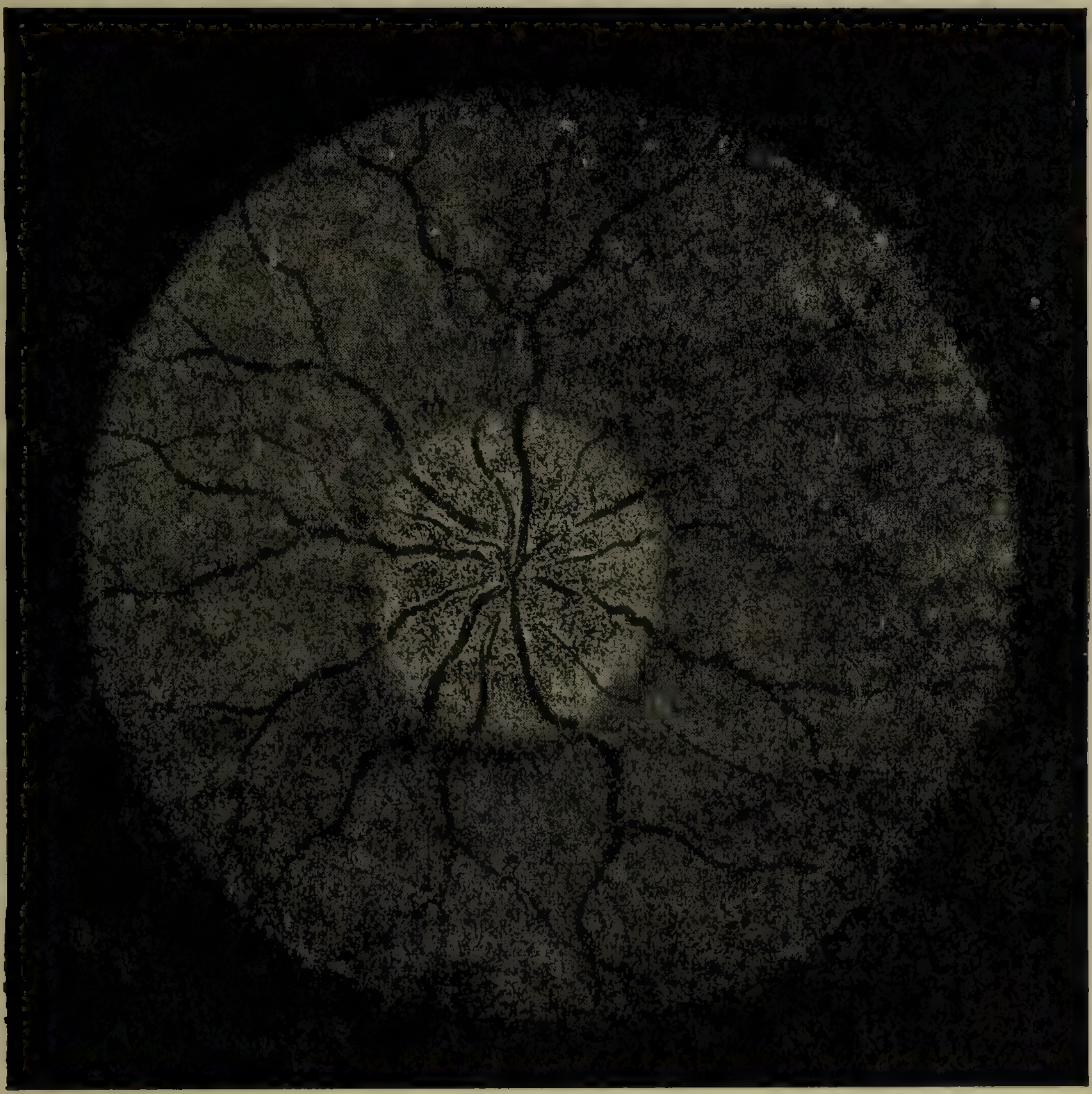


FIG. 365.—CHOKED DISC IN BRAIN TUMOR. (After Haab.)

The disc is swollen out into a mushroom-like protrusion so as to appear enlarged but still well defined (in contradistinction to inflammatory papillitis, Fig. 364). The arteries are thinner than normal, the veins somewhat distended and tortuous. Two of the veins show a brief interruption inside of the disc since here a bend of the vessel that dips down into the tissue of the papilla is concealed. Where the vessels descend over the overhanging margin of the disc to the retina, they all display a bend or an actual interruption. The retina surrounding the disc is the seat of a delicate radiating striation.

The *degree of protrusion* of the disc can be judged not only from the way the vessels bend over its edge, but, in the indirect method, by the parallactic displacement that the disc shows with reference to the retina and, in the direct method, by the difference in refraction between retina and disc. In choked disc this difference is regularly at least 2D and often much more. In making this estimate we must remember that the adjoining retina itself is usually swollen and prominent, so that the difference in level between it and the disc does not represent the real elevation of the latter. This prominence of the retina is evidenced by an increasing hyperopia, demonstrable with the ophthalmoscope but not necessarily manifested by any change in the subjective refraction, since the percipient layer (layer of rods and cones) may not be displaced (Cushing, Von Hippel).

In well-defined cases the distinction between the two conditions (papillitis and choked disc), is marked, but there are many *transition forms* between them.

The subjective symptoms consist in disturbed sight. In true neuritis this is generally very great; in bad cases the eye is absolutely blind. In

choked disc, the vision at the outset is good and may remain so for a long time, even when the swelling is considerable.

This is explained by assuming that in choked disc—in the beginning, at least—there is simply a state of œdema. The disturbance of vision is accordingly produced by compression of the nerve fibres due to the œdematous swelling. The degree of this compression, however, cannot by any means be determined from the ophthalmoscopic appearance, so that normal sight may be present along with a neuritis which with the ophthalmoscope appears very pronounced. In many of these cases the blindness does not come on till later, sometimes not till the advent of neuritic atrophy.

Characteristic of many cases of neuritis but especially of choked disc, is a sudden and momentary obscuration of sight, repeated many times a day. The light sense, especially the perception of the light difference (Percival), is often much reduced, especially in papillitis; in choked disc, the light sense may remain normal for a long time. Contraction of the field of vision is often found, sometimes under the form of hemianopsia. In choked disc, according to Leber, the contraction usually begins in the nasal field; but it may begin otherwise, and all sorts of defects may occur.

In some cases the only anomaly of the field is an enlargement of the blind spot. It has been thought that interlacing or reversed color fields (§ 274) are characteristic of neuritis due to increased intra-cranial pressure, but this is probably erroneous.

In pure choked disc, as distinguished from papillitis, the intraspinal pressure, as shown by lumbar puncture, is usually increased, although in cases due to skull injury this sign may be absent.

Neuritis runs a chronic course. It takes months, and in the case of choked disc sometimes one or two years, for the inflammatory or congestive signs to disappear, and then they are replaced by the symptoms of atrophy. The papilla grows paler, its outlines become once more clearly visible, and the vessels upon the disc, and in the retina are narrowed. This atrophy (*neuritic atrophy*, as it is called) is the greater the more intense the preceding neuritis; and the degree of the atrophy determines whether the vision grows better again after the inflammation has run its course, or remains permanently enfeebled, or is annihilated altogether. In any case, the prognosis of neuritis is serious.

810. Etiology.—Like the rest of the intra-ocular affections, neuritis is but rarely a local lesion; on the contrary, it usually originates in some deep-seated affection, and for this reason is almost always bilateral. The diagnosis of neuritis is therefore of importance, not only for the oculist, but for every physician engaged in treating internal disorders, as it affords him aid indispensable for the diagnosis of many diseases.

1. **Brain Diseases** are by far the most frequent cause of optic neuritis. They lead to disease of the optic nerve, either through producing congestion or through transfer of inflammation. (*a*) *Congestion* is chiefly of moment in those diseases of the brain which lead to an elevation of the pressure within the cranial cavity—i. e., most frequently in tumors of the brain and in hydrocephalus. A brain tumor, as a result of its growth, arrogates constantly more and more space to itself within the cranial cavity. Hence

as the skull is unyielding, there arises an increase in the intra-cranial pressure. Owing to this increase, the return flow of lymph in and about the optic nerve is impeded, and a stasis of lymph occurs producing an œdema of the nerve trunk. This œdema causes a compression of the central vessels—a compression which makes its influence felt sooner and to a higher degree in the central vein of the optic nerve than in the central artery. As there is constantly pouring into the papilla through the artery a quantity of blood which cannot be completely carried away again by the contracted central vein, venous engorgement of the optic nerve and consequently swelling of the latter are developed. This swelling of the nerve leads to its incarceration at the spot where it fits so tightly in the foramen scleræ, and consequently extreme œdema develops in the strangulated nerve head. This

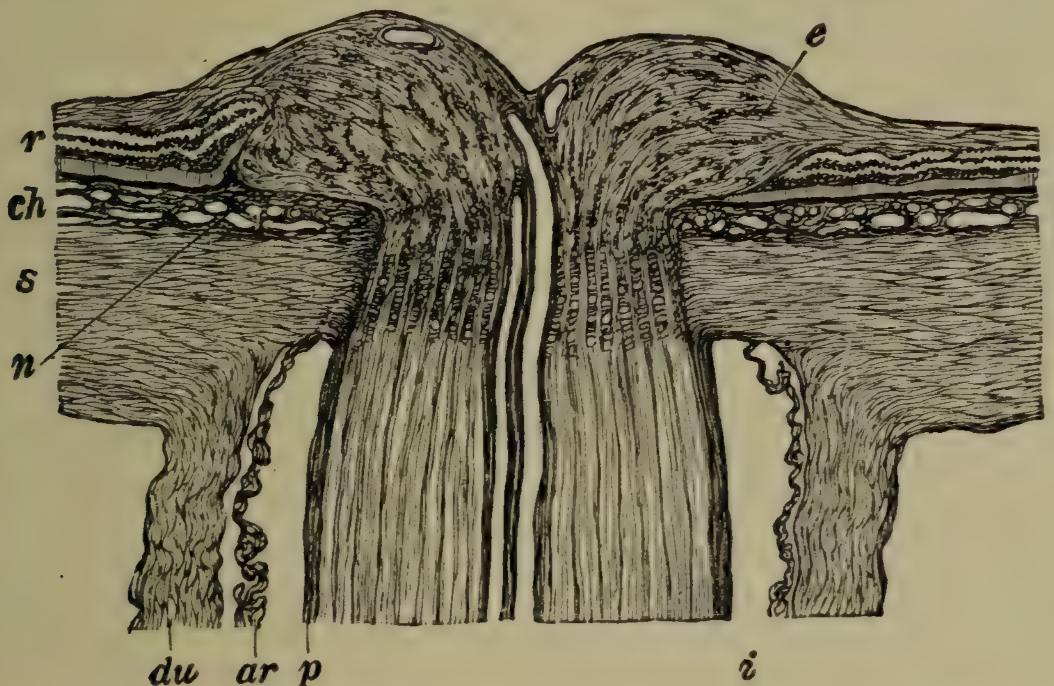


Fig. 366.—Longitudinal Section Through the Head of the Optic Nerve in Choked Disc. Magnified 14 × 2.

The disc is greatly swollen, so as to project above the level of the adjacent retina and form at the base an annular protrusion, *n*. There is a cellular infiltration, particularly along the minuter blood-vessels, *e*, for which reason the latter appear specially prominent. The retina, *r*, is thrown into folds about the circumference of the disc, in consequence of the swelling of the latter; the chorioid, *ch*, and the sclera, *s*, are normal, as is the optic nerve posterior to the lamina cribrosa. Here there is present simply a dilatation of the intervaginal space, *i*, through accumulation of fluid, by virtue of which the greatly folded arachnoid sheath, *ar*, becomes especially prominent; *du*, dural sheath; *p*, pial sheath.

œdema constitutes what is known as choked disc which, therefore, constitutes a very important symptom of increase of the cerebral pressure. (*b*) *Direct transmission* of inflammation from the brain to the optic nerve must be assumed to exist chiefly in those cases in which an inflammation is present in the brain itself, and particularly at its base, as is, for example, generally the case in syphilitic or tuberculous meningitis or in otitic processes. Here the inflammation is transmitted along the optic nerve and its sheaths to the papilla (*neuritis descendens*). The descending inflammation, as it travels down the nerve trunk, may be confined to the sheaths of the latter (*perineuritis*) or may also involve the peripheral portions of the nerve itself (*neuritis interstitialis peripherica*). The ophthalmoscopic picture presented may be that of a hyperæmia of the disc, a papillitis, or a choked disc. But the choked disc in this case is unattended by a rise of intra-spinal pressure, and in this respect differs from the ordinary choked disc

due to compression. Quite often in descending neuritis the sight is unimpaired. The spinal fluid shows the changes characteristic of the causal disease (syphilis, meningitis) (Von Hippel).

The part that increased *intra-cranial pressure* plays in causing choked disc is evident from the fact that often the swelling of the disc diminishes directly after the pressure has been reduced by lumbar puncture. Per contra, the disc will bulge if the intra-ocular pressure is suddenly reduced, even though the intra-cranial pressure is normal. Thus we see a disc that has been excavated by glaucoma swell out again when the tension of the eye has been reduced by operation. On the other hand, if the intra-ocular pressure rises unduly, the optic disc recedes before it.

According to Schmidt and Manz choked disc is primarily due to dropsy of the nerve sheath (Fig. 367). That is, the cerebrospinal fluid, squeezed into the intervaginal space by the heightened intra-cranial pressure, produces pressure on the nerve and the consequent congestion. But such dropsy of the sheath is by no means necessarily present in choked disc. Accordingly, other theories have been propounded. Perhaps the most probable is that of Schieck. He thinks that the fluid passing from the brain into the optic nerve meets there the fluids coming from the eye and with them passes out along the central vessels of the nerve into the orbit. If the fluid escaping from the brain is under undue pressure it will force back the fluid coming from the eye and cause pressure on the central vein and engorgement of the nerve head.

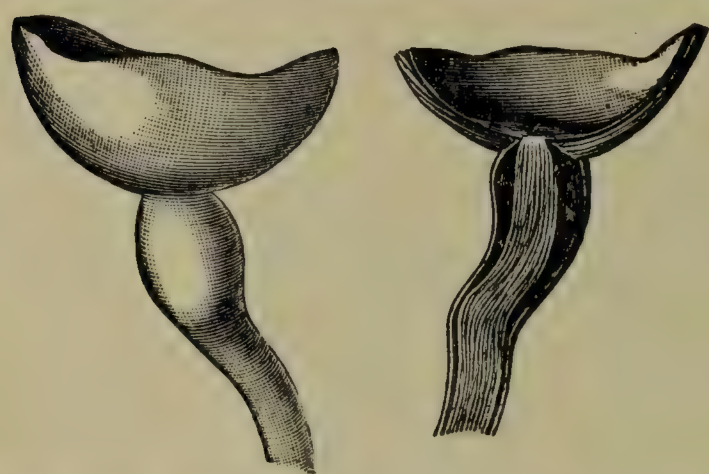


FIG. 367.—HYDROPS VAGINÆ NERVI OPTICI. (After Pagenstecher.)

Upon the left side is seen the ampulliform swelling of the optic nerve in its anterior portion. Upon the right are represented the relations as seen after making a longitudinal section of the nerve. One can see how the swelling of the nerve is caused by the distention of the outer sheath, which is separated a good way from the trunk of the nerve; and can recognize the projection of the papilla above the level of the retina.

The diseases of the brain which are complicated with optic neuritis are partly focal, partly diffuse affections. Among the former, it is above all the *tumors of the brain*, which result in neuritis, usually under the form of choked disc. Neuritis in this case is so frequent—it is said to be wanting in only 10 per cent, according to others in 20 to 30 per cent of cerebral tumors—that it forms one of their most important symptoms. This symptom is the more deserving of consideration inasmuch as a cerebral tumor may often run its course for a long time without producing any other positive symptoms—e.g., it may simply cause headache, or even this may be wanting. *Accordingly, in every case in which there is a suspicion of the existence of a cerebral affection, the fundus of the eye should be examined with the ophthalmoscope.* This is the more necessary, since choked disc sometimes fails to manifest itself by any disturbance of the vision.

With tumors of the brain a special form of *neuro-retinitis* sometimes occurs, in which, besides the appearances in the optic papilla, minute splashes of silvery lustre are visible in the macula lutea, so that a picture resembling that of retinitis albuminurica is produced (retinitis stellata, see § 779).

The *size* of the new growth is not of decisive importance in determining the development of a choked disc. Choked disc has been seen with tumors which scarcely reached the size of a walnut; at other times it is absent, although the tumors are very large. Evidently other factors have to be considered and especially the *site* of the tumor. Tumors in the posterior fossa (e.g., tumors of the cerebellum) are the ones most often causing choked disc. In the posterior fossa of the skull the space is hemmed in by the tentorium which stretches over it and is but slightly yielding. Thus there is readily produced compression of the aqueduct or of the vena magna Galeni, and consequently a stasis of the cerebrospinal fluid in the anterior ventricles of the brain (dropsy of the ventricles). On the other hand, the fact that basal tumors, e.g., those of the hypophysis,

rarely produce choked disc is accounted for on the ground that such tumors may block the orifice by which the intervaginal space of the optic nerve opens into the cranial cavity, so that the cerebrospinal fluid does not enter the space. Tumors of the frontal lobe give a characteristic picture (Kennedy). In the first stage (which may be lacking) there is bilateral papillœdema with good vision; in the second, subsidence of the papillœdema in the eye on the side of the tumor and the development in this eye of a central scotoma and subsequent optic atrophy, while papillœdema with good vision persists in the other eye. In the third stage the second eye also develops a central scotoma and atrophy. There is loss of the sense of smell on the side of the tumor.

In many, perhaps the majority of cases—though not with the regularity that Horsley contends for—the swelling of the disc due to brain tumor is greater in the eye on the same side as the tumor.

With tumors of the brain there may occur not only choked disc, but also descending neuritis and simple atrophy of the optic nerve. *Descending neuritis* takes place when the tumor excites, in its immediate neighborhood, an inflammation which is transmitted to the optic nerve. Descending neuritis, however, due to this cause is rare. *Simple atrophy* may be produced because a tumor exerts a direct pressure upon the chiasm or the optic tracts, and thus causes their effacement. An example of this is furnished by the tumors of the hypophysis, which, by pressing upon the chiasm cause atrophy of the optic nerves with temporal hemianopsia (see § 276). In other cases the tumor causes an accumulation of fluid in the third ventricle, so that the greatly distended anterior inferior extremity of the ventricle presses upon the chiasm. In this way amaurosis develops with cerebral tumors, either without any ophthalmoscopic evidence at all or under the guise of a primary atrophy.

Among the *focal affections* of the brain which may, although but rarely, cause neuritis must be enumerated foci of softening, abscesses, thrombosis of the sinuses, aneurysms, apoplexies, and cysts (among these, cysticercus and echinococcus cysts). Among the *diffuse affections*, disseminated sclerosis, acute and chronic meningitis, and hydrocephalus give rise to neuritis. The two affections last named, together with tubercles of the brain, are the most frequent causes of neuritis in children. Frequently such children are not brought to the oculist until later in life, when he finds a neuritic atrophy as the cause of the blindness, and can determine from the history of the case that a severe cerebral affection has preceded it. This form of blindness is incurable. Not to be confounded with it are those rare cases in which children become blind without known cause and without any ophthalmoscopic change in the fundus. This variety of blindness, the cause of which is at present unknown, sometimes disappears (Nettleship). Some cases of neuritis due to hydrocephalus are known in which a continual dropping of fluid (cerebrospinal fluid) takes place from the nose. Neuritis also occurs sometimes in *malformations of the skull* (particularly the kind known as oxycephaly or tower skull) and *injuries* of the skull (especially fractures on the base, with consequent meningitis). Penetrating or non-penetrating injuries of the skull due to projectiles are frequently followed, in from twelve to forty-eight hours, by a choked disc, which usually affects the vision but little and is not usually followed by atrophy. It yields readily to decompression and may disappear spontaneously (Jessop, Lister).

Neuritis has also been observed as a rare complication in *spinal diseases*, particularly in acute myelitis, in tetany and in multiple neuritis.

811. Other Causes of Neuritis.—2. *Syphilis*. This is a frequent cause of neuritis. The optic nerve may be attacked by the syphilitic affection directly. In other cases it becomes affected indirectly, owing to the development in the cranial cavity or in the orbit, of inflammations or of tumors which result from syphilis, and which secondarily implicate the optic nerve. Descending neuritis (§ 810) is usually due to syphilis (Von Hippel).

3. Acute infectious *febrile diseases*, chronic *disturbances of nutrition* of various kinds, and *poisoning* especially by lead.

Thus neuritis occurs, although rarely, in the acute exanthemata (measles, smallpo scarlet fever), and also in diphtheria, pneumonia, influenza, and whooping cough.] typhus it is said to be very frequent, appearing either under the guise of an exudati neuritis (papillitis) or a choked disc (Arnold). The chronic diseases in which neurit occurs are albuminuria, diabetes, tuberculosis, chlorosis, and anæmia. Congesti neuritis has also developed after thyroidectomy (Sherer), and from infection originati in the teeth, tonsils, and accessory sinuses of the nose (cf. § 815). In women, neurit sets in simultaneously with disturbances of menstruation, with pregnancy, or wit lactation (see § 821). These cases usually give a good prognosis, even when they get t the point of producing complete blindness for the time being. Externally similar to th conditions just spoken of, but etiologically quite different, are those cases in which a amenorrhœa and a papillitis develop as symptoms running parallel with one anothe both being the result of a brain disease (tumor or serous meningitis).

Of *poisoning* in the narrower sense of the term, there should be mentioned beside lead poisoning, that produced by alcohol, methyl alcohol, iodoform and arsenic (includin atoxyl and similar preparations). The optic neuritis occurring in syphilitics after the us of salvarsan is not to be ascribed to the toxic action of the latter, but to the liberatio of spirochætæ or their toxins, due to the provocative action of an insufficient dose of th salvarsan. Such a so-called *neuro-recidive* is avoided if mercury is given with or befor the salvarsan. When it occurs, so far from being an indication for dropping the salvarsa it rather calls for a more thorough-going administration of the drug, and this, in fac generally relieves the condition. Among *other causes* of neuritis occasionally observe is great chilling of the body; possibly here the real cause is a sinus infection. Lastly there should be mentioned the cases of neuritis and atrophy of the optic nerve that hav been produced by a stroke of lightning or electric shock.

4. Acute anæmia after great *loss of blood*, the most frequent variety being that due to hæmorrhage from the stomach and to metrorrhagia. In these cases, blindness, as a rule, does not set in until some days after the hæmorrhage, and is generally incurable.

5. *Heredity*. Sometimes without any apparent cause, neuritis attacks several members of the same family, often in different generations. Such hereditary neuritis is usually retrobulbar (see Leber's disease, § 815).

6. *Orbital affections*, such as inflammations or new growths in the orbit or tumors developing upon the optic nerve itself. These are the only cases in which neuritis can with certainty be regarded as a purely local disease.

812. Morbid Anatomy.—*Inflammation* of the optic nerve starts from its connective-tissue portions—that is, from the sheaths and the connective-tissue septa. In the sheaths there is found, besides the dropsical condition already mentioned, actual inflammation with a formation of a cellular exudate (perineuritis). Within the trunk of the optic nerve the inflammation attacks the septa which show thickening with multiplication of their nuclei (interstitial neuritis). Owing to this, the bundles of nerve fibres, which are inclosed by them, are compressed and, undergoing atrophy, are destroyed. Accordingly, in neuritis the nerve fibres act mainly a passive part.

In *choked disc* the inflammatory symptoms are limited to the nerve head, while the trunk of the nerve back of the lamina cribrosa is fairly normal. The main feature is the accumulation of œdematous liquid in the nerve head, causing it to protrude like a mushroom into the interior of the eye, and to become distended at its base so as to form an annular swelling there (Fig. 366, *n*). The retina is pushed aside and thrown into folds by the enlarged optic nerve. Besides the œdema there are also found extravasations of blood, swelling of the nerve fibres, and the evidences of a scanty cellular infiltration, particularly along the blood-vessels (Fig. 366, *e*). Later on, the cellular exudation becomes more and more prominent and in the subsequent course of the disease leads to a new formation of connective tissue within the papilla, due to organization of the exudate.

By the subsequent shrinking of the connective tissue, the fibres of the optic nerve are rendered atrophic, and the picture of neuritic atrophy of the optic nerve is produced. We then find in place of the papilla a network of connective-tissue strands, and among them, blood-vessels whose walls are thickened.

813. Treatment.—Treatment must be directed first of all against the lesion which causes the neuritis. In syphilitic cases energetic treatment with salvarsan combined with mercury is indicated; and, if the former appears to aggravate the condition, the indication is rather to push than to discontinue the remedy (see § 811). The salvarsan or a salvarsanized serum may be administered by intraspinal injection (§ 141). Local treatment, in addition to a suitable regimen of the eyes, comprises diaphoretic measures, and the administration of absorbent remedies, such as iodide of potassium, mercury, and in non-syphilitic cases, the salicyl preparations (Leber). In increase of brain pressure, decompressive operations like trephining of the skull and lumbar puncture are to be considered.

Trephining produces a permanent reduction of the intra-cranial pressure in brain tumors and in hydrocephalus, and thus subsidence of the neuritis and improvement of sight. It is particularly advisable in those cases in which the brain lesion itself is curable, and hence especially in meningitis serosa. In this disease by trephining promptly it may be possible to prevent a permanent blindness being left after the main disease has been cured. Even in inoperable brain tumors, trephining is indicated as a palliative measure for relieving pain and saving the sight. Horsley says that no case of optic neuritis, not due to general infectious disease or toxic causes, should be allowed to go on to blindness without operation. In pretentorial growths the site of the trephining should be temporal, in subtentorial growths should be suboccipital (Frazier cited by De Schweinitz).

Lumbar puncture is used not simply as a therapeutic but also as a diagnostic measure, to distinguish a choked disc, in which the intraspinal pressure is usually increased, from a papillitis, in which it often is not (§ 809). But it should be applied with caution, especially if a tumor of the posterior fossa of the skull is suspected, for in these cases the operation has sometimes caused sudden death. As a therapeutic measure it is imperatively indicated if sudden blindness develops with choked disc (Von Hippel).

(*b*) *Retrobulbar Neuritis*

814. Retrobulbar neuritis is a name given to an inflammation which is located in and confined to the orbital division of the optic nerve. It must be distinguished from a descending neuritis, which likewise affects the retrobulbar segment of the nerve, but ultimately also extends to the disc. A descending neuritis is transmitted along the nerve sheaths and the exterior portions of the nerve (§ 810), while a retrobulbar neuritis, in the restricted sense here used, is confined to the axial portion of the nerve trunk and is hence called *axial neuritis*, (Wilbrand and Saenger). Because in axial neuritis the inflammation is not transmitted to the disc, the ophthalmoscope shows in the papilla either no changes at all or changes that are insignificant and not characteristic; but later, after the disease has subsided, the signs of atrophy frequently make their appearance there. This is the case when destruction of the fibres of the optic nerve has taken place within the region occupied by the focus of inflammation. The peripheral portions of the divided fibres then undergo an atrophy, which is slowly transmitted to the papilla, where it becomes visible with the ophthalmoscope (descending

atrophy). Owing to the absence of distinct ophthalmoscopic changes i the recent cases, the diagnosis of retrobulbar neuritis must be made fron the other symptoms, and mainly, in fact, from the vision. In a few case this is so affected as to produce complete blindness, but in most cases ther is simply a central scotoma. The light sense, particularly the perception o the light difference, also is reduced.

A central scotoma due to optic-nerve disease is distinguished from one due t central chorioiditis or retinitis by the fact that in the former there is no change in th apparent size or shape of objects in or about the scotoma (retinal metamorphopsia, § 23 and that the first colors to disappear are red and green. In primary disease of the macula retinal metamorphopsia is usually present, and a scotoma for blue develops before tha for red or green (Mauthner.)

815. Course and Etiology.—Retrobulbar neuritis is either acute o chronic in its development. One portion of the toxic amblyopias (see § 820 is thought, by many, to be a chronic retrobulbar neuritis. In general retrobulbar neuritis affords a good prognosis, since in cases which ar not too far advanced, the sight can usually be restored to normal.

The *acute* form of retrobulbar neuritis is characterized by the suddenness with whic the disturbance of vision develops. In the severe cases this failure of sight may attai such a degree that in a day or two all perception of light is abolished. Often, even in thi case, the periphery of the field clears, leaving a very large central scotoma, which ma persist or may itself clear up. Other forms of field defects may be found. Externally the eye looks normal; the pupil, however, is often a little dilated. The ophthalmoscope too, shows scarcely anything besides some distention of the retinal vessels.[1] These symptoms are often accompanied by violent headache or by dull pain in the orbit, the latte being aggravated if the person moves his eye or tries to push it back in the orbit. Sometimes both eyes are attacked at the same time.

Acute retrobulbar neuritis is either the result of an *inflammation of the surroundin orbital tissue* or there is an idiopathic inflammation of the nerve. Cases of the forme kind originate most frequently in the nose in cases in which a violent coryza has developed from the effect of cold, influenza, etc. The catarrh of the nasal cavity passes to the accessory sinuses of the nose and especially to the ethmoid cells, and thence the inflammation is transmitted directly to the orbital tissue and to the optic nerve (compare § 886). As a direct proof of the presence of an inflammation of the retrobulbar cellular tissue, we not infrequently find in such cases a slight amount of exophthalmus, and also paralysis of some of the eye muscles, especially those which lie close to the inner and upper walls of the orbit (internal rectus, superior oblique, levator palpebræ, and the superior rectus). In neuritis due to disease of the posterior accessory nasal sinuses (rhinogenous neuritis), a symptom very often, though not invariably met with is an enlargement of the blind spot (Van Der Hoeve's sign). The enlargement is more marked for colors than for white and may be present before a central scotoma appears or may occur in cases in which no serious disturbance of vision develops. When then, as so frequently happens, a cold is given as the cause of the trouble, the nose is always to be examined, since upon the result of this examination will depend the treatment; and if there is a demonstrable disease of the accessory sinuses these will require to be treated and, to effect this, will often even require to be opened. In other cases cold appears to cause acute retrobulbar neuritis, as it does other peripheral neuritides, directly, i. e., without implication of the nose. These are examples of *idiopathic inflammation* of the optic nerve, which must also be assumed to exist in disseminated sclerosis, in which the retrobulbar neuritis so often occurs as a very early symptom that by many the great major-

[1] Sometimes, on the contrary, ischæmia of the retina is present when the central vessels are subjected to compression in the inflamed portion of the optic nerve.

ity of all cases of acute retrobulbar neuritis are attributed to this disease. Much more rarely, acute retrobulbar neuritis occurs in myelitis, in diseases of the tonsils or teeth, and also in general affections such as acute infectious diseases, toxic conditions, and various disturbances of nutrition including tuberculosis and affections of the female genital system (pregnancy, lactation, menstrual disorders), in which an acute retrobulbar neuritis may be found in place of a papillitis (cf. § 821).

An acute bilateral retrobulbar neuritis occurs as a *hereditary affection* (Leber's disease). The heredity is regularly sex linked (see § 103). Usually males alone are attacked and these generally between the ages of 13 and 28. When it occurs in women it develops not infrequently at the time of the menopause. A permanent central scotoma is generally left. The disease has been ascribed to abiosis (page 100), to autotoxis due to some hereditary influence affecting the endocrine glands, and to disturbances in the region of the hypophysis. In any event, in all cases of this sort the nasal sinusea and the pituitary region should be examined to discover some possible cause that may be removed. Some cases have been observed which presented all the other signs of Leber's disease, but in which no hereditary factor could be discovered.

In benign cases of acute retrobulbar neuritis (those due to cold or to nasal affections) the disease *subsides* very quickly, the sight gets better every day, so that in from two to four weeks it is once more pretty nearly normal. In other cases the cure takes a longer time and is then ordinarily incomplete, a central scotoma remaining, especially in the hereditary form. But in rare cases there is left a total, permanent blindness, so that it is impossible at the outset of the disease to state the prognosis with certainty.

Recurrences of the disease are sometimes observed even after the lapse of years.

The *treatment* of the disease requires above all the consideration of the causal factor (especially the nose). In the way of symptomatic treatment energetic diaphoresis proves particularly effective in the acute stages. As in papillitis, absorbents may be used; and the salicylates or aspirin may be given, especially when pain is present.

III. Atrophy of the Optic Nerve

816. Atrophy of the optic nerve develops either as a primary affection or as secondary to an antecedent inflammation. We accordingly distinguish between simple and inflammatory atrophy.

(*a*) *Simple* (primary or genuine or non-inflammatory) atrophy is distinguished by the papilla becoming paler, and at length perfectly white or bluish-white and also becoming sharply defined and slightly excavated (atrophic excavation; see § 827); the gray dots of the lamina cribrosa are visible more distinctly and over a larger area; the more minute blood-vessels of the papilla itself have disappeared, while the retinal vessels are not markedly altered (in contradistinction to inflammatory atrophy, in which the latter are narrowed, too). The *causes* of simple atrophy of the optic nerve are:

1. *Cerebrospinal diseases* including first of all tabes, which is by far the most frequent cause of the simple form of optic-nerve atrophy; second, a condition allied to tabes and like it a late manifestation of syphilis, namely, general paresis; third, disseminated sclerosis. In tabes optic-nerve atrophy usually develops in the initial stage at a time when the ataxic symptoms are slight or absent, and the diagnosis of tabes is not yet readily made. It is, therefore, a fact of great value to us that we know two other symptoms which likewise usually make their appearance very early. One of these symptoms regards the pupil, which no longer reacts to light (Argyll-Robertson pupil, § 377), and is generally also greatly contracted

(spinal miosis, § 374); the other is the absence of the patellar reflex. Othe important signs are a positive Wassermann reaction in the blood an especially in the spinal fluid and lymphocytosis of the latter with a positiv globulin reaction. Atrophy of the optic nerve due to tabes or general paresi always affects both eyes, although not necessarily both at the same tim It advances slowly but surely until there is complete blindness, and henc has rightly earned the name of progressive atrophy. With the diminishin sight is associated progressive restriction of the visual field (usually unde the form of progressive concentric contraction or of sector-like defects rarely under that of a central scotoma). The field for colors is affecte before that for white objects, the field for red and green disappearing earl and later that for blue. By this fact atrophy is distinguished from non congestive glaucoma which sometimes shows much resemblance to it, bu which is not ordinarily associated with color blindness till late in its cours Atrophy of the optic nerve due to disseminated sclerosis is characterized b an irregular course—at times by transient improvement of sight—anc rarely leads to complete blindness. It is often unilateral. The field varies but a central scotoma is relatively frequent (cf. § 815).

2. A *break in the line of conduction.* This may be intra-cranial in site a tumor or other focal affection compressing the nerve itself or the chiasm within the skull. In this case the atrophy is propagated gradually from the site of the break down to the intra-ocular extremity of the nerve (descending atrophy). The break may also evidently be located near the periphery of the nerve—i. e., in the orbit where the nerve may be thrown into a state of atrophy by inflammations, injuries, or as a result of compression by tumors. Atrophy by a break in the line of conduction may give the same ophthalmoscopic picture as tabetic atrophy. But while in the latter the pallor of the optic nerve can be made out at the very same time that the deterioration in sight begins, in the former it occurs subsequent to the deterioration in sight, since it takes some time—several weeks, at least, and usually longer—for the degeneration of the nerve fibres to travel from the point where the break is situated down to the papilla.

Simple atrophy is found most frequently in middle life. In children it almost never occurs except in the rare *infantile* or *juvenile tabes*, in which it is an almost constant and sometimes almost the only symptom (Barkan). The atrophy of childhood is, as a rule, neuritic. Men are more frequently attacked by simple atrophy than women. It is found in 10 to 15 per cent of all cases of tabes; 4 to 7 per cent of all cases of general paresis; and, usually as a partial atrophy, in over 40 per cent of all cases of multiple sclerosis. In *old people* a low degree of non-inflammatory atrophy of the optic nerve sometimes occurs, caused by atheromatous disease of the internal carotid or of the ophthalmic artery. In this case the vessels by the pressure they cause induce a partial atrophy of the optic nerve, which for a certain part of their course they directly adjoin (Bernheimer, Sachs, Otto).

In the simple atrophy of tabes the *anatomical changes* at the outset consist of discrete foci of disease, which appear gray upon cross section and which lie in the trunk of the optic nerve. In this case we are dealing with the same gray degeneration that exists in the posterior columns of the spinal cord in tabes. The nerve fibres lose their white medullary substance and are transformed into extremely minute fibrillæ, and hence the entire tissue acquires a gray and translucent appearance. Between the remains of the

nerve fibres are found cells filled with granules of fat, and in the later stages secondary changes occur consisting of a moderate thickening and sclerosis of the connective-tissue septa and multiplication of the glia nuclei. Along with the degenerative changes above outlined, inflammatory exudates occur, and many hold that the inflammation may precede the degeneration, so that the expression simple or primary atrophy is a misnomer (Stargardt, Spiller, Schoenberg, Wechsler). In disseminated sclerosis, a proliferative inflammation of the connective tissue and of the glia, occurring under the form of insular foci, appears in the nerve at the very outset. Later, the proliferated tissue shrinks and the nerve fibres lose their medulla. The axis-cylinder, however, remains intact for a long time and for this reason the sight is not as a general thing completely destroyed.

The anatomical condition found in descending or ascending atrophy is similar to that occurring in gray degeneration of the optic nerve. The atrophy reaches its highest degree in those cases in which the eyeball has been completely destroyed, the optic nerve in this instance shrinking in the course of time to a thin strand consisting simply of connective tissue.

817. (*b*) *Inflammatory atrophy* of the nerve is the form which occurs as the final result of a neuritis or a retinitis (neuritic or retinitic atrophy). Inflammatory is distinct from simple atrophy in its ophthalmoscopic features as well as its origin, because in it the disc is traversed by connective tissue formed by an organization of the exudate. In *neuritic* atrophy the disc is at first of grayish-white color, and its margins are slightly hazy; the veins are very distended and tortuous. Afterward the disc becomes of a pure white or bluish-white but we do not see the lamina cribrosa exposed to view as in simple atrophy. The disc is now sharply defined, but is often smaller than normal and irregular, as though it had been shrunken; both arteries and veins are contracted and are frequently inclosed within white streaks. The chorioid immediately adjoining the disc often shows an irregular decolorization. In *retinitic* atrophy the disc looks clouded and of a dirty grayish-red. Its outlines are faint, and the vessels are greatly thinned and often have entirely disappeared (Fig. 357).

818. The *prognosis* of atrophy of the optic nerve is in general unfavorable. Cases of simple atrophy for the most part lead to complete blindness. Inflammatory atrophy affords a somewhat better prognosis, since the amount of sight which the neuritis or retinitis has left is usually permanently preserved. *Treatment* consists primarily in the management of the causal disease. For the lesion of the optic nerve itself, in syphilitic cases, salvarsan (especially by intraspinal injection and combined with mercury to prevent neuro-recidives), mercury alone, and potassium iodide are employed; and in these and also in non-syphilitic cases injections of strychnine, and the constant or high frequency current applied to the eye itself; unfortunately, however, generally with but slight success. A beginning descending atrophy due to increased intra-cranial pressure may be helped by decompression and one due to hypophysis disease may be arrested by an operation to remove the diseased pituitary gland (Cushing).

In atrophy due to tabes or paresis, not directly traceable to syphilis, it has been the general belief that mercury may hasten the atrophic process. By good observers this belief is now regarded as erroneous (cf §§ 811, 813).

IV. Toxic Amblyopia

819. Poisons may interfere with vision in various ways, by causing either paralysis of the eye muscles, of the sphincter pupillæ, and of accommodation, or by causing disease of the light-perceiving apparatus, i. e., the retina and optic nerve. Affections of the latter class are comprised under the name of toxic amblyopias. These are either acute or chronic.

Acute Poisoning.—In acute poisoning, more or less complete blindness sets in either immediately or within a few days after the poison has entered the system. The poisons most commonly acting in this way are:

1. *Quinine.* This in doses of three gm. and upwards, but sometimes of 1 gm. or less, causes sudden blindness with hardness of hearing or complete deafness. In most cases the blindness disappears again gradually but incompletely, amblyopia and a markedly contracted visual field remaining permanently. Even at the beginning we find with the ophthalmoscope excessive attenuation of the retinal vessels, with which is afterward associated a pallor of the disc.

Anatomical investigation in quinine poisoning produced experimentally in dogs shows during the very first days a destruction of the ganglion cells of the retina (Holden, Druault). These are primarily attacked by the poison, and as a result of the death of ganglion cells a degeneration of the nerve fibres in the optic nerve very soon sets in.

2. *Wood Alcohol.* This produces a pupillœdema or at any rate a neuritic atrophy with sudden, complete, and usually permanent blindness. In a few cases this has occured from inhalation of the vapor (as in shellac workers), but generally from drinking cheap liquor, Jamaica ginger, or other liquids which have been adulterated with deodorized wood alcohol ("Columbian spirits"). Saline purges, diuretics, instillation of dionin, and lumbar puncture are said to help, and salvarsan has been recommended.

3. Sudden blindness also occurs after poisoning by salicyl preparations including oil of wintergreen, filix mas, pomegranate-root bark, optochin, atoxyl, arsacetin, osmic acid, potassium chlorate, and acetanilide. Acute, complete or partial blindness is also caused by inhaling the fumes of exploded dynamite and of nitrobenzol (Stephenson).

820. Chronic Poisoning.—

1. *Poisons Causing Formation of a Central Scotoma.*—Substances very frequently giving rise to poisoning of this type are tobacco and alcohol, especially the former. Very frequently the two poisons act together, the person affected being at the same time a great smoker and a great drinker. The symptoms in the two cases are the same. The disturbance of vision sets in so gradually that the patients are for the most part unable to tell exactly when it begins. At first medium-sized print can still be read, afterward the reading of ordinary print becomes impossible. The reduction in the visual acuity is almost always the same in both eyes—a fact which distinguishes this from other intra-ocular affections, such as cataract, chorioiditis, atrophy of the optic nerve, etc., in which the two eyes are usually affected to a different degree.

The symptom of *nyctalopia* is particularly characteristic. The patient declares that he sees much better in the evening than in the daytime; indeed, in recent cases he often imagines that in the evening he still sees as well as he used to do, and that it is only in the day-time that he has a troublesome cloud that dazzles his sight. Objective examination shows that really in most cases no observable improvement takes place when the illumination is reduced; but the annoying sense of dazzling is done away with, so that the patient believes that he sees better. In some cases, however, a real improvement is produced in the sight upon diminishing the illumination. Tests of the light sense show that the perception of light difference is specially affected (Percival).

Many patients also declare that they cannot recognize *red* colors, particularly small objects, as well as formerly. Their acquaintances, they find, look ill because their cheeks appear of a waxen yellow. Innkeepers, who are particularly apt to be affected with tobacco amblyopia, complain that they can no longer distinguish between copper and nickel coins, since they no longer appreciate the difference in color.

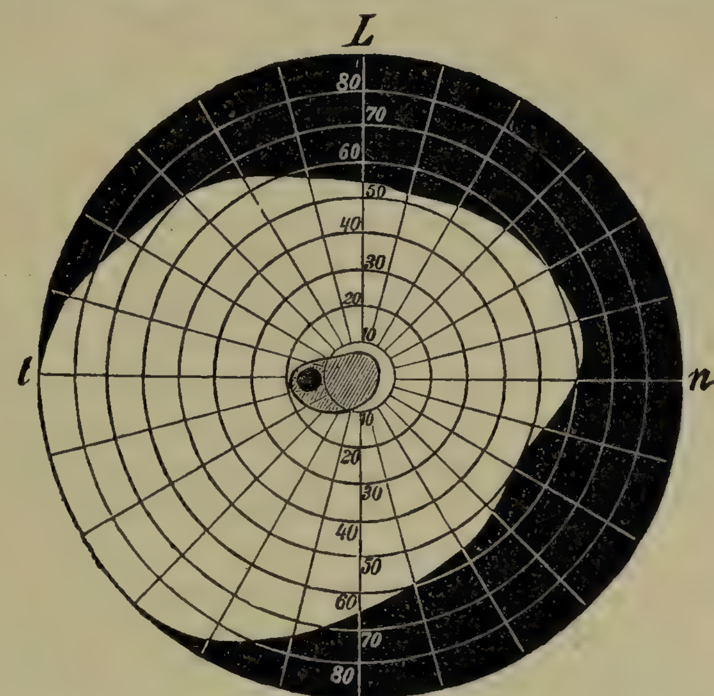


Fig. 368.—Field of Vision of the Left Eye of a Man Suffering from Tobacco Amblyopia.

The visual field when tested with a white object (shown by the portion left white in the figure) is normal. When, however, the examination is made with a red object, a central scotoma is found having an extent represented by the shaded area which forms an irregular oval. The small black circle comprised in this area represents Mariotte's blind spot.

Objective examination shows but *slight ophthalmoscopic changes.* In recent cases the papilla is usually somewhat hyperæmic; in the older cases, on the contrary, it has grown paler in its temporal half. But these changes are often so little pronounced that one may say that the result of examination is negative.

Examination of the *vision* shows a moderate diminution of the visual acuity, which has its cause in a central scotoma (Fig. 368). This scotoma forms a horizontal oval, extending from the macula lutea to the blind spot, and corresponding, therefore, to the maculo-papillary region of the retina (Figs. 40, 41). At first there is simply a color scotoma. No gap is found in the field of vision if it is tested by means of a white object; but a red or green mark undergoes a change of color in the region of the scotoma. It appears less highly colored than in the other portions of the field of vision, and later on appears perfectly colorless. Later still, the mark disappears altogether from view in this portion of the visual field; the scotoma has now become absolute (see § 271), and the vision has become reduced to the lowest point that it can reach in this disease. The outer limits of the visual field always remain normal, and complete blindness is therefore not to be apprehended, but direct vision is destroyed, and with it the ability to carry on any fine work. Owing to the chronic course of the disease, it takes a series of months for the sight to be reduced as low as this; and, moreover, this extreme reduction does not occur in every case.

The *cause* of tobacco amblyopia is the excessive use of tobacco, whether by smoking or chewing. The disease is hence found almost exclusively in the male sex, and in males not generally until middle life. It would thus appear as if the resistance to nicotine diminishes with age. The quantity of tobacco which is sufficient to bring on a tobacco amblyopia varies according to the susceptibility of the individual, in many cases a comparatively small amount sufficing for this purpose. The cheap varieties, which are usually richer in nicotine, and also moist tobacco, are more dangerous than the better dry qualities. The abuse of spirituous beverages, which, to be sure, is very usual with great smokers, favors the development of tobacco amblyopia; but amblyopia also occurs in smokers who abstain altogether from alcoholic drinks.

Treatment consists, above all, in abstinence from tobacco, and in light cases this alone is sufficient to effect a cure. To accelerate the cure we employ iodide of potassium

internally or hypodermic injections of strychnine. Stress has also been laid on the importance of preventing intestinal autotoxis (by suitable diet, purgation, free water-drinking, etc.). Furthermore, as stated in § 821, the urine ought to be examined in every case, and the treatment modified accordingly. In addition, we prescribe a suitable regimen for the eyes. Recent cases in which middle-sized print can still be read, and in which the scotoma has not yet become absolute, afford a good prognosis, since a perfect cure is usually obtained although one or two months are required for it. In older cases, however, in which even quite large print can no longer be read and the scotoma is absolute, a complete cure is for the most part impossible.

Alcohol amblyopia also does not usually begin before middle life, and occurs as a rule only in spirit drinkers. It is quite frequently combined with alcoholic neuritis of the peripheral nerves. The treatment is the same as that of tobacco amblyopia.

Samelsohn showed that the *anatomical changes* in nicotine and alcohol amblyopia are limited to the papillo-macular bundle, whose position and course within the optic nerve he was thus able to determine (§ 48). In the course of this bundle the nerve fibres ultimately disappear and nothing but glia tissue is left, while the connective-tissue septa lying between the nerve fibres are thickened (Fig. 41). (Some doubt has recently been cast on the validity of Samelsohn's deductions as to the size of the papillomacular bundle, it being thought to be really smaller than shown in the figure). The changes found have been commonly regarded as an interstitial neuritis, as a primary affection of the optic-nerve fibres with secondary interstitial changes, or with less probability as a lesion of the ganglion cells in the retina with ascending atrophy.

The same complex of symptoms as in nicotine and alcohol amblyopia occurs also in chronic poisoning due to various *other substances* which either are taken into the body for therapeutic purposes, like stramonium, chloral, iodoform, iodides, thiuret, thyreoidin, or which find their application in industries, like carbon bisulphide (in vulcanizing rubber) and aniline. Iodoform may cause complete blindness.

2. *Lead Poisoning.*—This usually causes papillitis—very rarely indeed, an amblyopia with central scotoma. The visual disturbance, which develops gradually, may go on to complete blindness.

821. Autotoxæmic Amblyopia.—Disturbances of vision due to neuritides which are usually retrobulbar and run sometimes an acute, sometimes a chronic course, are also found in cases in which owing to anomalies of metabolism the opportunity is afforded for the production of toxic substances (*auto-intoxication*). The most frequent occurring example of such a disturbance of vision is that found in *diabetes.*

In this case we sometimes observe an amblyopia of the type of a tobacco amblyopia. This may be simply the result of the diabetes itself, but mostly it affects patients who smoke, so that the influence of nicotine cannot be surely excluded. It seems that the resistance of the optic nerve toward nicotine is diminished by the disturbance of metabolism due to the diabetes, for which reason a diabetic patient ought to be warned not to smoke much; and for a like reason the urine ought to be examined for sugar in every case of amblyopia with central scotoma, even when such amblyopia apparently comes from smoking or drinking.

Probably also referable to auto-intoxication are the cases of papillitis and retrobulbar neuritis which are found in anomalies of menstruation, e. g., during pregnancy and lactation, and in gout.

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822. Injuries of the Optic Nerve.—The optic nerve may be injured within the orbit *directly* by penetrating foreign bodies, stab wounds, shot wounds, etc. Because of this break in the line of conduction, blindness—partial or complete according to the severity of the lesion—is present immediately after the injury. With this there are, at first, no demonstrable ophthalmoscopic changes. It is not until after weeks have elapsed, when the descending atrophy has traveled from the site of injury to the disc, that the latter becomes paler and presents the picture of simple atrophy. When, however, the optic nerve is injured so far forward that the central vessels are also divided, a picture

analogous to that of embolism of the central artery develops at once. The arteries of the disc and retina are bloodless, and the retina soon becomes clouded—a sign of its death. By some severe injuries the optic is torn completely from the eyeball (*evulsion of the optic nerve*). The ophthalmoscope then shows, beside intra-ocular hæmorrhages, a deep, well-like hole at the site formerly occupied by the disc (Blake). In a case of partial evulsion the optic disc was still present but imbedded in the bottom of an ectatic area of sclera (Simpson).

Indirect injuries of the optic nerve occur not infrequently in consequence of injuries of the skull by the impact of a blunt object (a blow or fall upon the head, etc.). In such cases there is partial or complete blindness associated with the symptoms of a severe injury of the skull (signs of concussion of the brain or of fracture of the base of the skull). Such blindness may be unilateral or bilateral. In these cases we are dealing with indirect fractures of the walls, particularly of the upper wall, of the orbit, which fractures are continued into the optic canal, so that the optic nerve in the latter is crushed or lacerated. Several weeks or months afterward there develops in the disc the picture of simple atrophy. These cases of blindness are incurable. Some believe that the injury to the nerve in this case is due not to fracture of the adjoining bone but to subdural hæmorrhage compressing the nerve. Such a *hœmatoma of the optic-nerve sheath* may be caused not only by injury, but also by the descent of blood from the brain (e. g. in cerebral hæmorrhage or in pachymeningitis), by the rupture of an aneurysm, and by circulatory disturbances, such as occur in diabetes, nephritis, and scurvy.

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823. Tuberculosis of the Optic Nerve.—In rare cases a conglobate tubercle occupies the disc, transforming it into a white, shining, smooth or nodular, tumor-like mass which is considerably larger than the normal disc and projects far into the vitreous. Small white foci may be present in the adjoining retina. The sight is comparatively good and improves under tuberculin treatment, which also causes subsidence of the swelling until nothing is left of it but a thin connective-tissue layer (Salzmann).

824. Tumors of the Optic Nerve.—Secondary tumors of the optic nerve develop from the growth of intra-ocular tumors, such as sarcoma of the chorioid and glioma of the retina, backward along the nerve. Primary tumors of the nerve are rare. Some, having the structure of endotheliomata spring from the dural sheath. Others (fibromata, sarcomata, myxomata, gliomata, etc.), which spring from the subarchnoid trabeculæ, pial sheath, or septa or glial tissue of the nerve, have a common character, being all manifestations of neurofibromatosis (Recklinghausen's disease). Other symptoms of neurofibromatosis (§ 836) are often found. Primary tumors of the optic nerve begin generally in youth, and grow very slowly. They cause an exophthalmus, distinguished from that occurring with other orbital tumors by the fact that the lateral displacement is either entirely absent or is at all events insignificant. The mobility of the eye remains good for a comparatively long time; on the other hand—and this is characteristic—blindness sets in very early. With the ophthalmoscope we find at first neuritis with the venous engorgement particularly marked; afterward atrophy. In one case reported by Ellett the fundus appeared normal, although there was complete blindness. Treatment consists in extirpating the tumor; in doing which we may, under certain circumstances. leave the eye in place (see § 998). Recurrences after operation are comparatively rare,

Tuberculosis of the nerve also appears under the guise of a tumor (§ 823).

Hyaline bodies (so-called colloids) in the form of pearly or yellowish, rounded elevations, usually massed in mulberry-like aggregations, are sometimes found on the papilla and the adjacent retina. They were formerly confounded with druses (see §742), from which, however, they differ in origin and structure (De Schweinitz, Leber). *Amyloid bodies* are also found in the same situation.

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825. Congenital Anomalies of the Optic Nerve.—They include coloboma (§ 769), aplasia (§ 806), pseudoneuritis (§ 406), medullated nerve fibres (not strictly congenital see § 806), and the presence of shreds or larger masses of connective tissue enveloping the vessels and projecting from the disc, like the masses in retinitis proliferans.

CHAPTER XXIX

GLAUCOMA

General Considerations

826. Nature.—The essence of glaucoma[1] lies in the *increase in the intraocular pressure,* from which all the other essential symptoms of glaucoma can be deduced (Von Graefe). In one series of cases the increase in pressure sets in without our being able to discover any reason for it in an antecedent disease of the eye (*primary glaucoma*). In other cases, on the contrary, the increase in pressure is the result of some other disease of the eye (*secondary glaucoma*). Primary glaucoma, accordingly, has increase in tension as its first and most important symptom, from which all the rest of its phenomena arise—it is glaucoma proper, the glaucoma par excellence. In secondary glaucoma, on the contrary, the increase in tension is only a consequence of other pathological conditions—is an accessory, as it were. The clinical picture of secondary glaucoma, therefore, is exceedingly polymorphous, varying according to the different affections which form the basis of it. While genuine or primary glaucoma always affects both eyes, although not always at the same time, secondary glaucoma remains confined to the eye which, by being diseased, has caused the increase in tension. The diagnosis of glaucoma is based mainly on the demonstration of its cardinal symptom, increase of tension, by palpation or by the tonometer (§ 394).

Glaucoma has been known from antiquity. Of course, it is only the congestive variety that has been thus known, since the non-congestive variety can be diagnosticated only by the ophthalmoscope. Hence this second variety and the other kinds of blindness, that are produced by diseases of the deeply situated membranes of the eye, and that have no external manifestations, were lumped together under the common name of amaurosis. Congestive glaucoma was usually regarded as having a connection with gout, and was hence called ophthalmia arthritica. The first to recognize the increase in tension as the most important symptom of glaucoma were Mackenzie, and particularly Von Graefe. Heinrich Müller, a man deserving the greatest praise in all that relates to the pathological anatomy of the eye, was the first to demonstrate anatomically the pressure excavation of the optic nerve (1856); soon afterward it was accurately diagnosticated in the ophthalmoscopic picture by Weber and Förster. Mackenzie, starting from the fact of the increase of tension, had already attempted to effect the cure of glaucoma by repeated paracentesis of the cornea, without, however, obtaining any lasting result. Such a result was first obtained by Von Graefe, who, in the year 1856, employed iridectomy for the first time in glaucoma, after having found it efficacious in various other diseases of the eye. This was one of the most pregnant discoveries in ophthalmology, and one which will for all time redound to the glory of Von Graefe. We have only to remember that formerly every case of glaucoma inevitably led to blindness, and that now, thanks to iridectomy, the majority of glaucomatous patients can be cured. How many thousands there are who formerly would have been forced to sink year by year irretrievably into the night of blindness, but who now are saved for vision through Von Graefe's discovery!

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[1] From γλαυκός sea-green, on account of the greenish reflex from the pupils.

827. Consequences.—The consequences of increase of tension, inevitably occurring if it lasts a long time, are excavation of the optic nerve, and reduction, with ultimate annihilation, of the sight.

Excavation of the optic nerve is dependent upon the recession of the lamina cribrosa. The lamina cribrosa is that part of the fibrous tunic of the eye (corneo-sclera) which has the least tenacity, and hence gives way

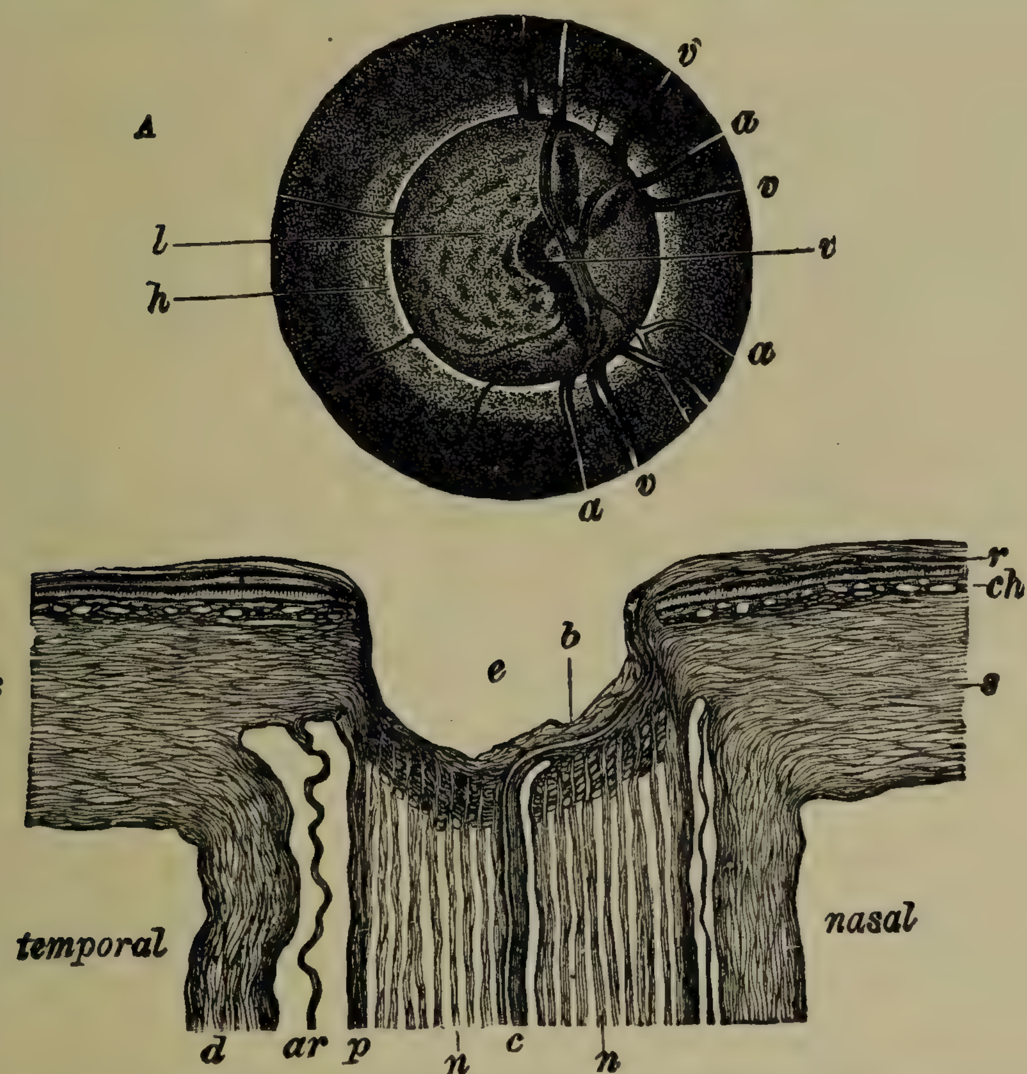

FIG. 369.—GLAUCOMATOUS EXCAVATION OF THE OPTIC NERVE. Magnified 14 × 1. Cf. the normal optic nerve in Figs. 36, 192, 193.

A. OPHTHALMOSCOPIC PICTURE OF THE DISC.—The disc is bounded by a sharp, overhanging edge, at which the arteries, *a*, and the veins, *v*, of the retina appear to terminate in recurved ends. This is due to the fact that their continuation on the floor of the excavation is displaced somewhat laterally as compared with the portion situated in the retina, because those portions of the vessels that descend on the lateral wall of the excavation do not run in a precisely sagittal direction, but somewhat obliquely. The vessels within the excavation are seen but indistinctly. In the outer half of the excavation are seen the gray dots of the lamina cribrosa, *l*. The zone, *h*, of the fundus, adjoining the disc, is decolorized (halo glaucomatosus).

B. LONGITUDINAL SECTION THROUGH THE HEAD OF THE OPTIC NERVE.—This shows a deep excavation, *e*, on the floor of which only a few remains of the nerve fibres, *b*, are visible. The central vessels, *c*, ascend upon the retina, *r*, at the nasal margin of the excavation; the innermost layer (fibre layer) of the retina is considerably diminished in size through atrophy. *ch*, chorioid; *s*, sclera. The volume of the trunk of the optic nerve has been considerably reduced through the atrophy of the bundles of nerve fibres, *n*. As a result of this, the interspaces between the sheaths of the optic nerve (the pial sheath, *p*, the arachnoid sheath, *ar*, and the dural sheath, *d*) are dilated, especially on the temporal side.

first to increased ocular pressure, which it does by bulging backward. But at the same time the optic-nerve fibres set in the foramina of the lamina cribrosa, also recede, so that the surface of the optic nerve itself sinks back (*e*, Fig. 369 B). Upon ophthalmoscopic examination the disc appears depressed below the level of the adjacent fundus—slightly at first, afterward a good deal—so that the margins of the disc dip down abruptly, or are even overhanging. This condition is recognized chiefly by the bending or actual interruption of the blood-vessels at the spot where they pass from the

retina over the edge of the disc and dip down into its depth (Fig. 369 A). Th nerve fibres, too, like the blood-vessels, undergo flexion or interruption a the edge of the disc. This interruption, with the high pressure to which th nerve fibres are exposed within the eye, makes them atrophy. Accordingly in the later stages, the disc is not only depressed, but bleached and bluish-white, because the nerve fibres are destroyed and the clear white lamina cribosa is exposed.

With reference to *excavation of the optic nerve,* three varieties are distingushed—the physiological, the atrophic, and the glaucomatous. The *physiological excavation* (Fig. 370 A) originates from the fact that the bundles of fibres of the optic nerve, when separating from each other so as to curve into the retina, do so, not in the plane of the retina, but behind it; the lamina cribrosa, however, is in its normal situation. The physiological excavation is always partial—i.e., even if it is very large it never takes up

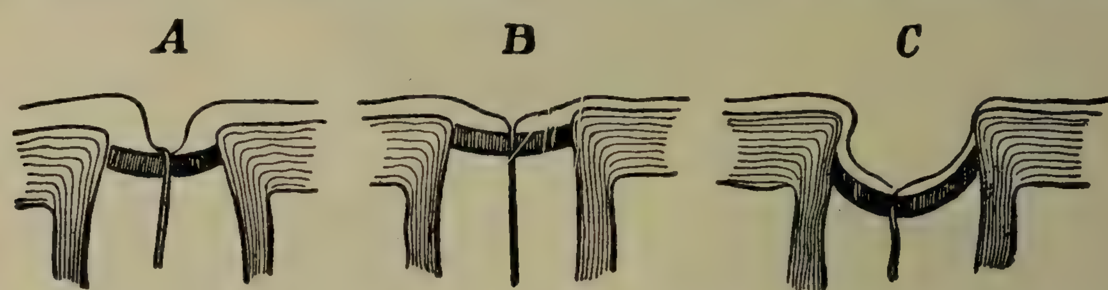

Fig. 370.—The Three Kinds of Excavation of the Optic Nerve. Schematic.
A, Physiological Excavation.—Funnel-shaped, partial, with normal lamina cribrosa.
B, Atrophic Excavation.—Bowl-shaped, total, with normal lamina cribrosa.
C, Glaucomatous Excavation.—Ampulliform, total, with the lamina cribrosa bulged out posteriorly.

the entire disc, because a certain space close to the edge of the disc must always be occupied by the nerve fibres which are passing over into the retina (§ 407; Fig. 36, *G*, and Figs. 190, 191, and 332.) The *atrophic excavation* (Fig. 370 B) is caused by the disappearance of the nerve fibres that form the head of the optic nerve and lie in front of the lamina cribrosa—the lamina cribrosa itself remaining in place. The atrophic excavation is total—i.e., extends over the entire disc, but always remains shallow for at most it can only attain a depth equal to that at which the lamina cribrosa lies behind the inner surface of the retina. In the atrophic excavation, the disc is at the same time bleached white on account of the disappearance of the nerve fibres. The physiological and the atrophic excavations have this in common, that the lamina cribrosa remains undisplaced; as the lamina cribrosa forms the floor of the excavation the depth of the latter is usually confined within narrow limits. The *glaucomatous excavation* (Fig. 370 C) is distinguished from the preceding kinds, above all, by the fact that it originates in a recession of the lamina cribrosa; it can hence attain a much more considerable depth than they. The glaucomatous excavation comprises the entire disc which, in the beginning, still shows the red coloration of health. Later on, the nerve bundles are destroyed by atrophy, so that the disc becomes white, and displays exposed upon its floor the lamina cribrosa. With this is associated a still further increase in the excavation, the depth of which is increased by a space equal to the thickness of the nerve head which has been destroyed.

The ophthalmoscopic signs distinguishing the three kinds of excavation are, therefore, as follows: A partial excavation is physiological, a total one is pathological, and either atrophic or glaucomatous. The atrophic excavation is shallow, and the papilla, at the same time, very white. The glaucomatous excavation may be shallow or deep according as it has existed for a longer or shorter time. In a shallow glaucomatous excavation the papilla usually still has a good color—a feature which distinguishes it from the atrophic excavation. If the excavation is deep and total, it can only be a glaucomatous one, whatever color the papilla may have. In practice, the distinction between the individual forms of excavation is sometimes very difficult to make, partic-

ularly so, as the physiological excavation is sometimes very deep as well as large, and is often overhanging. When a deep partial excavation of this sort occurs in a case of glaucoma, it seems difficult to believe that the latter is not, in part at least, responsible for its development. Again, in some cases of glaucoma the pallor of the disc occurs early, even before there is any marked excavation (Elliot). These cases would be readily confounded with atrophy.

The *ophthalmoscopic picture* of a glaucomatous excavation shows the disc to be paler, and, in advanced cases, bluish or greenish white. A shadow is seen running along the margin, while the centre of the excavation is the part lightest in hue. On the floor of the excavation may be recognized the gray dot-marks of the lamina cribrosa (Fig. 369 A, *l*). The vessels do not emerge at the centre, but, for the most part, close to the inner margin of the disc. Where they pass over the edge of the disc to go to the retina they show a bending, or, in deep excavations, an interruption, of their course, If the edge of the excavation is overhanging, the ascending portion of the vessel may be completely concealed behind it, so that the blood-vessels arising from the vascular entrance seem to disappear at the edge of the disc to emerge again in the retina at some other spot. It is only by the indirect method that the course of the vessel can be seen distinctly in its whole extent at once; with the direct method, the vessels on the disc and those in the retina are never seen clearly at the same time, since they lie at different depths and hence have a different refraction. From the difference in refraction between the margin and the floor of the excavation, the depth of the latter can be estimated (see § 411), and by repeated measurements of this sort we can determine whether, as time goes on, the depth of the excavation is increasing or diminishing. With the indirect method the difference of level manifests itself by parallactic displacement (§ 411). The calibre of the arteries is contracted, while the veins are distended and tortuous—in fact, sometimes there is a whole convoluted mass of vascular loops lying on the floor of the excavation. These changes of vascular calibre are easily accounted for by the effect which the increase of pressure exerts upon the vessels at the vascular entrance, permitting less blood to enter the arteries of the retina and, on the other hand, obstructing the outflow of blood from the veins. The former therefore, are filled too little, the latter are filled too much. We very often observe a pulsation in the veins, and not infrequently, also, a pulsation in the arteries within the disc. (For the explanation of this, see § 408.) When a glaucomatous excavation has lasted for a long time, the disc is usually found to be surrounded by a white or yellowish areola, which is the expression of an atrophy of the chorioid about the disc (halo glaucomatosus, Fig. 369 A, *h*). The rest of the fundus in the later stages often shows the network of chorioidal vessels with great distinctness (Fig. 333) because of the diminished pigmentation of the pigment epithelium.

Schnabel ascribes the cupping of the disc in glaucoma to the formation of lacunæ in the optic nerve (*cavernous atrophy*). In this view the recession of the lamina cribrosa would be due to traction from behind rather than to pressure in front. Such atrophic lacunæ do, in fact, exist in glaucoma, but are also found in high myopia and their situation is not such as to indicate that they exert traction on the lamina cribrosa. That, moreover, the cupping is due to increase of tension is shown by the fact that it may disappear after the tension has been restored to normal by operation or miotics (Fuchs).

828. Impairment of Vision.—The atrophy of the optic-nerve fibres is also the most important cause of the decrease of visual power which accompanies the elevation of tension. This finds expression, first, in the gradual diminution of acuity of central vision, second, in the limitation of the field, which begins, in the majority of cases, on the nasal side, as the temporal side of the retina becomes insensitive first. The light sense also is much affected. Even when by instrumental tests it is not reduced for central vision, the peripheral light sense is evidently lessened, since the patients have well-marked hemeralopia (Elliot). Finally, as the disease progresses, complete blindness supervenes.

The condition of the *sight* is not always in direct proportion to the depth of th excavation—in fact, it is not the recession of the lamina cribrosa as such that affects th vision, but the atrophy of the optic nerve, which, though produced by it, does no always by any means keep pace with the formation of the excavation. Thus we some times see cases with deep excavation and yet with normal visual acuity and a large fiel of vision. On the other hand, by a very considerable increase in pressure—as in glau coma fulminans—the sight may be completely extinguished within a few hours by paraly sis of the optic-nerve fibres without there being any excavation of the optic nerve, becaus the time is too short for it to be formed. Even in chronic glaucoma it is sometimes th case that the nerve atrophies and complete blindness actually ensues as the result of th increase of pressure before—sometimes even years before—an obvious glaucomatou excavation is formed. Accordingly, in form ing a judgment as to the acuity of vision, w must be guided rather by the color of the disc and the calibre of the retinal arteries than b the depth of the excavation, since atroph of the nerve fibres makes itself known mainl through the pallor of the disc and the narrow ing of the retinal vessels.

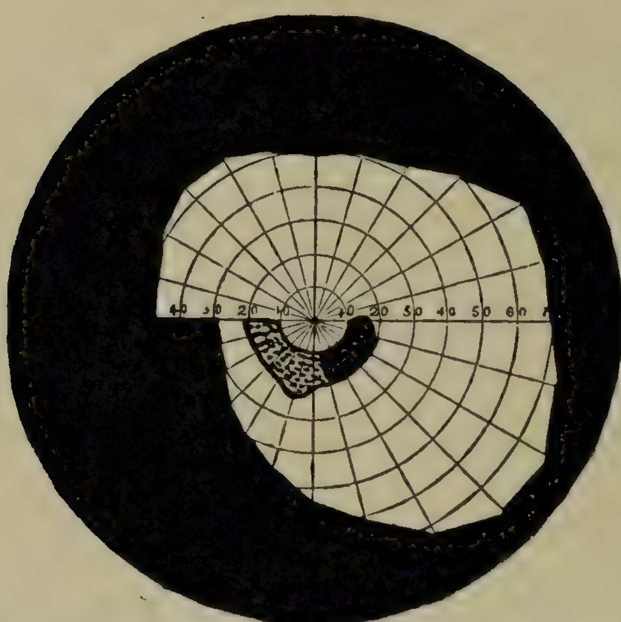

Fig. 371.—Bjerrum's Sign and Rönne's Step (after Elliot)

The chart shows the field of an eye affected with chronic glaucoma. There is a scotoma which extends down and in from the blind spot and curves round the point of fixation (Bjerrum's sign). This scotoma is partly absolute (black area) partly relative (dotted portion). At its nasal tip it ends in a horizontal line lying exactly in the horizontal meridian of the field, indicating that the corresponding supero-temperal bundle is implicated up to its very end in the horizontal meridian of the retina. The field also shows a peripheral contraction, more marked on the nasal side; also, in its intermediate zone, a defect, which in the lower field extends in to the meridian of 30° and above is limited sharply by the horizontal meridian. The defect in the upper field reaches in only to 48°, so that the two defects in the field where they meet, form a right-angled step (Rönne's step), the upper border of which is coincident with the horizontal meridian.

The restriction of the *visual field* expresses itself regularly in two ways. First, there is a *peripheral contraction* which is almost always more marked on the nasal side; second, there are defects in the cæcal zone connected with the blind spot. Of the *cæcal defects* one of the earliest seen is a tapering wing-like elongation of the blind spot upward or downward (Seidl's sign). This wing which is usually demonstrable only with small (3:1000) test objects, curves toward the point of fixation. By extension of this wing, Bjerrum's sign is produced. This consists of a sickle-shaped scotoma, continuous with the blind spot and circling above or below round the point of fixation (Fig. 371). This scotoma is usually demonstrable only with fine objects (2:2000, etc.), and may be relative even with these. If there are two such extensions of the blind spot, one above and the other below, an annular scotoma may be formed.

Both Seidl's and Bjerrum's signs are due to an interference with the supero-temporal and infero-temporal bundles (§ 47; cf. Fig. 40) in the cæcal zone. A similar interference with the more peripheral portions of these bundles occurs as the disease advances, causing defects which are *located in the intermediate zone* of the visual field (§ 47), and which merge with the peripheral defects. Since the temporal bundles stop short at the horizontal meridian of the retina, defects due to lesion of them (Bjerrum's scotoma, defects of the intermediate zone) are arrested at the horizontal meridian of the nasal field, their boundary there forming a sharp horizontal line. Two opposite defects of this sort, one in the upper, the other in the lower nasal field will therefore, if unequal in extent, form a sharp right angle or "step" in the visual field, one side of the step lying in the horizontal meridian (Fig. 371). This right angle is called Rönne's step. It occurs in the intermediate zone defects, and can be made out best with large (3:300) test-objects. It is found not only in glaucoma, but also in affections of the optic-nerve trunk, of the chiasm, and of the paths connecting the latter with the cortex (Rönne). It evidently indicates an unequal involvement of the supero-temporal and infero-temporal

bundles. When either of these is involved very much more than the other so that Rönne's step is large, the defect occupies mainly one *quadrant* (upper nasal or lower nasal) of the field.

It is to be noted that in typical cases both the defective and the retained portions of the cæcal zone are continuous with the blind spot. Much less common at the outset are isolated *paracentral*, or even *central scotomata*.

By the combination of the various defects just described, a continuous contraction of the field occurs, taking many forms. Ultimately a small central area of clear vision is left so that the patient has but *tubular sight* and can not find his way about. Then a central scotoma appears, leaving only a small *eccentric islet* of vision. Finally this also is lost.

I. Primary Glaucoma

829. Varieties.—Primary glaucoma, also called simply glaucoma, sets in with varying symptoms. If the pressure rises suddenly to a considerable height, inflammatory symptoms are excited; on the contrary, these symptoms are wanting when the increase in tension develops gradually and keeps within narrow limits. Accordingly, a congestive and a non-congestive form of glaucoma are distinguished.

Primary glaucoma is a common disease, constituting about one per cent of all cases of eye disease. Its accurate recognition is of the greatest importance for the general practitioner, the more so because here prompt and proper therapeutic interference can save everything, but a false diagnosis and improper treatment may destroy everything. Unfortunately, we still constantly get under observation many cases of glaucoma which have been incorrectly diagnosticated by the general practitioner, and which come to the ophthalmologist only when help is no longer possible. Cases of congestive glaucoma are often confounded with iritis, and are accordingly treated with atropine, which has a particularly deleterious action in glaucoma. Cases of glaucoma simplex which present no external symptoms of inflammation are not infrequently regarded as commencing cataract, and the patients are put off in expectation of the cataract's becoming ripe, until it is too late for treatment.

A. Congestive Glaucoma (Glaucoma Inflammatorium).

830. Congestive glaucoma runs a typical course, especially in the acute form (acute congestive glaucoma). In this the following stages are distinguished:

(1) **Stage of Prodromes.**—The prodromal stage, which in most cases precedes the inflammatory attack, is characterized chiefly by attacks of obscuration of vision. The patient declares that during these attacks there is a cloud before his eyes which partially hides objects. If there is a light in the room, he sees a rainbow-colored ring about it. During the attack there is frequently a feeling of tension in the eye, or a dull frontal headache. If the physician examines the eye during such an attack, he finds the cornea a little dull and diffusely clouded, like glass that has been breathed upon. The cloudiness is greatest at the centre, smallest at the periphery, and, on account of its uniform distribution, causes considerable disturbance of vision. It also produces the appearance of the rainbow-colored halo or ring about a light, acting, in this regard like a fog which makes us see a similar ring about the flame of a street lamp. Such a halo occurs in other conditions, but is particularly significant of glaucoma. The anterior chamber is somewhat shallower through advancement of the iris; the pupil

is more dilated than usual and reacts sluggishly; the tension of the eye i distinctly increased. Frequently, too, slight ciliary injection is present.

Such an attack ordinarily lasts several hours, after which the ey returns completely to the normal condition once more, both as regard appearance and function. The attacks at first make their appearance a quite long intervals (of months or weeks); later, they become constantl more frequent. Often specific causes for their existence can be demon-strated, such as working by a dim light, hearty meals, late hours, emotiona excitement, etc. In many cases they return, even without cause, period-ically, sometimes even every day, so that the patient, for example, declare that he always sees through a cloud in the morning, and commencing with the afternoon begins to see clearly, or vice versa. If the attacks come on in the evening, they always cease when the patient falls asleep; even in the daytime an attack may be cut short by his going to sleep.

In the intervals between the prodromal attacks the sight of the eye is normal; but the patient complains that, to see near by, he has to employ stronger and stronger glasses—rapid increase of presbyopia through dim-inution of the power of accommodation; often also a hyperopia previously latent now becomes manifest (Elliot).

The prodromal stage sometimes lasts only a few weeks, sometimes is protracted over months or even years. In the latter case, the eye gradually undergoes permanent changes, so that it is no longer normal even in the intervals between the attacks. The eye acquires externally the glaucoma-tous aspect, and an excavation usually total, and at first extremely shallow, but gradually deepening, forms, in consequence of the oft-repeated increase of pressure. Consequently the sight itself is no longer perfect even during the time in which no attacks occur. In such a case we can no longer speak of the disease being in the prodromal stage, but must regard it as being now a chronic congestive glaucoma into which the prodromal stage has been imperceptibly transformed.

Although congestive glaucoma is so well characterized a disease, it is still very often diagnosticated too late. In the *prodromal stage* the diagnosis is obscured because the physician usually sees the patient in the interval between the attacks, when the eye looks normal both exteriorly and with the ophthalmoscope. The tension also may be normal, but repeated tests should be taken to see if at some time it does not rise above the normal limit. The physician is, however, mainly dependent for his diagnosis upon the description that the patient gives of his attacks. These are characterized by disturbance of sight (hazy vision, the appearance of colored rings about a light) and by headache (§108). On the other hand, we cannot with certainty make the diagnosis of glaucoma even when the patient makes the direct statement that he has hazy vision and sees colored rings. For, these two sorts of disturbance of sight occur as a result of any diffuse cloudiness of the media and are hence not confined to glaucoma. For instance, they occur in chronic conjunctivitis when some mucus lies on the cornea. But in this case the phen-omenon vanishes at once when the mucus is wiped off by rubbing the eye. Many persons, again, under appropriate conditions (e.g., if a flame is placed in front of a dark back-ground in a darkened room) always see a colored areola. In this case we have to do with faint diffuse opacities, chiefly in the lens, which are commonly too minute to be demon-strated objectively. These cases also are easily distinguishable from glaucoma, for, on the one hand, the phenomenon is far less pronounced than it is in glaucoma, and, on the other hand, does not occur in periodical attacks, but can be elicited at any moment.

Accordingly, in cases in which no changes in the eye are objectively discoverable we cannot make the diagnosis of glaucoma with certainty simply from the description that the patient gives of his attacks—not with enough certainty, for instance, to make us willing to suggest an iridectomy on the strength of this description alone. We must require that we ourselves see the patient during a prodromal attack, in order to demonstrate the visible objective changes that this produces. We hence charge the patient to present himself without delay as soon as he is again attacked with disturbance of sight or with headache. If he can not do this, we may use *pilocarpine* (or eserine) for making the diagnosis more certain, the patient being told to drop this in at once on the occasion of the next attack. If the morbid symptoms are completely relieved by this means in from ten to fifteen minutes, it is an argument in favor of glaucoma, for miotics have no effect on non-glaucomatous forms of cloudiness of the media nor usually on non-glaucomatous headaches. (Miotics may occasionally relieve a headache, especially one of migrainous type, due to other causes than glaucoma.)

Another way of determining the presence of glaucoma in a suspected case is to instil a *mydriatic* (Jackson). One of comparatively transient action (euphthalmine or ½-per-cent homatropine) should be used, the tension being first taken with the tonometer. If the mydriatic determines an increase of tension, and still more if it determines a glaucomatous attack, the diagnosis is assured. In either case eserine should be instilled without delay, and the instillation repeated until the pupil is contracted—the patient being kept under the physician's observation continuously until this occurs. If this precaution is observed there is little risk in the procedure, which is the more justifiable since an eye that would develop a glaucomatous attack under these conditions would quite surely develop an attack spontaneously later, and perhaps, too, when the disease had made greater inroads on the sight and the field of vision.

831. (2) **Glaucoma Evolutum.**—This is ushered in by an attack of *acute glaucoma*. This sets in suddenly, after the prodromal stage has lasted a longer or shorter time. The cause of an acute attack—in case such a cause is discoverable at all—is like those which determine the prodromal attacks. Chief among them are to be mentioned states of congestion of the venous system, especially those due to enfeeblement of the heart's action; also mental emotions, particularly those of a depressing character; and, lastly, dilatation of the pupils. For the last-named reason a drop of atropine in an eye which is predisposed to glaucoma may excite an attack. For the same reason bandaging the eyes, or long stay in the dark may also induce it.

The acute attack manifests itself by violent pain radiating from the eye along the first and second branches of the trigeminus. The patient complains of pains in the head, the ears, and the teeth, which may reach an intolerable pitch. They deprive him of appetite and sleep; not infrequently vomiting and fever likewise set in. Simultaneously with the appearance of the pain the visual power falls rapidly away, so that only large objects—such as, for instance, the hand moved to and fro before the eye—can be recognized. The field of vision is considerably narrowed, and mostly on the nasal side. Objective examination shows the appearances of a violent external inflammation—œdema of the lids, and œdema or even chemosis of the conjunctiva, which is greatly congested. The injection, in accordance with its pre-eminently venous character, has a dusky-red color. The cornea is punctately dotted, has a pronounced smoky cloudiness, and is almost or quite insensitive to the touch. The anterior chamber is shallower, the iris is discolored and narrowed. Consequently, the pupil is dilated; very often, too, it is oval and eccentrically situated, the narrowing of the iris being then

particularly great in special spots—most frequently above. The reaction of the iris is abolished. From the pupil we get a grayish-green reflex[2]. Ophthalmoscopic examination is impossible, on account of the marked cloudiness of the cornea. The tension of the eye is considerably elevated.

It will be seen that the symptoms of the acute attack are the same as those of the prodromal attack, except that they are much more marked and are accompanied by congestive symptoms (injection, œdema of the lids and conjunctiva, and pain). The prodromal attacks may therefore be regarded in the light of abortive attacks of glaucoma, which retrocede before they have developed to their full height. But at length on onset of this sort takes place, that rises to the height of an acute attack, and after this a perfect return to the normal is no longer possible. The tension now remains permanently elevated, and the eye retains the glaucomatous aspect.

The *course* of the attack of congestive glaucoma is that after some days or some weeks—according to the severity of the attack—improvement, or even an apparent cure, sets in. After some days the pain diminishes in violence and afterward disappears altogether. The eye becomes free from discoloration, the cornea clears up, and the sight becomes better again. If the sight was still normal before the attack, it may improve to such an extent that the patient can still read and write; but the more the sight has been injured before the attack by a prodromal stage of long duration, the less is its improvement after the decline of the attack. We may say in general that after an attack has passed off, the vision never again reaches the same height that it had before the attack occurred. The attack, furthermore, leaves behind it objective changes in the eye, that give at the first glance proof of the sort of disease that is present. The over-distention of the anterior ciliary veins remains; the anterior chamber is shallower, and the iris is narrower, is turned to a slate-gray color, and reacts sluggishly or not at all; the tension is permanently increased. We then say that the eye exhibits the glaucomatous aspect (habitus glaucomatosus). Ophthalmoscopic examination, which becomes feasible again after the cornea has cleared up, shows at the optic-nerve entrance the signs of hyperæmia which really is only one of the evidences of the general hyperæmia that was present during the congestive attack. The excavation of the optic nerve is not present directly after the attack, because for its formation quite a long period of increased tension is requisite; it hence does not develop until later on. It is only in those cases in which there has been a long preceding stage of prodromes that the excavation is present during the attack.

The nausea may persist for several weeks after an attack, and its recurrence may be the premonitory sign of a new attack (Morax).

After the subsidence of the attack the eye remains quiescent for quite a long time, and the patient entertains the hope of a permanent cure. Then a new attack sets in. This, as far as inflammatory symptoms and pain are

[2] This reflex is by no means distinctive of glaucoma. It is always found when the pupil is dilated and at the same time the media are not completely transparent, as happens in most old people. Hence, an old man's eye, when atropinized, looks on superficial examination very like a glaucomatous eye.

concerned, is usually less intense than the first, but results in a still further reduction of the sight. Inasmuch as new attacks now constantly follow each other, at shorter or longer intervals, the sight at length becomes entirely extinct. The disease has then entered upon the third stage (§ 832).

The *acute congestive attack* is often misunderstood. In this the *pain* radiates from the eye into the whole of one side of the head, so that sometimes the patients are not for a moment aware that the pain is proceeding from the eye, but complain simply of violent "rheumatic" headache. If the attack has been accompanied by marked swelling of the lids, a history of erysipelas may be given. One should not allow himself to be led astray by such declarations as these, but should form his decision in accordance with the results of the objective examination. The principal thing that this shows us is the dull and uniformly cloudy look of the cornea. Another important symptom is the dilatation and rigidity of the pupil, and by this we are guarded against any confusion with iritis or iridocyclitis, since in these diseases the pupil is contracted.

A characteristic feature of glaucoma is the frequent, often periodical, *alternations in the course* of the disease. These are afforded by the transient obscurations of vision occurring in the prodromal stage, by the inflammatory attacks recurring at intervals in the inflammatory stage; even after the blindness is complete, there is still a constant alternation of bright and dark days, according to the patient's subjective luminous impressions.

The *character of the pupil* has a very great influence on the phenomena of glaucoma. Its contraction has a favorable effect, since it generally diminishes the tension in glaucoma, while dilatation of the pupil, on the contrary, increases the tension. Hence miotics have the power of cutting short the prodromal attacks, and of ameliorating the symptoms even in the inflammatory attack. The fact that the prodromal attacks can be cut short by the patient's going to sleep is probably also referable to the marked contraction which the pupil undergoes in sleep. Mydriatics, on the contrary, can excite an inflammatory attack in an eye that is predisposed to it, and that not only the powerful mydriatics, like atropine, but also homatropine, euphthalmine, cocaine and, according to some, even dionin and holocaine. We should always, therefore, take care that there is no suspicion of the existence of glaucoma, before making an instillation of a mydriatic in the case of an elderly man. If, however, we have had the misfortune to set up in this way an attack of inflammatory glaucoma, it may be possible, by the prompt and energetic instillation of eserine, to cause the attack to disappear, and that perhaps permanently.

Those who hold that the *ciliary muscle* promotes outflow from the eye (see § 77) believe that a glaucomatous attack may be set up by inaction of this muscle and be prevented by its exercise (e.g., by reading, especially in a strong light) (Elliot).

It is a fact frequently observed that operation, done in a glaucomatous eye, may give rise to a congestive attack in the second eye, if it is already predisposed to glaucoma. Nevertheless, it is not the operation as such, but the associated *mental and physical depression* which, as on other occasions, may here, too, excite a glaucomatous attack. Probably there are two factors that act together in the case of glaucoma produced by violent emotion—the disturbance of the circulation and the reflex dilatation of the pupil. In an operation upon a glaucomatous eye, pilocarpine or eserine may be instilled into the second eye to prevent the outbreak of glaucoma in it; but even this precaution does not afford complete security.

Febrile diseases of various sorts likewise determine an attack of glaucoma—a fact that has been observed quite often, especially in the great influenza epidemics. In India epidemic dropsy is very frequently associated with a rapidly progressive acute glaucoma with early cupping (Maynard). In these cases the blood pressure is unusually high and this occurring in comparatively young subjects may be a factor in causing the attack. Glaucoma has also been observed after herpes zoster.

Injuries, such as blows on the head, abrasions and burns, or corrosive injuries near the limbus, etc., may set up an attack of glaucoma, usually transient (Kuemmell, Elliot).

832. (3) **Glaucoma Absolutum.**—In this, the third stage of glaucoma, the eye is completely blind, and presents the following picture: Contrast-

ing with the porcelain-like, bluish-white sclera are the distended anterior ciliary veins, which unite round the cornea to form a bluish-red circle of dilated vessels. The cornea is shining and transparent, but insensitive; the anterior chamber is very shallow. The iris is reduced to a narrow gray marginal strip, which in places is almost entirely concealed by the limbus, and which at its pupillary margin is encircled by a broad black border. The dilated and rigid pupil is greenish, or of a dirty gray. The optic disc is deeply excavated, the eye is hard as stone.

Later on, degenerative changes make their appearance in the blinded eye, which are designated under the name of *glaucomatous degeneration.*

The changes affect the cornea, sclera, and lens. Those found in the *cornea* are: (1) most frequently marked cloudiness of the parenchyma with glassy, gelatinous or hyaline deposits on the surface; (2) keratitis vesiculosa or bullosa due to degenerative pannus (§ 597 and Fig. 262); (3) zonular opacity (§ 598); (4) ulcers especially under the form of ulcus serpens (§ 550) and often leading to perforation. These affections of the cornea, so various in character, are referable to diminished resistance due partly to the alteration in nutrition produced by the disturbance in circulation, partly to the paralysis of the corneal nerves.

The *sclera* especially in the equatorial region shows dark ectatic prominences.

The *lens* always becomes cataractous (*cataracta glaucomatosa*). Such a cataract produced by glaucoma is to be distinguished from an ordinary cataract (senile, traumatic, etc.) occurring by chance in a glaucomatous eye, and also from a cataract which has produced the glaucoma. The distinction lies in the appearance of the cataract and in the vision. Glaucomatous cataract is distinguished by its marked distention, its bluish-white color, and the vivid silky lustre of its surface, while an ordinary cataract has the appearance corresponding to its origin and its nature. If the cataract has caused the glaucoma, we shall find an intumescent cataract and usually the history of traumatism or operation. In glaucomatous cataract the eye, as a result of the glaucomatous process, is completely blind, and hence an operation for cataract is useless. In the other forms, if the glaucoma is not too far advanced, such a degree of sight (perception of light) may still exist as promises a good result from the extraction of the cataract. The extraction, however, should in no case be performed immediately; on the contrary, the increase of tension should first be done away with by an iridectomy, and this may be succeeded, say some four weeks later, by the cataract operation. If we should extract a lens, without taking any such preliminary precaution, in an eye affected with increase of tension, we would run the risk of losing the eye through severe intra-ocular hæmorrhage (see §533).

Even in the degenerative stage, when the eye has been blind for a long time, the patient still constantly believes that he perceives light, especially under the form of a luminous haze, which on some days is more pronounced, on other days less so. These subjective luminous appearances for a long uphold the patient in the belief that he will be able to regain his sight. Furthermore, attacks of pain keep making their appearance at intervals in the blinded eye.

The *final outcome* in glaucoma is usually atrophy of the eyeball. After the eye has been hard for years it at length becomes softer, smaller, and atrophic. In other cases, ulcus serpens develops with perforation and consecutive irido-cyclitis or even panophthalmitis, together with phthisis bulbi. Occasionally the eye undergoes spontaneous rupture (Harmon, Rutherford). Not until the glaucomatous eye has become shriveled does it allow its unfortunate possessor to have any lasting rest.

833. Varieties in Course.—The course of glaucoma with a violent attack of congestion, here pictured, corresponds to that form which is designated as *acute congestive glaucoma* (*glaucoma inflammatorium acutum*). In the severest cases, which Von Graefe has described under the name of *glaucoma fulminans*, incurable blindness, in conjunction with the most violent symptoms of inflammation, may develop within a few hours. Of much more frequent occurrence than these cases, which luckily are rare, are those which run a less acute and typical course than belongs to acute glaucoma. These cases are designated as *chronic congestive glaucoma* (glaucoma inflammatorium chronicum). Here no pronounced congestive attack takes place; on the contrary, the prodromal stage passes imperceptibly into the stage of inflammation, the eye growing gradually red and sensitive, the cornea acquiring a smoky cloudiness, the iris becoming atrophied. The pain is neither so violent nor so continuous as in acute glaucoma. Often this sort of chronic course sets in after the first congestive attack, no complete subsidence of the symptoms of inflammation taking place. The final outcome is the same as in acute congestive glaucoma; and, anyhow, no sharp line of distinction exists between the two forms.

Glaucoma almost always attacks both eyes. Nevertheless, they are but rarely affected at the same time; it more frequently happens that the disease of the second eye follows months, or even years, after that of the first. The experienced physician, however, even in those cases in which one eye is still perfectly healthy, will frequently recognize in the latter a certain *disposition toward glaucoma.* This manifests itself in a shallow anterior chamber, a somewhat dilated and sluggish pupil, and a comparatively high, even though not yet pathological, tension of the eye.

The eye often is small and the cornea therefore narrower than usual. In patients coming from a suspected stock, these features may be regarded as danger signs (Priestley Smith).

834. Predisposing Conditions.—Congestive glaucoma is usually a disease of advanced life; it is found most frequently between the fiftieth and seventieth year. It never occurs in childhood and but very rarely indeed in youth. It attacks women about twice as frequently as men, especially women in whom the menopause occurs before the time. Rigidity of the vessel walls (arteriosclerosis) and habitual constipation predispose to glaucoma. Again, among Jews, Egyptians, the inhabitants of India, and certain varieties of negroes congestive glaucoma is much more frequent than among other races. Moreover, there are many families in which glaucoma is inherited.

Familial glaucoma, according to Lawford, is continuous in descent, is transmitted by both sexes, exhibits the phenomena of anticipation, and occurs in all forms (Elliot).

Congestive glaucoma occurs more frequently with hyperopia than with either myopia or emmetropia. Highly myopic eyes are said to be almost immune, perhaps because in such eyes, when the intra-ocular pressure rises unduly, the whole back portion of the sclera instead of simply the lamina cribrosa gives way before it, just as in childhood. Low myopia, however, confers no immunity.

B. Non-Congestive Glaucoma (Glaucoma Simplex)

835. In non-congestive glaucoma the increase of tension sets in very gradually, so that no inflammatory phenomena are produced. The eye either looks quite normal externally, or it gives evidence of the lesion affecting it in the greater prominence of the distended anterior ciliary veins, and also in a somewhat dilated and sluggish state of the pupil. The tension of the eye is shown to be elevated, but usually not to any considerable degree. Often on the first examination no increase of tension is found at all; it is not until we examine the eye repeatedly, and especially at different times of the day, that we succeed in demonstrating that the tension is elevated. At these times a slight, smoky cloudiness of the cornea is also sometimes present, such as appertains to the prodromal attacks of congestive glaucoma. Finally, there are cases of non-congestive glaucoma in which the tension is so low all the time that on palpation with the finger it is regarded as normal, while, nevertheless, the tonometer shows a very slight but still certain elevation above the normal. In a few cases no tension surpassing the normal can be demonstrated even with the tonometer.

In consideration of the fact that in non-congestive glaucoma marked external symptoms, and sometimes, indeed, even any increase of tension, are wanting, we are thrown back upon the ophthalmoscopic examination for the establishment of the diagnosis. Such an examination shows the presence of a total excavation of the optic nerve, the depth of which corresponds to the duration of the process.

The subjective symptoms of non-congestive glaucoma, since the inflammatory attacks and the pain are wanting, consist almost exlusively of the disturbance of vision. This manifests itself by gradual diminution of the sight, and in many cases also by transient slight attacks of obscuration, like those belonging to the prodromal stage of congestive glaucoma. The decrease in sight expresses itself in a contraction of the field of vision (see § 828), as well as in a diminution of the central visual acuity. The diminution in central vision often develops late, when the field of vision has already become very small, so that not infrequently the patients are still in a condition to read or carry on fine work, while they are scarcely able to go about any longer alone (see § 263). Before this point is attained, generally a long time (even many years) has elapsed, inasmuch as the reduction of vision takes place very slowly and gradually. For this reason the patient himself often does not become aware of the existence of his disease until late. The blinded eye may either remain always sound externally, or those inflammatory attacks which are characteristic of congestive glaucoma may occur—often, indeed, before the blindness has become complete. Non-congestive, accordingly, not infrequently changes into congestive glaucoma.

Non-congestive glaucoma probably always attacks both eyes. In contradistinction to the congestive form, it sometimes occurs in young people and attacks men as frequently as women. Again it is found as often in myopic eyes as in those that are emmetropic or hyperopic (Gilbert and others cited by Ellliot), whereas congestive glaucoma is found particularly often in hyperopia (see § 834).

The *relation of non-congestive to congestive glaucoma* has been the subject of manifold discussions. Since the former, on account of the absence of inflammatory symptoms, is entirely different externally from congestive glaucoma, it was not recognized as glaucoma at all until the discovery of the ophthalmoscope. Even then the conditions were regarded as different, the cupping of the nerve in glaucoma simplex being attributed to a special form of optic-nerve disease (Jäger and others; cf. Schnabel's theory of cavernous atrophy, §827). But at present the majority of ophthalmologists rank non-congestive glaucoma under glaucoma proper, since it has in common with its most essential symptom, the increase in tension. The interconnection of non-congestive and congestive glaucoma is also proved by the numerous intermediate varieties which form a continuous transition from one to the other, so that no sharp line of distinction can be drawn between the two. A non-congestive glaucoma, later in its course, often passes into the acute or chronic congestive form, and cases not infrequently occur in which congestive glaucoma is present in the eye first affected, non-congestive glaucoma in the one which was affected afterward.

Some cases of non-congestive glaucoma with very slight increase of tension are not always clearly distinguishable from simple optic-nerve atrophy with unusually deep atrophic excavation. In doubtful cases testing of the color perception may furnish a diagnostic guide. In optic-nerve atrophy, color blindness sets in early, while in glaucoma the ability to distinguish colors is retained for a comparatively long time.

836. Buphthalmus.—Buphthalmus[3] also called *hydrophthalmus* is a condition of infantile glaucoma resulting in a uniform enlargement of the eye. In this enlargement the cornea also participates (keratoglobus). The thin sclera is bluish, in consequence of the uveal pigment appearing through it; the cornea is larger and either clear and lustrous or, as in congestive glaucoma, dull and diffusely clouded. The anterior chamber is unusually deep, the iris is tremulous, and the papilla, after the process has lasted a pretty long time, is deeply excavated. The tension of the eye is considerably increased. The refraction is generally moderately myopic.

The disease may come to a stop spontaneously, or may continue until it produces blindness. In the former case, the increase of tension subsides after a time; the unnatural bigness of the eyeball persists, indeed, but does not increase, and the eye retains a moderate amount of sight, the degree of which is mainly dependent upon the condition of the optic nerve. Much more often, the enlargement of the eye keeps on—sometimes until quite extraordinary dimensions are attained—and in the mean time complete blindness ensues. Signs of increase of tension—cloudiness and dulness of the cornea, degenerative pannus, excavation of the optic nerve—accompany this progress, and the more so, the older the patient.

A characteristic sign of buphthalmus are the fine, faint, sometimes branched or crossing striæ in the cornea, which depend upon spontaneous *ruptures of Descemet's membrane*. With oblique illumination and a loupe, the edges of such a rupture appear as a pair of very minute, lustrous, parallel lines; with axial illumination or in the direct method with high power the edges look black (Fig. 372).

The *lens*, is the only part of the buphthalmic eye that retains its normal dimensions —in fact, it not infrequently falls behind them. Hence the lens is too small compared with the surrounding parts, and the space between lens margin and ciliary processes keeps growing larger. Thus the zonule of Zinn undergoes an elongation which leads to its partial atrophy. Hence in buphthalmus of pretty high degree we always find defective

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[3] From βοῦς, ox, and ὀφθαλμός, eye.

fixation of the lens manifested in tremulousness of lens and iris, and not infrequently inducing subsequent luxation of the lens, with its disastrous consequences.

Buphthalmus is either congenital or develops in the first year of infancy and generally in both eyes. Heredity plays an important part in its production. The nature of the disease has not yet been fully cleared up, but increase of tension is certainly the most important factor in it, leading, on the one hand, to the enlargement of the eye, and, on the other hand, to blindness through excavation of the optic nerve. The difference in external appearance between buphthalmus and the glaucoma of adults is accounted for in the main by the fact that in childhood, the extensibility of the sclera renders it possible for the heightened pressure to result in enlargement of the eye as a whole, while in the adult, the sclera is so rigid that it can expand only at its weakest points—namely, the lamina cribrosa and the equatorial region of the eye.

FIG. 372.—FISSURES IN DESCEMET'S MEMBRANE IN HYDROPHTHALMUS (after Salzmann).

In the patient, a boy now 15 years of age, an iridectomy had been done in the right eye (*R*) at the age of 3. In this the cornea is considerably enlarged (15 mm. in diameter). Descemet's membrane shows numerous fissures. The vision is 0.1 with correction (—4.5 D). Fundus normal. The left eye (*L*) which was not operated on, shows only a single fissure, and except for a myopia of 3 D is normal. Cornea 13 mm. in diameter.

Varying conditions have been found to account for the increase of tension: Peripheral synechiæ in some cases; in others condensation of the tissue of the ligamentum pectinatum, obliterating its interspaces; in others abolition of Schlemm's canal or its replacement by narrow capillary passages.

Some authors (Horner, Haab) distinguish between the form above described (infantile glaucoma with early development of fissures in Descemet's membrane) and genuine *megalophthalmus* (megalocornea) which is a simple overgrowth of the eye, and is unattended by increase of tension or by the characteristic fissures. It is impossible, however, to draw any sharp dividing line between the two forms.

Buphthalmus may also be a symptom of *neurofibromatosis multiplex* (Recklinghausen's disease), which is compounded of a number of changes, mostly congenital. These changes, which, it must be admitted, are never all found together in the same individual, are—in the skin, multiple neurofibromata, fibrous mollusca, and flat pigment moles; on the face, neuroma plexiforme and lymphangioma of the lids and orbit and unilateral hypertrophy of the face; lastly tumors of the optic and auditory nerves (§ 824).

Theories of Glaucoma

837. Increase of Tension the Basis of Glaucoma.—All the essential symptoms of glaucoma can be accounted for as being the results of the increase in pressure. The recognition of this fact by Mackenzie, and particularly by Von Graefe, was the most important step made in establishing the theory of glaucoma. The increase of tension produces disturbance of

circulation and this together with the direct effect of the pressure on the tunics of the eye is accountable for the objective signs with the underlying anatomical changes and for the subjective symptoms.

The disturbance of the circulation of blood, which is the first effect of the elevated tension, consists essentially of a *venous stasis.* The heightened tension, that is, brings about a compression of the veins in the interior of the eye, and especially in the vasa vorticosa, which, on account of their oblique course through the sclera, are particularly exposed to the influence of the intra-ocular pressure. The blood flowing away from the uvea is hence compelled, in great part, to travel through the anterior ciliary veins; these are consequently dilated, and, in old cases of glaucoma, form a dense venous network encircling the cornea. In non-congestive glaucoma the symptoms of obstructed circulation are confined to the dilatation of the anterior ciliary veins and to the ophthalmoscopically visible distention of the retinal veins. But in congestive glaucoma, in which the increase in tension and with it the disturbance of circulation set in suddenly, these changes induce the phenomena of *inflammatory œdema* in the same way as the incarceration of a hernia, for example, results in inflammatory œdema of the incarcerated coils of intestine. Inflammatory œdema is characterized by hyperæmia of the tissues and marked swelling, due to their serous infiltration, while—in contradistinction to plastic inflammation— exudates and the adhesions produced by them are wanting. Accordingly the symptoms of acute congestive glaucoma, so far as they affect the uvea, for example, differ greatly from the clinical picture of an irido-cyclitis; posterior synechiæ are only exceptionally observed in it, and exudations of greater degree, such as hypopyon, pupillary membranes, etc., are never observed. This absence of exudation, in spite of the violent external symptoms of inflammation, is, in fact, just the characteristic feature of inflammatory œdema.

838. Objective Signs Produced by the Increased Tension.—The various objective signs of glaucoma are either the direct result of the increased pressure or are due to the circulatory disturbance (œdema etc.) that it causes.

1. That œdema of the *cornea* produces the corneal cloudiness found in glaucoma is proved by anatomical investigation (§ 839). From this can be understood the suddenness of development of the corneal cloudiness, and also the promptness of its recession upon reduction of the pressure—e. g., after puncture of the cornea, or iridectomy. If we were dealing with an inflammatory infiltration of the cornea—i. e., with a keratitis—it could not possibly disappear again within a few hours.

2. Inflammatory œdema of the *iris* manifests itself mainly by discoloration and by the obliteration of the details of its structure. The anterior chamber becomes shallower because the lens is pressed forward together with the iris, in consequence of the elevation of pressure in the vitreous chamber. Added to this there is a displacement forward of the attachment of the iris—that is, the greatly swollen ciliary processes press the root of the iris forward, so that it is applied to the most anterior portion of the sclera and to the margin of the cornea (Fig. 373). Consequently, the iris appears to take its origin farther forward (Figs. 373 and 374). The ciliary nerves are compressed and paralyzed by the high intra-ocular pressure; hence the insensitiveness of the cornea and the paralysis of the iris (iridoplegia) with which are associated dilatation and loss of reaction of the pupil. The dilatation afterward increases still more in consequence of the atrophy of the iris, that develops as a result of the pressure exerted upon the latter.

3. The redness and clouded appearance of the *optic disc* during the inflammatory attack are caused by hyperæmia combined with some œdema; the excavation which subsequently takes place is the direct result of the elevation of pressure (see § 827).

839. Anatomical Changes Produced by Increased Tension.—The anatomical changes corresponding to the objective signs above described are as follows:

In the *cornea* the cause of the cloudiness is found to be œdema of the epithelium. The fluid producing the œdema is found under the form of minute drops, between Bowman's membrane and the epithelium, and also between and in the epithelial cells themselves (Fig. 375). Even though this liquid itself may be quite clear it must cause a cloudiness of the whole epithelial layer, if it has an index of refraction differing from that of the epithelial cells. The dullness of the corneal surface is due to the fact that the epithelial cells are forced apart by the liquid and are pushed up in places so that the surface of the cornea is covered with minute inequalities (Fig. 375). When this elevation of the epithelium takes place over a pretty large area, small vesicles are formed upon the surface of the cornea.

In the *sclera* an increase in density has been demonstrated, and also a fatty degeneration of the fibres, which look as if sprinkled with minute drops of oil.

The *aqueous* is more albuminous, and coagulates readily in the air and in hardening fluids.

The most important changes affect the *uvea*. In recent inflammatory cases it

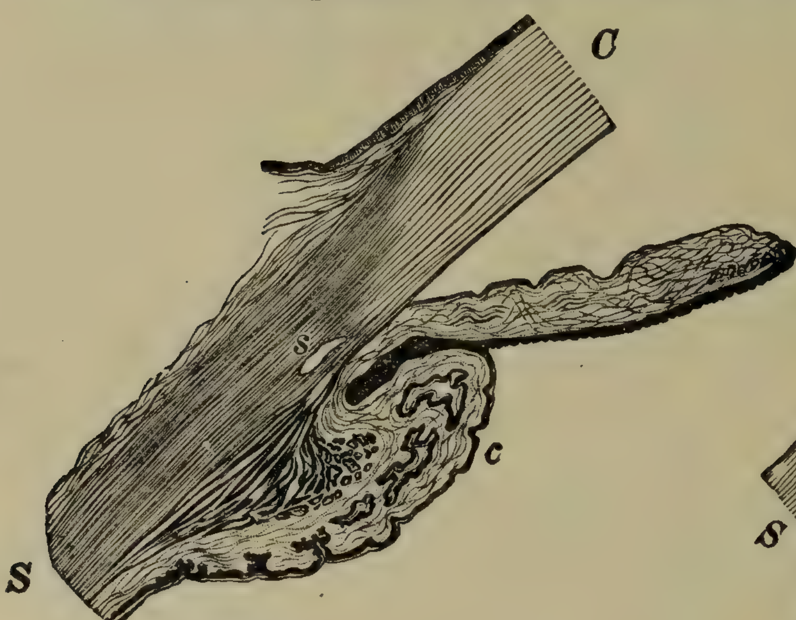

FIG. 373.

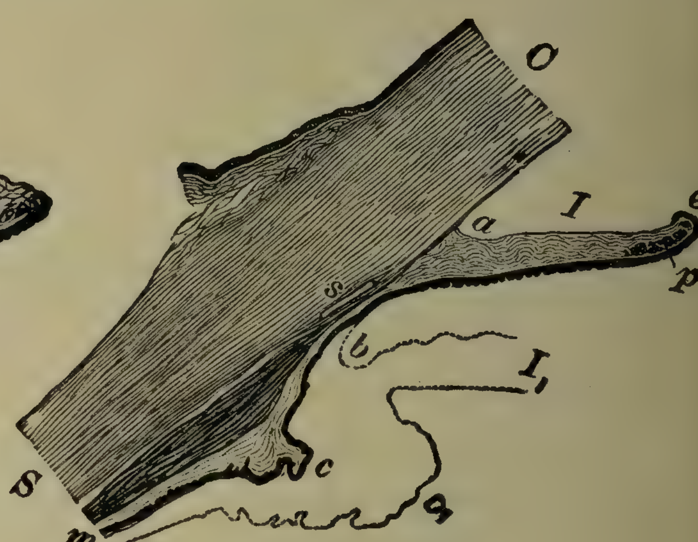

FIG. 374

FIG. 373.—IRIS AND CILIARY BODY IN RECENT CONGESTIVE GLAUCOMA. Magnified 9 × 1 The ciliary process, *c*, is so greatly swollen that it pushes the root of the iris forward and presses it against the sclera, *S*, and the cornea, *C*. The sinus of the anterior chamber, which should lie somewhat behind Schlemm's canal, *s*, is thus closed. The ciliary muscle shows the pronounced development of the circular muscular fibres (Müller's portion), characteristic of the hyperopic eye.

FIG. 374.—IRIS AND CILIARY BODY IN OLD CONGESTIVE GLAUCOMA. Magnified 9 × 1. The dotted line gives the outline of the iris, I_1, and the ciliary body, c_1, in the normal condition. The root of the iris is adherent to the sclera, *S*, and the cornea, *C*, wherever it has been pressed against them by the ciliary body. The attachment of the iris is hence displaced forward and lies in front of Schlemm's canal, *s*. So, too, the sinus of the anterior chamber is displaced from *b* to *a*. Wherever the iris has become adherent, it has been thinned through atrophy, so that in places—*b*, for example— it consists of scarcely anything more than the pigment layer. Even the free portion of the iris, *I*, appears in consequence of its atrophy narrower than the normal iris, I_1. Over the pupillary border, *e*, the retinal layer of pigment turns forward farther than usual, and the sphincter pupillæ, *p* also shares to some extent in this eversion. The ciliary body owing to its having become atrophic, has again separated from the iris, and in fact more so than in the normal condition, so that it is now removed from the iris by a broad interval. The atrophy affects both the ciliary muscle, *m*, and the ciliary process, *c*.

presents the appearances of inflammatory œdema—i. e., infiltration with an abundant, readily coagulable liquid, while white blood corpuscles that have emigrated from the vessels are present in but scanty numbers; but what is most striking is the marked distention of all the venous vessels, in consequence of which extravasations of blood are produced in many spots. The ciliary processes in particular, which of all the structures of the eye possess the most veins, are greatly swollen through the turgidity and distention of the vessels, and press the periphery of the iris against the sclera and the cornea. These structures become agglutinated, so that the periphery of the iris remains permanently connected with the sclera and cornea (peripheral anterior synechia), and so continues even when later on the ciliary processes draw away again from the iris (Fig. 374). This retraction takes place in consequence of the atrophy which, after the subsidence of the early congestive symptoms, sets in in all parts of the uvea. In the *iris*, the atrophy finds expression in its becoming narrower and thinner. Rigid connective

tissue, from which the blood-vessels have for the most part disappeared, takes the place of the delicate network of anastomosing cells. In the vessels which are still present the walls are thickened, and the lumen is thus contracted or, even entirely obliterated (Ulrich) The muscular bundles of the sphincter pupillæ, too become atrophied. The part which. is best preserved is the retinal pigment layer, which by the great shrinking of the anterior layers of the iris, is drawn continually farther and farther forward over the edge of the pupil (ectropion of the pigment layer; Fig. 374, *e*). Hence, when we look at the eye from in front we find the margin of the pupil encircled by an unusually broad black rim, which sometimes covers half the breadth, or even more, of the surface of the iris. The atrophy attains its greatest height at the root of the iris—i. e., at that part which is adherent to the sclera and cornea (Fig. 374, *b*.) In old cases nothing of the iris is left in this spot but the retinal pigment layer and one or two of the larger vascular trunks. These remains of the iris are intimately adherent to the wall of the eyeball; the ligamentum pectinatum is condensed into a tough, fibrous tissue; and, finally, even Schlemm's canal disappears. Through atrophy also the *ciliary body* diminishes in size, so that it draws away again from its contact with the iris, and afterward becomes flatter and flatter, until at length it scarcely forms any projection at all (Fig. 374, *c*). The atrophy affects the ciliary muscle as well as the ciliary processes. In the *chorioid* the atrophy finds expression in the obliteration of the blood-vessels and the rarefaction of the pigment, so that the chorioid is finally in some spots reduced to a thin, transparent pellicle. This sort of atrophy of the chorioid takes place above all in the neighborhood of the papilla, and by it there is formed the halo glaucomatosus visible with the ophthalmoscope (*h*, Fig. 369 A). The atrophy also reaches a high degree in the spots where the vasa vorticosa pass from the chorioid into the sclera. The chorioid here grows fast to the sclera, which becomes thinned, and, together with the chorioid, bulges out to form an equatorial staphyloma. In the vasa vorticosa themselves is found proliferation of the vascular endothelium, leading to contraction, or even obliteration, of the lumen of the veins (Czermak and Birnbacher).

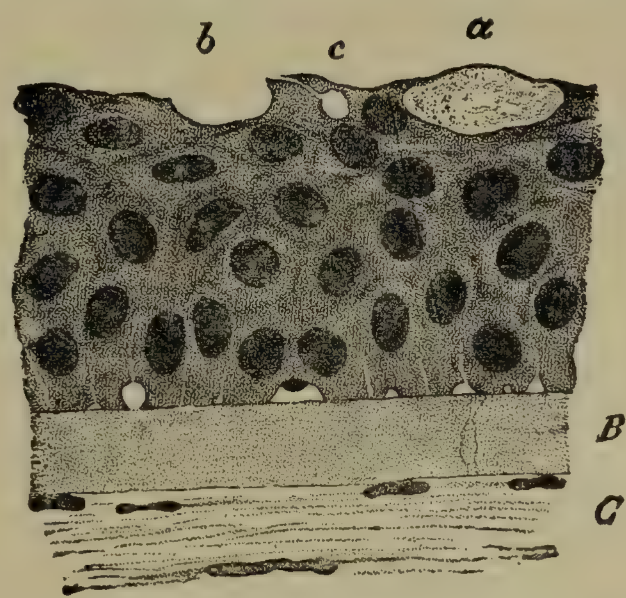

FIG. 375.—CORNEAL EPITHELIUM IN A CASE OF INCREASE OF TENSION. Magnified 500 × 1.

The specimen was from a case in which increase of tension set in after irido-cyclitis. *C*, parenchyma of the cornea with the flat nuclei of the corneal corpuscles. *B*, Bowman's membrane, through which in two places minute nerve filaments are seen passing. At the anterior ends of these nerve filaments and also at other spots there are seen between the bases of the lowermost cylindrical cells (foot cells) light-colored rounded spaces, which represent very minute droplets of liquid. The lines of division between the foot cells are in general represented by lighter colored lines, which indicate that the cells are to a certain extent pressed apart by fluid and their interconnections broken up. On the other hand, the cells of the middle layer are bordered by broad, dark lines representing the interlocking teeth of these, the pectinate cells. In the uppermost layers there are numerus cells altered by imbibition of fluid. In the cell *a*, this fluid occupies the greater part of the cell body, so that the cell itself is enlarged. At *b*, the anterior wall of a cell that was filled with fluid has fallen off. At *c*, the liquefied contents of a cell have been discharged through a slender opening. Owing to these changes in the uppermost cells, the surface of the epithelium as a whole is uneven.

At the *optic-nerve entrance* the particularly striking feature is the displacement of the lamina cribrosa. This is condensed by the compression of its layers, and is displaced backward so that not infrequently it even gets to lie behind the outer surface of the sclera (Fig. 369 B, *e*). The excavation of the papilla thus produced contains upon its floor atrophic nerve fibres, neuroglia, and some connective tissue (Fig. 369 B, *b*). Large excavations get to have overhanging edges (become ampulliform), because the short canal in the sclera, which is designed for the optic nerve, and which is laid bare by the excavation, is wider behind than in front (see Fig. 193).

In the head of the optic nerve and also behind the lamina cribrosa, gaps (lacunæ) of greater or less size are frequently formed (Schnabel). (See § 827).

In consequence of the destruction of the head of the optic nerve, the retina and also the trunk of the optic nerve atrophy; the latter becomes thinner, as a whole, and shows that its connective-tissue trabeculæ have been enlarged at the expense of its nerve fibres (Fig. 369 B, *n*).

Precise as is our information in regard to the anatomical changes above described, and many others, too, in glaucoma, we must be correspondingly cautious in the interpretation of their significance, if we are bent upon finding out the anatomical cause of glaucoma. Most of these changes, if not all, are simply the result of the increase of pressure. To find those changes which precede the increase of tension and cause it, we should have to examine the eye in the earliest stages of glaucoma, and the opportunity for doing this is but rarely offered.

840. Subjective Symptoms Produced by Increased Tension.—The violent *pain* in congestive glaucoma is excited by the compression of the extremely numerous sensory nerves of the ciliary body and the iris.

The *disturbance of vision* is also the result of the heightened intra-ocular pressure. It may occur in *acute* fashion, being then due to—

(*a*) The opacity of the cornea, which affects chiefly central vision, and that, too, to a marked degree, on account of its uniform diffusion.

(*b*) Acute ischæmia of the retina, due to the compression of the retinal arteries, by which is caused a contraction of the field of vision.

This compression makes itself felt first in those sections of the arterial channel in which the blood pressure is lowest. But, since the blood pressure in the vessels is less the farther we get away from the heart, the terminal expansions of the arteries lying in the periphery of the retina are first affected by the compression. Hence the periphery of the retina first becomes insensitive to light, a thing which manifests itself by contraction of the field of vision.

Both the opacity of the cornea and the acute ischæmia of the retina belong solely to congestive glaucoma, and it is they which are the principal cause of the very great reduction of sight during the acute attack. They are of transitory nature, disappearing again as the elevated tension abates, and at the same time with their disappearance an improvement of the visual acuity and an enlargement of the field of vision set in.

A *chronic* steadily progressive disturbance of vision occurs in all cases of glaucoma, congestive and non-congestive. This is due mainly to—

(*a*) Direct pressure upon the optic-nerve fibres and their consequent atrophy—atrophy which takes place during the formation of the glaucomatous excavation. Until atrophy sets in vision diminished in this way may be restored, provided the pressure is removed; but a disturbance due to actual atrophy is permanent.

The pressure evidently exerts itself most on the supero-temporal and infero-temporal bundles (§ 47), the explanation for this apparently being that the fibres in these bundles are so crowded on the disc that they are more readily nipped by pressure in their passage over the edge of the excavation. It is lesion of these bundles that produces Seidel's and Bjerrum's signs and Rönne's step (§ 828) so often present in glaucoma.

Not only pressure but also *traction* may be exerted on the nerve fibres. In the process of excavation the fibres from the periphery of the retina, which probably pass to the centre of the disc, would be more subjected to traction than the fibres of the central area of the retina, which end at the margin of the disc, and this predominant traction on the peripheral fibres may account for the peripheral contraction of the field occurring quite constantly in glaucoma (Elliot).

Other factors which may possibly cause chronic progressive disturbance of vision and particularly the contraction of the field are—

(*b*) Chronic ischæmia of the retina, acting like acute ischæmia, but more gradually.

(*c*) Pressure on the retinal nerve-terminals.

841. Explanation of the Increase of Tension.—Easy as it is to deduce the symptoms of glaucoma from the elevation of tension, it is quite as difficult to account for the origin of the latter, and thus explain the essential nature of glaucoma. Of the many theories which have hitherto been propounded regarding this, no single one is altogether satisfactory.

In weighing these theories one must consider *upon what circumstances increase of tension in general depends.*

The intra-ocular pressure is determined by the relation between the internal capacity and the elasticity of the envelopes of the eyeball, on the one hand, and the amount of its contents, on the other. If the latter factor increases or the former factors diminish, the pressure is elevated. The volume of the eyeball changes very slightly and its elasticity is small, so that no very great changes in the internal capacity of the eyeball can occur. The prevalent theories therefore presuppose as the cause of the elevation of tension an increase in the amount of contents of the eyeball, the envelopes of the latter not being sufficiently elastic to adapt themselves to their increased contents without marked heightening of the pressure. Now the amount of matter contained in the eyeball depends, on the one hand, upon the amount of ocular fluids which are constantly being emitted from the blood-vessels, and, on the other hand, upon the quantity of fluid which leaves the eye again through the lymph passages; it corresponds, that is, to the relation between inflow and outflow.

In the normal eye this relation always remains about the same, since with any increase of the inflow the liquid, which is now subjected to an increased pressure, also leaves the eye more quickly, so that the normal pressure is soon restored. For any permanent increase of pressure to occur, a disturbance of this self-regulating action must be present. According to the prevalent theory, that of Knics and Weber, such a disturbance can be looked for only in the excretory apparatus, it being held that so long as this works normally, every increase in the amount of fluid would soon be compensated for by increase in the outflow while, if the outflow is interfered with, a normal or even a diminished inflow of liquid must lead ultimately to over-distention of the eyeball. The essence of glaucoma is, therefore, regarded as consisting in a *disturbance of the outflow.* The most important path for the outflow lies in the angle of the anterior chamber and passes through the ligamentum pectinatum into Schlemm's canal. It is at this spot that the obstacle must be looked for. Such an obstacle is produced in the following way:

A genuine glaucoma develops only in an eye which has a *predisposition* to it. As Priestley Smith showed, this predisposition depends largely on insufficient spatial relations. These particularly affect the region of the anterior and posterior chambers. They are afforded by: 1. Smallness of

the eye as a whole, the average diameter of the cornea in glaucoma being only 11.1 mm.; 2. Shallowness of the anterior chamber;[4] 3. Undue protrusion of the ciliary processes; 4. Disproportionate size of the lens (Fig. 376). By these conditions the space between the lens and the ciliary processes is made too narrow.

It is through this space that the lymph flows from the vitreous to the aqueous cavity. When this *circumlental space is contracted* the lymph becomes dammed up in the vitreous. The latter consequently increases in volume, pushes the lens and iris forward, and thus makes the anterior chamber still shallower. An actual increase of tension, however, is not yet

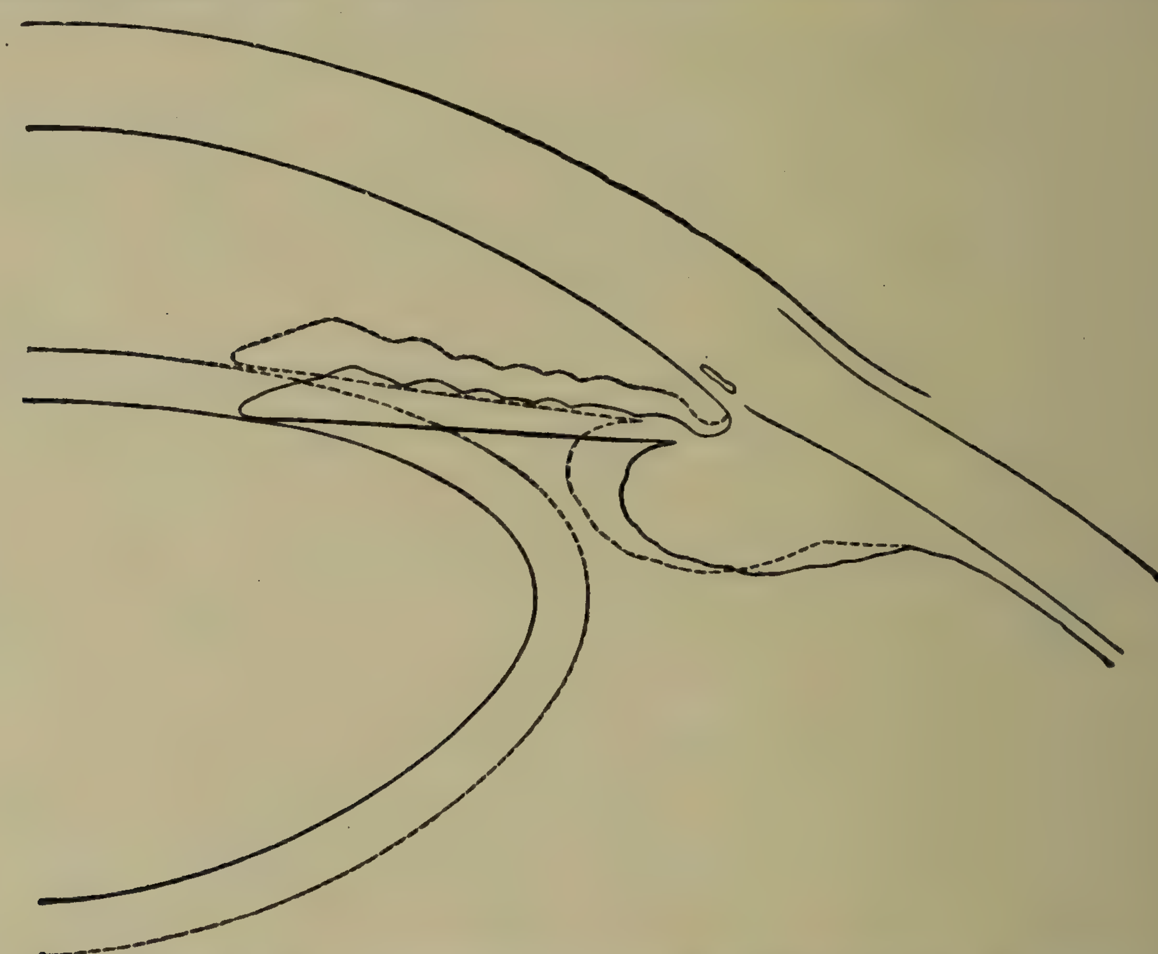

FIG. 376.—SCHEMATIC REPRESENTATION OF THE PREDISPOSITION TO GLAUCOMA.

The unbroken line corresponds to the spatial relations in the young emmetropic eye, the dotted line to the relations in the senile hypermetropic eye. In the latter the ciliary body juts further forward. The lens is enlarged. Owing to the increase of its equatorial circumference, its margin extends further toward the periphery, and, owing to the enlargement of the sagittal diameter, the iris is pushed forward so that the anterior chamber is shallower. The figure has some resemblance to Fig. 84, which represents the process of accommodation. The important difference between the two consists in the fact that in the latter figure the margin of the lens recedes by the same amount that the ciliary body advances, so that the circumlental space remains of constant width. In Fig. 376, on the contrary, the ciliary body and the margin of the lens advance toward each other and thus contract the circumlental space.

present in such an eye because the sinus of the chamber is still present, so that an increased amount of aqueous, such as would correspond to the increased volume of the vitreous, can be still discharged exteriorly. Actual glaucoma does not occur in the predisposed eye until this passage of outflow is blocked. This takes place from *closure of the angle of the anterior chamber.*

[4] As Priestley Smith says, the shallowness of the anterior chamber in persons affected with glaucoma has a double significance. In part it is a pre-existing condition, constituting one of the causes of glaucoma; in part it is a condition produced by the glaucomatous process itself and particularly by the glaucomatous attack (see § 831). The pre-existing shallowness is due to the continuous growth of the lens making it too big and too broad and perhaps also to peculiarities in shape of the suspensory ligament. The additional shallowness produced by the attack is probably due to turgidity of the ciliary processes resulting from sudden congestion. The swollen processes either imprison the vitreous behind the lens and so push the latter forward, or as they swell advance and draw the lens with them (Elliot).

Chief among the conditions that act to close the angle are congestion of the ciliary processes and dilatation of the pupil. During the congestion the ciliary processes swell up and, as the circumlental space is so narrow, soon extend to the margin of the lens. In this way the communication between the vitreous cavity and the anterior chamber is still more interfered with, and the vitreous, owing to the retention of lymph, swells up to a still greater estent than before, and pushes forward still more strongly upon the swollen ciliary processes. Since the latter are already abutting against the margin of the lens, they can give way toward the front only. They swell forward into the posterior chamber until they reach the root of the iris, and jam the latter forward against the corneo-sclera (Fig. 373). But in doing so the iris shuts up the passage of outflow for the aqueous, and, hence, the intra-ocular pressure at once necessarily rises.

Doubtless, one of the chief factors in causing ciliary congestion and in inducing both congestive and simple glaucoma is loss of vaso-motor control in the body or in the eye. The glaucoma set up by irritation of the fifth nerve and the sympathetic are to be ascribed to this factor, the effect of which is also seen in the various exciting causes of glaucoma in general and especially of the acute attack, viz., hunger, cold, sleeplessness, nervous and bodily exhaustion, anxiety, emotional stress, constipation, cardiac obstruction, etc. (Elliot).

Fig. 377.—Iris and Ciliary Body in a Very Hypermetropic Eye in Dilatation of the Pupil. Magnified 9 × 1.

The iris is contracted and thickened, so that at *a* it comes into contact with the posterior surface of the cornea and cuts off the sinus from the rest of the anterior chamber. The ciliary body is unusually large; the circumlental space narrow.

Glaucoma may also be set up in a predisposed eye by *dilatation of the pupil* (as a result of psychical excitation, or by mydriatics, etc.) When the iris grows narrow in the process of dilatation of the pupil, it increases respondingly in thickness. If this takes place in an eye which has markedly projecting ciliary processes, on the one hand, and a shallow anterior chamber, on the other, the thick iris will block the narrow sinus of the chamber (Fig. 377) and thus excite increase of tension (Priestley Smith). It is not the root of the iris that in this case is pushed against the cornea, for the root of the iris is very thin. But directly to the inner side of the root the iris attains its full thickness, so that here its anterior surface turns up and passes abruptly forward; and it is this point (*a* in Fig. 377) that first comes into contact with the posterior surface of the cornea when the iris is thickened. In this way the sinus is closed off so as to form a ring-shaped space which no longer communicates with the anterior chamber. Then in both anterior and posterior chambers, the pressure rises and forces the most peripheral, or root-portion of the iris against the sclera.

Additional factors that may tend to block the channels of outflow are fibrosis of the pectinate ligament (Henderson), degenerative changes in the ciliary body, alteration in the character of the ocular fluids rendering them more viscid, autogenous intoxication producing temporary or permanent alterations in the channels of outflow or possibly interfering with the pump action of the ciliary muscle (Thomson—see § 77).

The Glaucomatous Attack.—According to circumstances the above factors may produce a *gradual* or a sudden stoppage of outflow at the angle of the chamber. A *sudden* stoppage produces an acute attack of glaucoma. If in this case the swelling of the ciliary processes soon recedes or the pupil soon contracts again, the iris returns to its former position, the sinus of the chamber becomes free, the tension falls, and the attack passes off. This is what occurs in a prodromal attack. Czermak accounts for the return to the normal in this case upon the supposition that owing to the increase of tension a state of irritation sets in, which by reflex action causes contraction of the pupil, so that the iris is again drawn away from the cornea. But, in order for this to take place, it is

necessary that the sphincter pupillæ should be strong enough, and, moreover, no adhesion must have formed between the iris and the cornea. But if a return to the normal condition does not take place soon, the root of the iris becomes agglutinated to the corneosclera, and out of this agglutination an adhesion afterward develops (Fig. 374). Then a return to the normal conditions has become impossible, and the glaucoma is permanent.

The Effect of Age.—A number of the factors preventing the passage of lymph from the vitreous to the aqueous chamber or from the aqueous chamber out of the eye *increase with age.* In age, for example the eye becomes slightly smaller, the anterior chamber grows shallower, and the lens which keeps growing all through life becomes in some cases too big for the eye (Priestley Smith). Furthermore autogenous intoxications, fibrosis of the pectinate ligament, and vaso-motor disturbances would be particularly evident in advanced life, not only because more often present then but also because of the cumulative effect of their repeated action in past years and because of the diminished resistance of the system in the aged. Accordingly, age is one of the chief factors in the development of glaucoma, especially congestive glaucoma. Glaucoma occurs but rarely before the age of thirty; after that, its absolute and still more its relative incidence increases with every decade.

Glaucoma also occurs more frequently in the *hyperopic eye* because the latter on the whole is smaller, its anterior chamber shallower and the ciliary processes protrude more than usual on account of the hypertrophy of the ciliary muscle.

842. Other Theories of Glaucoma.—Of the other theories which seek to account for glaucoma, that of Donders, which ascribed it to *increased secretion* on the part of the uvea, holds the first place. This increased secretion Donders ascribed to irritation of the secretory nerves, Von Graefe to inflammation of the chorioid, Bjerrum to inflammation of the ciliary body. This theory, discarded for a time, has been renewed in recent years by some of the best investigators, and there are certain arguments in its favor.

Still *other theories* that have been advanced are (*a*) increase of blood pressure in the vessels of the eye (Stellwag); (*b*) increase of general blood pressure[5]; (*c*) œdematous and vascular conditions at the posterior pole of the eye (Fergus and others); (*d*) cavernous atrophy of the optic nerve (Schnabel—see § 827); (*e*) increase in the amount of hydrophilous colloids in the eye, due to acidosis and causing œdema of the eyeball (Fischer and others). None of these seems tenable as explanations for glaucoma in general (Elliot).

So far, no explanation of glaucoma has yet been propounded which is satisfactory in every respect. The reason for this is perhaps to be found in the statement that all cases of primary glaucoma probably do not develop in the same way, so that *one* explanation could not possibly fit all cases. And, in particular, it might be possible that congestive and non-congestive glaucoma would have to be referred to different causes. In non-congestive glaucoma the sinus of the anterior chamber is often found to be open, so that closure of the latter cannot be the cause of the increase of tension. Here altered composition of the ocular fluids or excessive secretion may be the important factor. In many cases, doubtless, various conditions contribute to produce glaucoma.

Treatment of Primary Glaucoma

843. Operative Treatment.—There are two main methods of treating glaucoma—by operation and by miotics and massage. The chief purpose of operation is to reduce the tension by opening a channel for the ocular liquids to escape either into the normal channels, or into the perichorioidal space, or through the cornea or sclera to the exterior of the eye. The operation may aim to produce a temperary or a permanent effect.

A. **Operations producing a temporary effect** are *paracentesis of the cornea* (§ 941) and *posterior sclerotomy* (§ 976). They are done either in

[5] It seems likely that when, as often happens, high vascular tension and glaucoma are associated, one condition is not dependent on the other, but both arise independently from the same exciting causes constantly repeated. Nevertheless, recent careful researches have shown in a comparatively small number of cases the high incidence of vascular disturbances and at least incipient renal changes, and in cases under fifty a very high incidence of syphilis, which acts doubtless through the vascular changes it induces (Charlin).

cases in which the high tension is probably transient (hence more often in secondary glaucoma) or simply as a preliminary to a more radical interference.

Thus in a case in which the anterior chamber is very shallow, a posterior sclerotomy by relieving the tension in the vitreous chamber may enable the iris to recede so that the conditions will be better for an iridectomy.

B. **Operations designed to produce a permanent effect** on the tension are both numerous and varied. Opinions are still much divided as to their respective merits and indications. Following are the main operations employed and their indications according to the view of the translator.

I. Operations designed to *restore natural channels of outflow.*—Of these the prime one is—

1. *Iridectomy* (§§ 947 and 952). This by common consent is the operation of choice in acute congestive glaucoma. It is also done in chronic congestive glaucoma and more especially in the cases marked by continuous high tension or by repeated acute or subacute exacerbations in which the tension rises more or less noticeably. The earlier it is done in either acute or chronic congestive glaucoma the more likely it is to be successful. For iridectomy acts primarily by opening the closed chamber angle, a thing which is possible only when the angle has not been permanently closed by exudation. After permanent closure by a peripheral synechia iridectomy can do good only in case it produces a filtering cicatrix. In such cases sclerectomy is preferable. This is also true of cases of non-congestive glaucoma in which the value of iridectomy is dubious and in buphthalmus in which on account of the large circumlental space iridectomy may be associated with loss of vitreous or in older patients even with expulsive hæmorrhage.

In certain cases iridectomy is difficult or dangerous, e. g., when the anterior chamber is almost or quite abolished, the tension is very high, in hæmorrhagic glaucoma, in advanced cases in which the field is greatly contracted (see § 844), in cases in which the iris is bound down by adhesions or is atrophic, in dislocation of lens into the vitreous (in which case iridectomy would be followed by loss of vitreous), and in aphakia. In such cases another operation (trephining or cyclodialysis) must be substituted.

In certain cases, e. g., when the anterior chamber is abolished or the tension very high, a substitute operation—cyclodialysis, posterior sclerotomy, or even paracentesis may be done as a preliminary to iridectomy, which such operations render feasible.

Another operation designed to open the natural channels of outflow by re-establishing communication with Schlemm's canal is De Vincentiis' *puncture of the chamber angle* (§ 960). This has been done at times with good result when other operations have failed (Fuchs). It is particularly applicable in cases of non-congestive glaucoma in which the anterior chamber is fairly deep.

2. *Cyclodialysis* (§ 960) is an operation effecting communication not with the regular channels of outflow, but with the perichorioidal space. The results are good, but frequently they are not lasting. The operation is hence reserved for cases in which other operations have failed or have

proved fatal to the other eye or cases in which iridectomy is difficult or dangerous (see above). In such cases even if not permanently successful it may so alter the conditions (e. g., by restoring the anterior chamber or reducing an excessive tension) that an iridectomy, previously impossible, can be done with safety.

II. Operations *producing a filtering cicatrix.* These operations help when the normal channels of outflow are blocked by a peripheral synechia (§ 839). In not a few cases, as Von Graefe recognized, especially when the wound gapes and iris tissue protrudes, iridectomy acts by producing a filtering cicatrix. But this effect is more directly and much more surely obtained by operations specifically designed for this end, (§ 955) especially sclerectomy. These operations include—

1. *Anterior sclerotomy* (§ 956). This includes the bridge sclerotomy of De Wecker and the small-flap sclerotomy of Herbert.

De Wecker's operation, formerly much practiced, was found in many cases not to afford lasting results, so that most operators came to confine its use to cases in which iridectomy was either inapplicable or had been tried without success (Fuchs). In recent years it has been largely superseded by the more certainly acting sclerectomy.

2. *Sclerotomy with inclusion of iris* (iridotasis, iridencleisis—see § 957).

These operations appear to be effective but would seem to carry with them a greater danger of secondary infection. Somewhat resembling them, but with the inclusion of a thread instead of the iris is seton-drainage. (§ 957).

3. *Sclerectomy* (§§ 958 and 959), including Elliot's trephine operation, Lagrange's sclerectomy, Fergus's combined trephining and cyclodialysis, and Holth's punch operation.

Of all the operations for producing a filtering cicatrix the best seems to be that of Elliot. It is particularly applicable in the more chronic and particularly in the later forms of chronic congestive glaucoma and also in non-congestive glaucoma and buphthalmus. Its chief defect is the danger of late infection which occurs in a moderate proportion of cases.

III. Operations to *relieve pain.*—In a blind eye any one of the above operations that is technically feasible may be done to relieve pain, or prevent the development of glaucomatous degeneration. If the pain cannot be helped in this way or if the eye is much disfigured by degenerative changes, *enucleation* or one of its substitutes should be performed.

844. Prognosis of Operation.—The success of an operation in respect to vision can be estimated approximately beforehand, if account is taken of what morbid changes can and what can not be removed by the operation. Iridectomy and the other operations reduce the intra-ocular pressure to the normal amount. They remove the glaucomatous cloudiness of the cornea and the disturbance of vision produced by it, as well as the disturbance of vision caused by the compression of the retinal vessels. But the excavation and the atrophy of the optic-nerve fibres associated with it either do not abate at all or do so in but very slight degree, so that the disturbance of sight, as far as it is dependent upon them, persists. From these facts is deduced the effect of operation in the separate forms of glaucoma:

(*a*) In *congestive glaucoma* the result of operation in recent *acute cases* is extremely favorable. The pain accompanying the glaucomatous attack ceases a few hours after the operation, the cornea in the next few hours or days becomes clear and sensitive once more, and the other inflammatory symptoms likewise speedily disappear. The sight, which during the attack was very much reduced through the cloudiness of the cornea and the compression of the retinal vessels, increases very considerably after the disappearance of these factors. If it was still normal before the attack, it becomes almost normal again after the operation. But if a long prodromal stage with the formation of an excavation has preceded the attack, both the acuity of direct vision and the visual field have already ceased to be normal some time before the attack, and will then be more imperfect still after the operation. We may accordingly put it down as a rule in acute congestive glaucoma that, provided we make an iridectomy *soon after the outbreak of the inflammatory attack*, a degree of sight is secured, *which is somewhat, but not much, smaller than it was before the attack*. The later the operation is done after the inflammatory attack, the less successful it is. In a few cases, to be sure, the result of the operation is to this extent not so favorable, that, in spite of the operation's being correctly performed, the increase of tension either keeps up or sets in anew. In these cases it is generally possible to attain our end by a second operation (iridectomy or sclerectomy). Finally, there are cases in which, in spite of all attempts at operative interference, complete blindness supervenes. These unfavorable cases, however, are rare in acute glaucoma; so that, as a rule, one may count upon a good result from the operation, and one, too, which is lasting.

In the unfavorable cases blindness ensues either because a very slight increase of tension insidiously sets in or without increase of tension and simply as a result of a gradually advancing atrophy of the optic nerve, just as is the case in non-congestive glaucoma (see infra). But as this outcome does not usually set in until after years have elapsed, and as furthermore glaucoma is a disease of advanced life, most of the patients do not survive to experience this melancholy sequel, so that in general the prognosis of iridectomy in congestive glaucoma may be regarded as favorable.

In glaucoma the sight may be impaired or even lost by accidents due to the operation itself, including traumatic cataract and hæmorrhage, or by late infection (see §§ 948, 956, 958, 959). Detachment of the chorioid also often occurs but usually does little harm.

In *chronic* congestive glaucoma in determining the prognosis of operation we must estimate how much of the disturbance of vision present is to be charged to the cloudiness of the media, and how much is due to the excavation and atrophy of the papilla of the optic nerve. The former portion is removed by operation, the latter not.

(*b*) In *non-congestive glaucoma* the result of operation is less pronounced and also less permanent than in congestive glaucoma. The visual disturbance in the variety of non-congestive glaucoma, in which the media are clear, is caused solely by the changes in the papilla of the optic nerve. Since operation cannot remove these changes, it is impossible to restore by means of it the normal acuity of vision. Operation can only do away with the increase in tension, and thus check the advance of the process. If it is

successful, the regular result is that the sight is maintained in statu quo by it, or at most, is slightly improved. In many cases a repetition of the operation is necessary in order to obtain even this result, or the failure of sight goes on unchecked in spite of the operation. This may even take place when, in consequence of the latter, the intra-ocular pressure has become permanently normal. We then assume that an atrophy of the optic nerve fibres, when once initiated, keeps on progressing in spite of the reduction of the intra-ocular pressure. Particularly unfavorable are those cases in which operation, especially iridectomy, actually exerts a bad effect upon the sight, the latter falling away very rapidly after the operation, so that blindness sets in earlier than it would have done without it.

This bad result is to be apprehended when the field of vision was so very much contracted before the operation that its limits at one spot reached nearly up to the point of fixation. Then a slight intussusception of the confines of the visual field carries them beyond the point of fixation, so that central vision is lost. Hence the rule is to operate as early as possible, while the field of vision is still large.

In considering the prognosis of operation in non-congestive glaucoma, we must always bear in mind that no case can be called a definite success until the lapse of years has proved it so. Some cases, probably many, are apparently successful, simply because the patients die before the disease has advanced to the point of blindness.

Sometimes inflammatory symptoms and pain make their appearance just after the operation, when they were not present before. The eye feels hard directly after the operation, the anterior chamber fails to re-form, and the eye becomes blind rapidly and with symptoms of violent pain. These cases, which, to be sure, are rare, are known as *glaucoma malignum.*

In *buphthalmus*, operation, especially iridectomy, is more difficult and dangerous than in adult glaucoma (see § 843). Nevertheless, good results are often obtained by operation, especially one like trephining which does not open the sclera widely.

845. Time and Choice of Operation.—In considering the prognosis and choice of operation in glaucoma, one factor of determining importance is the time at which the operation is done. Nothing is more certain than that in glaucoma of all kinds, if operation is to be done at all, it should be done as early as possible. In congestive glaucoma it should be done in the prodromal stage, in case the patient can make up his mind to it. If he waits until the inflammatory attack, we cannot tell how severe it is going to be, and, in any case, we operate then under less favorable conditions. Under all circumstances the operation is demanded in the prodromal stage when the other eye is already rendered blind by glaucoma; in this case, too, the patient will more readily comprehend the necessity of the operation. When operation is done thus early in glaucoma (in the prodromal or acute congestive stages) iridectomy is indicated if technically feasible; if not, some other operation may be done. In some cases this operation may be merely a provisional one, like a posterior sclerotomy or a cyclodialysis, done with the intention of modifying the conditions so that an iridectomy can be done directly afterward.

In the more advanced and chronic stages of congestive glaucoma sclerectomy, especially trephining is preferable to iridectomy.

In non-congestive glaucoma it is not a question of interfering within a few days or weeks, as is often the case in congestive glaucoma, but even here the earlier we operate, the better. In this case the prognosis depends largely on the field, the larger the latter, the better being the chance of success. On the other hand, with a very small field not only may we fail to check the process, but may actually hasten it (§ 844). In no case should we allow a field steadily to contract without operating. Another factor in the prognosis is the tension. The more evidently this is elevated, the more likely is operation to help. The operation apparently of most service here is sclerectomy (trephining or the Lagrange operation) or if this fails cyclodialysis. These latter operations are specially indicated both in non-congestive and in chronic congestive glaucoma when the field is much contracted, and also in hæmorrhagic and in absolute glaucoma.

If one operation fails or is technically impossible or has been performed in the other eye with bad results, one of the others must be substituted (cf. § 843).

846. Non-Operative Treatment.—The measures employed in the non-operative treatment of glaucoma are: (a) miotics; (b) massage; (c) constitutional and symptomatic treatment.

(a) *Miotics* (eserine and pilocarpine) act powerfully to reduce increase of tension. The favorable effect of the miosis is attributed to two features. First, the iris being stretched in a radial way is drawn away from the wall of the eyeball to which it has been applied so that the angle of the chamber again becomes free; second, the absorbing surface of the iris is increased. Unfortunately the effect of the miotics upon the ocular tension is not lasting, inasmuch as it vanishes with the disappearance of the miosis. Moreover, when the angle of the chamber is permanently blocked, miotics are no longer of value; and naturally they are not when the iris is completely atrophic.

The mydriatics are as prejudicial in glaucoma as the miotics are useful.

(b) *Massage* (§123) is also an effective agent in temporarily reducing tension. It may be applied as needed by the patient himself three or four times daily and for periods ranging from one-half a minute to three or four minutes (Elliot). It may be followed with advantage by the application of hot compresses.

In acute congestive glaucoma, non-operative treatment is used mainly as a preliminary[6] to operation or as a temporary substitute for the latter when it is impossible to perform it at once. Such treatment ameliorates the pain and by diminishing the tension, lessening the cloudiness of the cornea and contracting the pupil, renders the operation easier to perform.

To produce such an effect it must be pushed vigorously. We put the patient to bed; instill eserine in ¼ to 1 per cent. solution (combined with 2 per cent. solution of cocaine) for three hours until the pupil contracts,

[6] There is some evidence to show that these drugs may affect the tension in some way other than by their miotic action. So too, the effect of atropine and like agents may not be altogether due to the mydriasis they cause.

then less frequently; apply leeches to the temple and hot fomentations to the eyes; administer bromides and morphine; give an active purge and in suitable cases, a hot bath.

Ziegler in acute, especially fulminating glaucoma, has had good results from the hypodermic injection of hyoscine (0.6 mg.) followed by an injection of pilocarpine (8 to 10 mg.) The former, he thinks, arrests a perverted lymph secretion, the latter stimulates a normal flow of lymph. After this combined treatment, eserine will sometimes act, when previously ineffective.

After an operation for glaucoma massage may be used to promote outflow.

Miotics and other non-operative measures have been used to control prodromal attacks of congestive glaucoma. This practice is not commendable, since it tends to put off the operation for which such attacks are an absolute indication.

In the more chronic forms of congestive glaucoma and particularly in non-congestive glaucoma, miotics and massage have been used in the belief that they will serve permanently to arrest the glaucomatous process. While it seems proved that they can check the latter for a time, it is doubtful whether they can be relied on to do this indefinitely except in a few cases only. The danger always is that in relying on these means we shall let the disease advance to the point where it is no longer remediable. The safe plan is not to undertake non-operative treatment unless the patient can be more or less continuously under observation and to discontinue such treatment as soon as the tension is no longer controlled, and more especially as soon as the field shows evidence of a continuing constriction. Even then it may be found that we have put the operation off too long.

When miotics are thus used to produce a continuous effect, they should be administered several times a day and particularly just before retiring at night, since then the tendency to a rise in tension is more marked; and they may be even given again in the early hours of the morning before the hour of rising (De Schweinitz). The dosage should be as low as possible to produce miosis without marked spasm of the muscles. Thus Posey advises beginning with a 0.02 per cent. solution of eserine and gradually increasing the strength to 0.6 per cent. Or pilocarpine may be used in double this dosage. It seems good usage to increase the dosage at the night instillation. This is especially true of eserine which is better borne then than in the daytime. The miotics may usefully be combined or alternated with massage and hot fomentations.

(c) *Constitutional treatment* is most important in all varieties and stages of glaucoma. As Elliot points out, every means should be taken to prevent loss of vaso-motor control and to eliminate the factors that make for the latter and the factors that may cause autogenous intoxication (overwork, worry, nervous and bodily exhaustion, improper diet, disturbance of endocrine secretion, all depressing conditions, including concealed sources of infection in the teeth, tonsils, nasal sinuses, intestinal tract, etc.)

II. Secondary Glaucoma

847. By secondary glaucoma we understand an increase of tension which appears in the course of other diseases of the eye, and as a consequence of them. Accordingly, the increase of tension here forms the complication of an already existing affection, although, all the same, it entails the results peculiar to itself, just as in the case of primary glaucoma. If it is associated with inflammatory symptoms, it produces in the cornea, the iris, etc., the changes belonging to congestive glaucoma. In other cases it manifests itself merely through the increase in tension perceptible to the touch, and also through the pressure excavation of the optic nerve with the disturbance of sight that is caused by it—namely, the contraction of the visual field and the diminution in central vision. Moreover, its termination in blindness and in degeneration of the eyeball is the same as in primary glaucoma.

848. Etiology.—The clinical picture of secondary glaucoma varies according to the disease which it accompanies. The affections of the eye leading to increase of tension are as follows:

1. *Ectasiæ of the cornea and the sclera.* Of the former, it is above all those connected with incarceration of the iris—i. e., the staphylomata—that almost universally lead to secondary glaucoma. It is only the exception that ectasiæ without incarceration of the iris, such as keratectasia ex ulcere or keratectasia after pannus or after keratitis parenchymatosa, give rise to increase of tension. Of scleral staphylomata, those which occur after rupture of the sclera may entail increase of tension, and so also may the ectasiæ that develop after scleritis. Most ectasiæ of the sclera, however, are the result, not the cause, of the increase in tension.

2. *Incarceration of the iris* in a cicatrix of the cornea or sclera, and also the partial apposition of the iris against the posterior surface of the cornea may lead to increase of tension even without any ectasia being present.

3. *Irido-cyclitis,* especially in those cases in which, except the deposits on the cornea, no exudates worth mentioning are found. The increase of tension in these cases is often only transitory.

4. *Seclusio pupillæ,* whether originating in adhesion of the entire pupillary margin to the capsule of the lens or in the inclusion of the former in a cicatrix of the cornea. Seclusio pupillæ leads to the accumulation of aqueous in the posterior chamber and to consequent protrusion of the iris, which is accompanied by increase of tension.

5. The *lens* becomes a cause of secondary glaucoma in two ways—by being luxated and by being swollen. All forms of luxation are of significance in this regard; but the most dangerous cases are those in which the lens is wedged into the pupil or lies wholly in the anterior chamber. Sudden swelling of the lens after injury or operation may likewise cause increase of tension, especially in the case of elderly people, whose scleræ are rigid. Increase of tension also occurs at times after cataract extraction and discission of secondary cataract.

6. *Intra-ocular tumors,* such as sarcomata and gliomata, in a certain stage of their development excite the symptoms of secondary glaucoma.

7. *Hœmorrhages into the retina* are the expression of changes in the vessels or disturbances of circulation, which sometimes lead to elevation of tension. This is most frequently the case in old people with arteriosclerosis; the elevation here usually makes its appearance under the form of congestive glaucoma—glaucoma hæmorrhagicum.

8. *Chorioiditis* and *myopia* of a high degree often give rise to an increase of tension under the form of a non-congestive glaucoma.

Glaucoma due to *autogenous toxœmia,* which probably sets up a low-grade inflammation blocking up the channels of outflow (§ 841), if not to be classed under secondary glaucoma, is at least akin to it (Elliot).

849. Factors Causing the Increased Tension.—These vary greatly according to the varying nature of the cases themselves, and very often we do not at all know how to account for the development of the increase of tension. In some cases secondary glaucoma is due to *perverted secretion*—i. e., to alterations in the character of the liquid poured into the eye. This is particularly the case in the form accompanying irido-cyclitis (Elliot). Other varieties of secondary glaucoma are due to *anatomical changes.* These are:

1. Obliteration of the sinus of the chamber due to the fact that the iris has been drawn forward and brought into contact with the cornea. This is probably the usual cause of increase of tension in the case of anterior synechiæ, by which the iris is drawn forward and brought close to the cornea, and the more so the more peripheral the site of the incarceration of the iris. The narrowing or the blocking of the sinus of the chamber in these cases does not extend all the way around as in genuine glaucoma, but is confined to the sector which corresponds to the anterior synechia (Fig. 269). Whether in this case increase of tension sets in or not depends upon whether the portion of the sinus that is still free is adequate for the excretion of the liquids of the eye. After an iridectomy the stump of iris which remains at the site of the coloboma may push itself into the wound, become united there, and to a corresponding extent block the sinus. After an extraction of the lens made with the iridectomy the incarceration of a tag of capsule may have the same result, the capsule jamming the iris stump against the corneo-sclera.

2. Pushing of the iris against the cornea by a greatly swollen or dislocated lens, by the vitreous when the lens is absent and the lens capsule ruptured, or by a tumor of the ciliary body.

3. Agglutination of the iris to the posterior surface of the cornea after the two have been for a long time kept in contact because of a corneal fistula.

4. Attachment of the periphery of the iris to the corneo-scleral junction because of inflammatory adhesion, as occurs in many cases, and especially in the chronic cases, of irido-cyclitis.

5. Blocking of the sinus of the chamber caused by infiltration of the ligamentum pectinatum, by cysts of the iris, by tumors of the iris, and in the case of aphakia by tags of vitreous extending into the anterior chamber that cannot pass the meshes of the ligamentum pectinatum.

6. Blocking of the sinus of the chamber by tissue which has remained there since fetal life. This is the case in aniridia, in which, the iris is never completely absent, but is simply reduced to a short stump, in the angle between which and the corneo-sclera the tissue above mentioned is situated. (For the conditions in buphthalmus see § 836).

7. Impermeability of the ligamentum pectinatum due to blocking of its open spaces by blood, leucocytes, or precipitates from the aqueous. It is changes of this sort that probably cause the increase of tension in recent cyclitis, also that due to the presence of many lens fragments or of fluid vitreous in the anterior chamber.

8. Impermeability of the ligamentum pectinatum due to the formation of an epithelial lining (anterior-chamber cyst—§ 635), e. g., after cataract operation.

9. Abolition of the communication between the posterior and anterior chambers in the case of an adhesion of the entire margin of the pupil to the lens or to the cornea (seclusio pupillæ). In luxation of the lens into the anterior chamber, a cut-off action of this sort may result because the iris is jammed against the posterior surface of the lens by the aqueous which has been secreted by the ciliary processes, so that the lens acts like a ball valve (Fig. 298). A similar effect is produced when after cataract extraction a laceration of the lens capsule allows a mass of vitreous to protrude through the pupil, and become adherent to its margin (Elliot).

10. Elevation of the pressure in the vitreous cavity produced by venous stasis in thrombosis of the retinal veins (glaucoma hæmorrhagicum) and in blocking of the venæ vorticosæ (by tumors, by endophlebitis, and also by experimental ligation).

11. Elevation of the pressure in the vitreous cavity due to increase in the volume of the vitreous; occurring suddenly in hæmorrhage or exudation (panophthalmitis), and gradually in tumors.

12. Elevation of the pressure in the vitreous cavity due to protrusion of the retina or chorioid into it as the result of hæmorrhage, exudation, or the formation of new growths in these membranes.

850. Treatment.—The treatment of secondary glaucoma must above all endeavor to remove the cause underlying the elevation of tension. For instance, in seclusio pupillæ the communication between the two chambers should be restored by means of an iridectomy, a dislocated or swollen lens should be removed, if possible, and so on. For the symptomatic treatment of the increase of tension itself, the use of miotics, dionin, cold compresses (in cases of a swollen lens), etc., may be considered according to the exigencies of the case. Of operations at our command, paracentesis of the cornea diminishes the pressure by letting out the aqueous, but does so only temporarily, so that it is suited simply to those cases in which the increase in tension is foreseen to be of short duration—e. g., in swelling of the lens and in irido-cyclitis. The paracentesis may be repeated several times, according to the demands of the case. A lasting elevation of tension can be combated only by iridectomy or sclerectomy. Glaucoma hæmorrhagicum gives the most unfavorable prognosis. Here we cannot count with certainty upon the effect of iridectomy, since sometimes it is immediately succeeded by blindness coming on rapidly and with great pain. Trephining affords better results. Eyes which contain a new growth, or which are both blind and painful, require enucleation.

Hypotony

851. Diminished Intra-Ocular Pressure (Hypotony).—This is found in very diverse affections of the eyeball. It is always a sign that the contents of the eyeball have diminished in volume.

Hence a high degree of diminution of tension is observed when, after the perforation of the eyeball, either the aqueous has flowed away or the lens or vitreous has escaped. This may be the result of an injury or of the spontaneous perforation of an ulcer. If the perforation in healing leaves a fistula or a cystoid cicatrix, through which aqueous continually escapes, the softness of the eye may persist for a long time (even many years). This also occurs sometimes after trephining. After the use of a bandage which has been too tightly applied, we find the eye softer for a short time, because, under the increased

pressure due to the bandage, an increase in the outflow of fluids from the eye has taken place. So, too, the eye becomes very soft when the volume of the vitreous is diminished by the shrinking of exudates—and hence, in those cases in which an atrophy of the eyeball is developing after irido-cyclitis. Hence progressive softening of the eyeball in the course of an irido-cyclitis is an ominous symptom. Slight degrees of decrease of tension accompany many cases of inflammation of the cornea, both suppurative and non-suppurative, and also occur not infrequently after slight injuries (erosions) of the cornea, especially if these were associated with a contusion. Of the affections of the deep parts, detachment of the retina is particularly associated with diminution of tension. Finally, slight degrees of the latter are found in paralysis of the sympathetic, and also after the instillation of cocaine, pilocarpine, and eserine (§ 80).

Cases occur which are known as *ophthalmomalacia*, or essential phthisis, in which diminution of tension appears spontaneously, without known cause. The eye suddenly becomes very soft, smaller, and injected, and not infrequently marked photophobia and neuralgic pain are associated with the condition. This state of things may last for hours or days, when it gives place to the normal condition. In many instances such attacks recur at intervals (intermitting ophthalmomalacia). The cause of this rare disease in many cases remains unknown; in other cases there has been an injury preceding it. The prognosis is good, as the ophthalmomalacia usually leaves no lasting ill results behind.

CHAPTER XXX

INJURIES OF THE EYE

852. Non-Penetrating Wounds; Contusions.—The injuries of the different parts of the eye have been described in previous chapters. The general aspects of such injuries, particularly when the latter are of graver character, and the economic significance attaching to them will receive a separate consideration here.

Non-penetrating wounds of the eyeball are necessarily confined to the conjunctiva, cornea, and sclera. These with wounds of the lids have received consideration in the preceding chapters, while injuries of the orbit will be described in Chapter XXXI.

Contusions of the eye and the adjoining parts are associated with changes varying in location and ranging from a trifling ecchymosis to the destructive injuries caused by rupture of the eyeball (§ 854). The following comprise the changes found:

In the *lids*, ecchymoses, emphysema, solutions of continuity.

In the *margin of the orbit*, fractures with or without displacement of the fragments.

In the *position of the eyeball*, exophthalmus which may be caused by the effusion of blood or air (emphysema) into the retrobulbar tissue or by the formation of an arterio-venous aneurysm due to rupture of the carotid into the cavernous sinus (§ 881). Enophthalmus may also occur (§ 865)

In the *tension* of the eyeball. This is greatly reduced when a perforation of the tunics of the eye has taken place. But the tension may be found to be temporarily diminished even apart from any gross material lesion, this alteration being then due to a decrease in the fluids of the eye and particularly of the vitreous (Leplat), which again must be regarded as a consequence of a disturbance of nutrition dependent upon nervous influence (cf. § 851).

In the *conjunctiva*, ecchymoses, lacerations (with or without coincident scleral rupture).

In the *cornea*, epithelial desquamation, and, later on, deep non-suppurative inflammations; rarely purulent processes or rupture of the cornea.

In the *aqueous chamber* and the *vitreous*, effusion of blood (hyphæma, hæmophthalmus).

In the *iris*, iridodialysis, partial or complete (aniridia traumatica), radial lacerations, recession of the iris, likewise paralysis of the iris (mydriasis) with or without paralysis of accommodation, irido-cyclitis, (which even in cases without perforation may go on to destruction of the eye—De Lapersonne).

In the *lens*, astigmatism, subluxation, and luxation, due to partial or complete laceration of the zonula, likewise the formation of cataract.

In the *sclera*, rupture in the anterior division, attended, it may be, with prolapse of the uvea, the lens, or the vitreous.

In the *chorioid* and *retina*, extravasations of blood, detachment, rupture; in the retina alone, cloudiness (commotio retinæ), formation of a hole in the fovea.

In the *optic nerve*, compression by effusion of blood, and contusion or rupture by fracture taking place in the optic canal; evulsion of the papilla from the scleral canal.

853. Perforating Injuries.—Every perforating injury is to be regarded as intrinsically serious because, given a perforation, there is a possibility of

a coincident infection of the interior of the eye; but this almost always leads to a severe form of inflammation that is very destructive to the eye.

Perforating injuries of the eye are very frequent in the working class and furnish a large contingent of the blind. This is particularly the case in regions where many industries are carried on. The following report of Cohn gives a good idea of the frequency of the injuries to which the eyes of many workmen are exposed: Among twelve hundred and eighty-three workers in metals employed in six factories, each man received on an average from two to three injuries in a year. Of course, the great majority of these injuries were of a slight character; most, indeed, consisted merely in the penetration of small metallic particles into the surface of the cornea, which were, for the most part, removed at the factory itself. About half of the workmen were compelled to seek medical aid; and out of every thousand, 28 suffered a partial impairment of sight, and 16 had lost one eye altogether. Every year 50 per cent of the stone workers in Aberdeen have to resort to treatment for eye injuries. From extended statistics it appears that in England and America eye injuries constitute something over 5 per cent of all injuries occurring in mines, quarries, factories, and workshops (Collis). The majority of these injuries are slight; yet even of these slight injuries many, especially in stone-masons and coal miners, give rise to dangerous infection and particularly to ulcus serpens (Shufflebotham, Collis). From 2.5 to 3.0 per cent of these industrial eye injuries result in blindness of one or both eyes (Collis).

The most important *symptoms of the presence of a perforation* are:

(*a*) Reduction of the intra-ocular tension. This symptom is particularly valuable in the case of small wounds in the sclera, which are concealed by the ecchymosed conjunctiva, and are hence not directly accessible to inspection. The diminution of tension, of course, lasts only as long as the wound is open. It must be borne in mind that a reduction of tension may also occur in injuries of the eye without perforation (see § 851).

(*b*) If the perforation enters the anterior chamber, the latter is shallower or altogether obliterated, as long as the wound remains open.

(*c*) In the case of somewhat larger wounds the prolapse of the subjacent structures gives evidence of the presence of a perforation. Most frequently it is the uvea which protrudes from the wound under the form of a darkly pigmented mass. According to the situation of the wound, the prolapsed portion belongs to the iris, to the ciliary body, or to the chorioid. If the uvea is ruptured, some vitreous is often found hanging out of the wound.

If vitreous has been prolapsed through a wound it gradually becomes clouded wherever it lies outside of the eye, so that it gets to look like a shred of mucus adhering to the region of the wound. It takes often several weeks before such a shred is finally cast off. Such a shred affords a ready pathway for infection to the interior of the eye.

Very frequently there are extravasations of the blood in the interior of the eye, although these often likewise occur in the case of non-perforating contusions. Blood extravasated into the anterior chamber generally sinks to the bottom of it soon after the occurrence of the injury, so that, like a hypopyon, it fills up the lowermost part of the chamber and is bounded above by a horizontal line (*hyphœma*). Blood in the vitreous (*hœmophthalmus*) often makes itself evident by a reddish reflex from the pupil. (With large masses of blood the interior looks black with the ophthalmoscope).

854. Varieties of Perforating Injuries; Ruptures of the Eyeball.— Perforating wounds are distinguished into incised, punctured, and lacerated

wounds. To the two former belong, besides wounds produced by operation, those caused by the penetration of sharp-pointed or sharp-edged foreign bodies. Lacerated wounds are most frequently the consequence of a *rupture* of the eyeball. This is produced by the action of a blunt instrument upon the eye (contusion)—e. g., by a blow with the fist or with a cane, by a stone, by the impact of large flying fragments of wood or metal, by a thrust from a cow's horn (not infrequent among farmers), by striking the eye against a projecting corner, etc. Blows of this sort caused by slow moving objects cause a rupture of the sclera which is generally quite

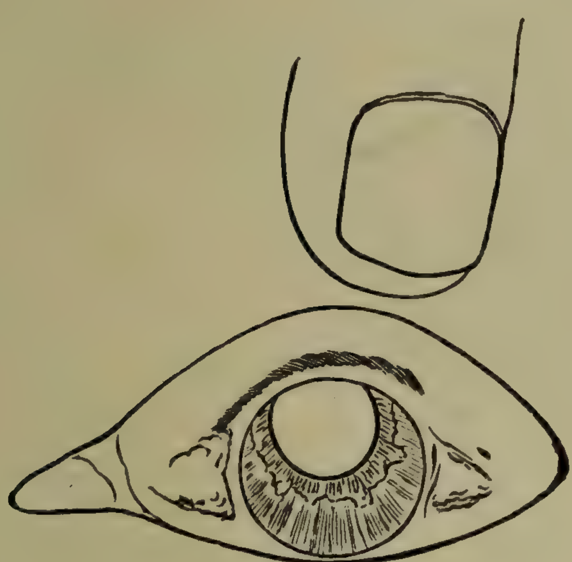

Fig. 378.

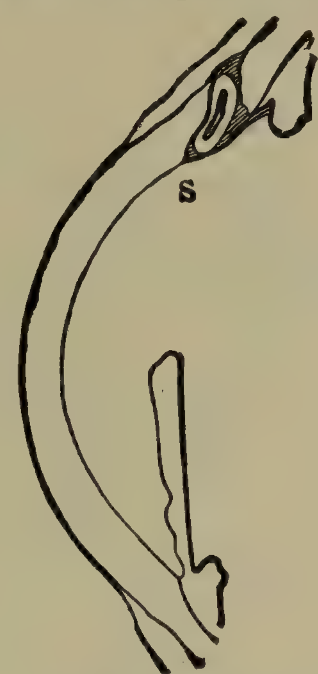

Fig. 379.

Fig. 378.—Rupture of the Sclera. Injury produced by the eye being hit with a stone thirty years before. Rupture healed with retention of the visual power. The site of the rupture can be made out along the convex gray line (scar) which runs concentric with the upper margin of the cornea. The cornea is clear, the anterior chamber rather deeper than normal. The pupil is displaced bodily upward (the lower margin of the pupil lies approximately behind the centre of the cornea) and extends to the upper margin of the cornea. The lesser circle of the iris can be followed on both sides of the coloboma, to a point behind the limbus. On both sides of the cornea is a pinguecula.

Fig. 379.—Same Case in Section. Magnified 3 × 1. The rupture runs from Schlemm's canal, *s*, obliquely upward so that that portion of the scar which is visible externally lies in the sclera 2 mm. back of the limbus. The lips of the wound are separated; between them lies scar tissue, and imbedded in this is the iris. The latter is torn away from its insertion in the ciliary body and is rolled up; the section has happened to strike it in such a way that the iris here looks like a closed ring, on the inner circumference of which the retinal pigment layer is visible. The ciliary body is somewhat lacerated at its insertion and the scar tissue extends into the chink that is thus produced.

long, is curved, and lies near the margin, especially the upper and inner margin of the cornea and concentric with it (Fig. 378).

Ruptures caused by rapidly flying missiles, which graze but do not penetrate the eye, occur on the side opposite the point of impact (Lister). A projectile of some size entering the eye from in front causes radial ruptures splitting the eye into petal-like lobes. As a rule, in rupture of the eyeball, the process does not stop at laceration of the sclera, but a part of the contents of the eye, most generally the lens, is expelled, and the vitreous is sometimes seen hanging out of the wound. The iris, at a point corresponding to the site of the rupture, appears to be wanting, and the eye often looks as if the portion of the iris in question had been removed by iridectomy (see § 725). Frequently the conjunctiva over the lacerated sclera, thanks to its great extensibility, remains uninjured. In this case the extruded lens is sometimes found lying beneath the conjunctiva (Fig. 383).

In *rupture of the sclera* the blow may act directly upon the eye itself or a blunt body —e. g., the tip of a cow's horn—may enter between the eyeball and one wall of the orbit and squeeze the eye against the opposite wall.

We distinguish between direct and indirect rupture. The former is the kind that originates at the point of impact of the foreign body by which the blow is inflicted; the

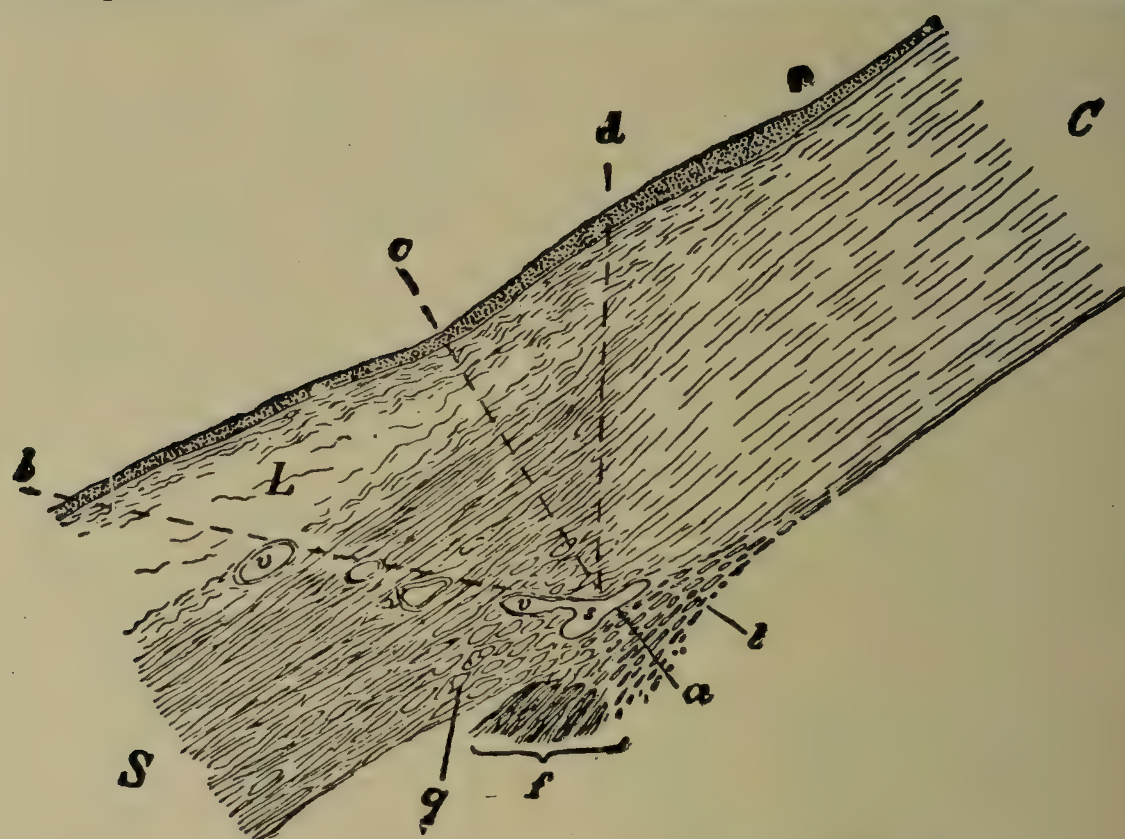

FIG. 380.—COURSE OF A SCLERAL RUPTURE. Magnified 22 × 1.

C, cornea; *S*, sclera; *r*, end of Bowman's membrane, and beginning of the limbus *L*. *f*, anterior end of the longitudinal fibres of the ciliary muscle, which pass over into the lamellæ of the ligamentum pectinatum, *l*. The capsule of the eyeball is less firm at the corneo-scleral margin, first, because here a larger number of the fibres of the sclera (those namely which in the figure are seen in cross section at *q*) take a circular course; second, because in front of these fibres Schlemm's canal, *s*, is imbedded in the scleral tissue, which consequently has its inner layers interrupted at this spot. From Schlemm's canal to a point right beneath the conjunctiva, the anterior ciliary veins, *v*, *v*, run continuously through the sclera. Most scleral ruptures follow these cavities in the direction, *ab*, so that the external orifice of the rupture lies some millimetres behind the limbus. More rarely the ruptures of the sclera pass perpendicularly in the direction *ac*, or even run obliquely forward in the direction *ad*. Ruptures of the latter sort have usually but little length so that nothing but the iris prolapses and this only to a slight extent (Fig. 381), and the rupture itself does not become visible until the iris is excised (Fig. 382). These small ruptures almost always afford a good prognosis.

FIG. 381.—SMALL SCLERAL RUPTURE SITUATED IN THE LIMBUS. Magnified 2 × 1. The iris has prolapsed through the rupture and hence the pupil is displaced in the direction of the latter. The iris at the opposite side is broadened, and hence the contraction furrows are separated from each other, as comparison with Fig. 382 shows.

FIG. 382.—THE SAME CASE AFTER EXCISION OF THE IRIS. Magnified 2 × 1. The short rupture which is situated in the limbus has now become visible. *a*, a_1, the angles of the sphincter which are now properly placed.

latter, that which occurs at a spot remote from the point of impact. In *indirect* ruptures the only efficient factor is the compression of the eyeball. In *direct* ruptures, there is added to this compression a local indentation of the eyeball, by which the situation of the rupture is determined. This situation, therefore, varies greatly and often is not in the sclera but in the cornea. When compression is the determining factor it puts the contents of the eye suddenly in a condition of increased tension which acts

on the capsule of the eyeball like an explosive force working from within outward. The fact that ruptures are so frequently situated above the cornea is attributed by some to the deficient support that the eye receives here, by others to the less resistant character of the sclera in this spot (Fig. 380), and the fact that most scleral ruptures start from a point situated above and inward is ascribed to the pressure of the trochlea which rips open the eye that is forced against it (Müller).

Sometimes *very small* ruptures occur right in the limbus itself or even in the transparent cornea (Figs. 380, *ac* and *ad*). In these, owing to the close attachment of the conjunctiva, the latter is necessarily ruptured and the iris almost always prolapses. The prognosis of these small ruptures is good, provided the prolapsing iris is excised. They occur comparatively often in young people (even in children), while the more extensive ruptures occur in the old.

Very rarely we meet with *incomplete* rupture of the sclera. We then find the conjunctiva and sclera ecchymosed and swollen, and a few days after the injury a bluish black line appears near the limbus and concentric with it. This dark area may become ectatic later, and increase of tension may set in. There may be displacement of the pupil.

Ruptures of the sclera are generally such serious injuries, because a force that is strong enough to break an eye open always causes *lesions elsewhere,* in the interior of the eye. The iris (Fig. 379 and 383, *b*) is almost always torn away from its insertion at a point corresponding to the extent of the scleral rupture (iridodialysis), and is either incarcerated in the wound or is extruded through the latter beneath the conjunctiva, to which it becomes adherent. The eye then presents a coloboma over an area corresponding to the rip in the sclera. The portion of the iris remaining in the eye is commonly found to be considerably retracted (Fig. 383, lower part). The anterior chamber is consequently unusually deep, and particularly so if, in addition, the lens and a part of the vitreous have been expelled. In many cases a piece of the iris, or even the entire iris, is torn altogether out of the eye. In only a very few cases does the lens remain in position and in place. Usually it is either expelled entirely from the eye, or it remains lying beneath the conjunctiva, provided the latter is unruptured (Fig. 383, *f*). The vitreous is often densely permeated with blood; and laceration or hæmorrhagic detachment of the retina or chorioid may be present. Since to the severity of such lesions there is added the

Fig. 383.—Rupture of the Sclera and Luxation of the Lens Beneath the Conjunctiva. Magnified 3 × 1.

Vertical section through an eyeball which had been injured by a calf's horn. Since seven weeks later symptoms of sympathetic inflammation of the other eye set in, the eye was enucleated. The rupture in the sclera lies close to the upper margin of the cornea, not quite one millimetre behind the corneo-scleral junction, so that the lower lip of the wound contains a narrow rim of sclera. The tear in the sclera gapes to the extent of nearly a millimetre, and is filled with a delicate cicatricial tissue, *a*, which extends back from this point into the interior of the eye, passing between the ciliary body, *e*, and the iris, *b*. The latter at a point corresponding to the scleral rupture is torn off from the ciliary body (iridodialysis) and is rolled into a ball just as in Fig. 379. The ciliary body, *c*, likewise greatly altered, is found behind the upper edge of the rupture. It is continued into the chorioid, *d*, which represents marked inflammatory infiltration about the large blood-vessels (inflammation of the kind that gives rise to sympathetic trouble). The retina, *e*, is detached up to the ora serrata, and at the latter point is torn loose and is much puckered. Below the scleral rupture is seen the cornea in section, compressed vertically and as a result of the compression puckered on its posterior surface. At the lower portion of the eyeball the ciliary body is swollen, and the iris is carried backward so as to be in part applied to the surface of the ciliary body. The detachment of the chorioid, d_1, and of the retina, e_1, seen here in the cut, did not exist during life, but is a result of the way the specimen was made. The scleral rupture is covered in front by a mass which from its concentric striation is recognized to be the lens. The conjunctiva, *f*, overlying the lens, is detached by it from the sclera as far as the limbus, *g*. *h*, remains of the conjunctiva at the lower margin of the cornea.

danger of subsequent infection of the wound, it can be readily understood that most eyes which have suffered a rupture of the sclera undergo destruction. It is an exception, in fact, for such an injury to recover with the retention of serviceable vision. A farmer once presented himself at my clinic who had been gored first in one eye and then, some years afterward, in the other also, by a cow's horn. In both eyes there was a healed rupture of the sclera on the inner side, with what looked like a well-made coloboma of the iris. Both lenses were absent, but the fundus was healthy and with cataract glasses the sight was very good. This man, therefore, may be said to have had a double extraction performed by the cow, and that, too, with more success than many operators are accustomed to have with their operations.

Ruptures of the cornea are much less frequent than ruptures of the sclera. They are usually direct, and for this reason, unlike scleral ruptures, follow no rule as to their site or direction. In most cases the ruptures are nearly rectilinear, but sometimes they look jagged and are flap-shaped.

855. Results of Perforating Injury.—The course after a perforating injury varies according as a foreign body has remained in the eye or not. If there is no foreign body lodged in the eye, the only thing to be considered is the healing of the wound itself; and in regard to this the main thing to determine is whether the injury has been followed by *infection* of the wound or not, as upon this depends the question whether healing takes place with or without inflammation.

(*a*) *Healing without inflammation* is to be expected only in the case of clean, non-infected wounds. The most favorable conditions are presented by clean incised or punctured wounds, such as, for instance, are made by operations, where at the same time care is taken that no foreign tissue, like uvea or vitreous, is introduced into the wound. But even if this latter should be the case, healing without inflammation is still possible, although the edges of the wound can not unite with each other directly, because they are not in contact. The tissue consisting of uvea or vitreous introduced between them is gradually converted into cicatricial tissue, and thus takes part in the definitive closure of the wound. It then, to be sure, always remains attached to the cicatrix, a thing which later on often entails evil consequences. In this way—i. e., by the interposition of a cicatricial tissue taking place between the edges of the wound—even lacerated wounds, such as develop, for example, after rupture of the sclera, may, if the case runs a fortunate course, heal, and heal without inflammation so that the eye remains serviceable for vision.

(*b*) Perforating wounds are followed by violent *inflammation* whenever infection of the wound or of the interior of the eye has occurred. Infection takes place either by means of the very body which causes the injury, it being covered with dirt and thus conveying infectious germs, or secondarily from the fact that the opening in the envelope of the eyeball affords a point of entrance for germs, especially from the conjunctival sac. The interior of the eye is uncommonly susceptible of infection, since it obviously affords a good medium for the cultivation of germs of various kinds. The inflammatory process mainly affects the uvea (see §§ 696 et seq. and 752-755). In the acutest cases there is a purulent inflammation of the latter, which leads to the formation of abscess in the vitreous or to an actual sup-

puration of the whole eye (panophthalmitis). In the less violent cases there is a plastic irido-cyclitis—i. e., the inflammation of the iris and ciliary body produces an exudate which subsequently becomes organized into a false membrane. In this case, too, the eye is generally lost, although it is not destroyed in such a violent fashion as in panophthalmitis, but by a process of protracted inflammation. The exudates which undergo organization shrink, and thus gradually diminish the size of the eyeball (atrophy of the eyeball). This outcome of an injury is even more dangerous for the patient than panophthalmitis, since in the former case sympathetic inflammation of the other eye very frequently sets in, which is not the case in panophthalmitis.

856. Foreign Bodies in the Eye.—The presence of a foreign body in the interior of the eye converts every injury, be it ever so insignificant otherwise, into a serious lesion, which, in most cases, entails the destruction of the eye. Hence in every injury attended with perforation we must at once propound the query whether or not there is a foreign body left in the eye. In most cases the history of the case itself supplies points important for the determination of this fact. If, for instance, a person has run a pair of scissors into his eye, we would naturally suppose that there was no foreign body there; conversely, in the case of a man who has had a perforating injury of the eye produced by the explosion of a percussion cap or while he was hammering iron, the presence of a foreign body in the eye is extremely probable. The character of the foreign bodies in question varies exceedingly. Most commonly we have to do with fine splinters, the points and sharp edges of which enable them to penetrate the sclera. In this category belong chiefly splinters of metal, splinters of glass, and fragments of stone—less commonly slivers of wood, etc. The foreign body may be situated in any part of the eye; indeed, if it has sufficient projectile force, it may even, after traversing the entire eyeball, perforate the sclera a second time on the opposite side, and penetrate into the tissue of the orbit. The precise determination of the place in which a foreign body is located within the eye is generally attended with great difficulties. As a rule, it is only during the time immediately succeeding the injury that it is possible to see the foreign body directly, although even then inspection of the interior of the eye is often rendered impossible by the presence of hæmorrhages. Subsequently, the difficulty of this inspection is still further heightened by the cloudiness which soon develops in the media and by the exudates which envelop the foreign body and render it unrecognizable. If we are dealing with metallic fragments of not too small dimensions, we usually can make them out and localize them by means of the x-rays; and for chips of iron we may also employ a magnet. If these means are unavailing, we are often driven to conjectures with regard to the location of the foreign body—conjectures based upon the direction pursued by the body in its flight, the situation of the aperture by which it entered, the sensitiveness of certain portions of the eye to touch, the presence of a circumscribed obscuration (scotoma) in the field of vision, etc.

857. X-ray Localization.—The localization of foreign bodies in the eye by means of the x-rays has reached a high degree of accuracy, thanks to the instruments devised by Sweet, Mackenzie Davidson, Dixon, and others. In these instruments a marker or pair of markers placed close to the eye is used. Skiagrams of the eye are taken from two

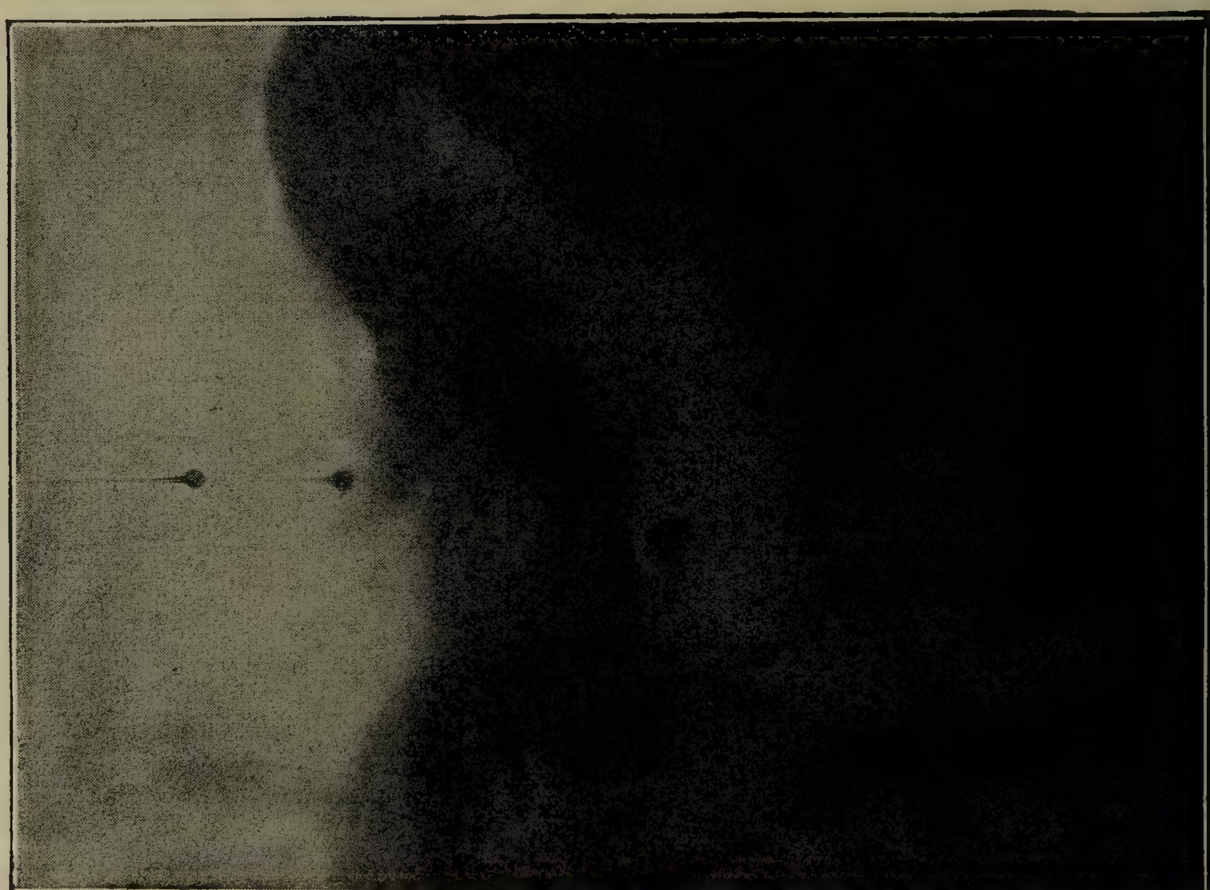

FIG. 384.—SKIAGRAM OF FOREIGN BODY IN EYE. (After De Schweinitz and Randall.)

The picture is taken with the x-ray tube placed in the horizontal plane passing through the two markers. The shadow of the foreign body and of the two markers is seen on the plate. Another plate taken with the tube in a different position would show the shadows in a different relation. A comparison of the two plates enables us to determine the position of the foreign body.

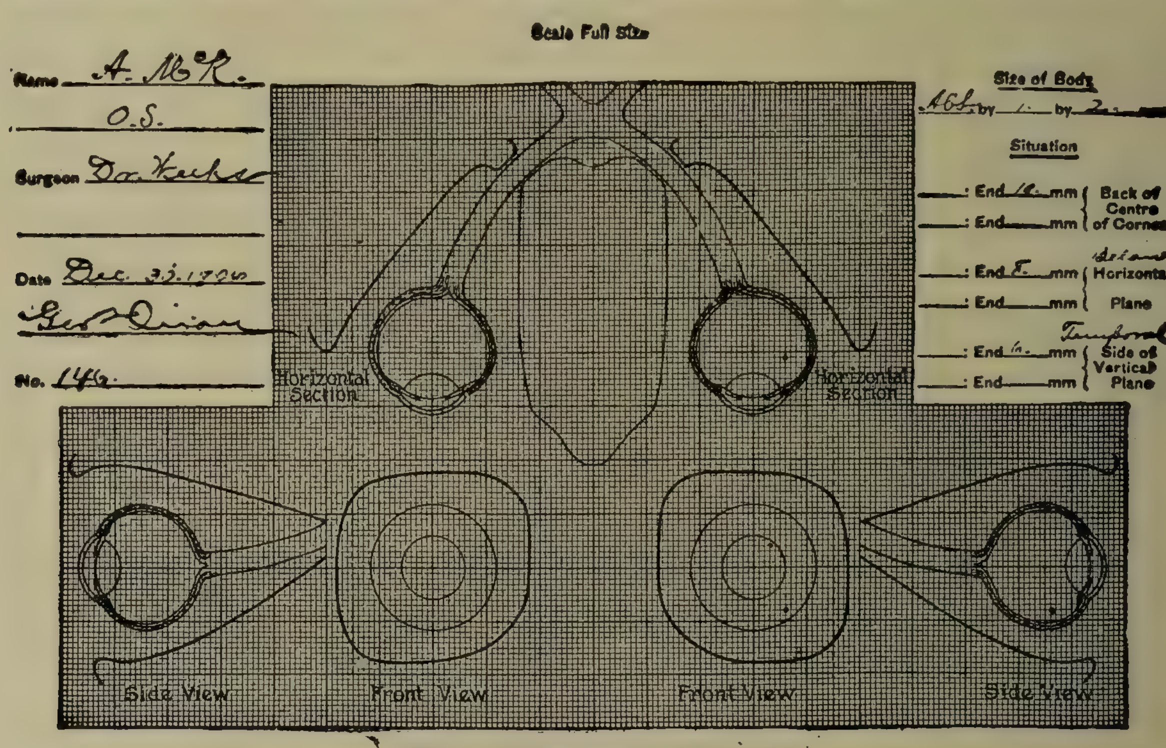

FIG. 385.—CHART FOR PLOTTING LOCATION OF FOREIGN BODIES IN EYE. (After Weeks.)

The chart modified from the one devised by Sweet is divided into mm. squares in order to facilitate measurement and plotting. The three views (front, side, and horizontal) enable the location of the foreign body to be indicated precisely.

different positions. The differing relations existing on the two plates between the shadow of the foreign body and of the markers (see Fig. 384) determine the actual position of the foreign body with relation to the markers and therefore its actual position in the eye. This position can then be plotted on a diagram (Fig. 385).

The presence of iron or steel in the eye is shown by the *magnet* (Fig. 451). If, when this is applied to the eye there is bulging of the iris or pain, iron or some other magnetic metal must be present.

858. Inflammation Produced by Foreign Bodies.—It is only in rare instances that a foreign body is tolerated for any length of time in the eye without setting up inflammation, the body itself either remaining free or becoming encapsulated in an organized exudate. But even such eyes as these are by no means secure from a sudden outbreak of inflammation—occurring sometimes years afterward—which causes their destruction. In the great majority of cases the inflammation follows close upon the heels of the injury. Such an inflammation is either a panophthalmitis, an abscess in the vitreous, or a plastic irido-cyclitis, just as in the case of simple perforating injuries.

Leber has determined, by a series of experiments upon animals, the reason why the presence of a foreign body in the eye regularly results in a severe inflammation. This inflammation is either excited by the presence of micro-organisms which make their way into the interior of the eye from the conjunctival sac, along with the foreign body or subsequently, or it is the consequence of a chemical irritation of the tissues produced by those foreign bodies which are not chemically indifferent. For example, purulent inflammation could be produced by bits of copper, and still more by particles of mercury, which were introduced aseptically into the anterior chamber. Hence, such inflammation does not necessarily presuppose the entrance of schizomycetes into the eye. Observations upon man agree in general with the facts obtained by experiment. Whether a body which penetrates into the eye is well borne by it or not depends upon the following circumstances: 1. First of all, upon the fact of its being *aseptic* or not. Small fragments of metal are commonly aseptic, if they fly off when the metal is being hammered or turned, because they are then strongly heated and thus sterilized. Large metal fragments are usually not heated to the point at which they become perfectly sterile, and hence give a bad prognosis even if they are removed very soon from the eye. 2. Upon its *chemical* character. Chemically indifferent bodies (such as, for instance fragments of glass), if they get into the eye aseptically, are the ones most likely to remain there without producing any further ill effect. The contrary is true of the foreign bodies that are of most frequent occurrence—i.e., chips of metal. These almost always set up a severe inflammation, even when they are sterile, because they gradually dissolve in the tissues of the eye and become oxidized there, so as to act as chemical irritants. This is the case with iron, and still more with copper; metals which, like lead and the noble metals, are innocent in this regard, are of comparatively rare occurrence in the eye. 3. The *tolerance* of the separate tissues of the eye toward foreign bodies varies. The uvea, and especially the iris and ciliary body, exhibit the greatest reaction to injury of any kind. The lens, on the contrary, possibly on account of the sluggishness of its tissue metamorphosis, is the part of the eye in which foreign bodies are relatively the best borne. If, for example, a small chip of iron has become imbedded in the lens the latter, to be sure, becomes clouded, but inflammation ordinarily fails to take place. In such a case sometimes the lens is afterward colored brown by the oxide which is formed. A deep brown coloration is produced, mainly under the guise of rust-colored dots which lie beneath the anterior capsule of the lens and form a crown, corresponding nearly in situation to the margin of the pupil when dilated (Samelsohn). Later the coloration may extend to the iris, which, if previously gray or blue, assumes a rusty brown hue. This impregnation with iron also occurs in the other tissues of the eye, particularly in the retina, which may in consequence become atrophic, so that blindness may ultimately ensue even when the fragment is tolerated without inflammation (Hippel, Jr.). The impregnation of the tissues with iron is called *siderosis bulbi* (from σίδηρος iron). Siderosis is rare. It may also come from unabsorbed hæmorrhage (Kipp, Clegg). The condition

may develop as early as two months after the injury (Natanson), but usually not till much later. The staining of the tissues may clear after removal of the foreign body (Clegg).

Small fragments of iron may in time be entirely dissolved by oxidation.

The foreign bodies causing a destructive irido-cyclitis may be very minute. This was often seen in the war, when loss of the eye was produced by very fine fragments entering the eye in explosive injuries (De Lapersonne).

859. Prognosis of Perforating Wounds.—This is deducible from the exposition given above. In every case it is grave, for even the minutest prick made with a fine needle may induce suppuration of the eyeball, if the needle was contaminated with septic substances. As we do not, for the most part, know whether the body causing the injury was aseptic or not, and as the consequences of an infection of the wound do not set in until several days have elapsed, we must be very cautious in stating the prognosis during the first few days after the injury. In general, the nature of the wound and the facts as to the presence of a foreign body in the eye serve to determine the prognosis. With regard to the former, we must take into consideration the situation and extent of the wound, and the condition of its edges; also whether or not the inner tunics of the eye have prolapsed into the wound, and how much, if any, vitreous has escaped. Large wounds with extensive prolapse of the inner tunics of the eye are always followed by inflammation and by shriveling of the eyeball. The question in regard to foreign bodies is often difficult to decide. If there is a foreign body in the eye and it cannot be removed at once, the eye is almost always lost. In some cases, it is true, a foreign body is carried in the eye for years without creating trouble. But in comparison with the frequency of perforating injuries these cases are infinitesimally few, and even in these cases the eye is never safe, since inflammation may be lighted up in it at any time by a foreign body which has remained thus apparently innocuous. Again, in stating the prognosis, the danger which threatens the other eye, because of sympathetic inflammation, must not be forgotten.

Wounds involving the *ciliary region* are held to be particularly dangerous both to the affected eye and to its fellow, since they often cause a plastic irido-cyclitis in the one and a sympathetic inflammation in the other.

The unfavorable prognosis which perforating wounds of the sclera generally offer holds good, at least in part, even for those cases in which, to begin with, there is a smooth healing of the wound. Such eyes, which often recover from the injury with the restoration of good sight, nevertheless not rarely become blind afterward because of secondary changes, which are the consequences of the cicatrix in the sclera. If the uvea is incorporated in the scar, this may give rise to inflammation or increase of tension. In scleral wounds which lie farther back, in the region of the retina, the latter may become attached to the cicatrix. By subsequent contraction of the cicatricial tissue the retina is drawn more and more into the cicatrix, and thus is loosened from its bed; the eye grows blind from detachment of the retina (Von Graefe). In this way, too, many eyes are destroyed which have been operated upon with apparently brilliant success by section of the sclera, as for the extraction of a foreign body or of a cysticercus. A further danger accrues to the eyes from the fact that the scleral cicatrices later on readily become ectatic, and lead to the formation of scleral staphylomata and also to increase of tension.

860. Treatment.—In consideration of the great frequency of eye injuries (§ 853), every attempt should be made to *prevent* them, especially by the use of suitable eye guards (§ 115).

When we get a recent perforating wound to treat, which is so extensive that we have no prospect of retaining the eye in a serviceable condition, we advise the patient to have enucleation done at once (*primary enucleation*). By this he is saved from a protracted illness as well as from sympathetic disease of the other eye.

If the injury is such that there is a *prospect of retaining an eye* that will be able to see and there is no indication of the presence of a foreign body in the interior of the eye, we cleanse the wound from any adherent dirt with moistened pledgets of cotton. If the iris has prolapsed into the wound, which can be the case only in wounds situated in the cornea or in the most anterior portion of the sclera (Figs. 381, 382), it must be carefully excised. But if the ciliary body or chorioid projects into the wound, they should not be removed, because, if they are, the vitreous would prolapse. Small wounds soon close of themselves by cicatrization; large gaping wounds should be united by sutures passed either through the edges of the sclera itself (only, however, through its superficial layers) or, better, through the conjunctiva overlying it. They may also be covered in with conjunctival flaps (§ 917). After we have thus treated the wound we instil atropine into the eye, put the patient to bed, and apply a bandage or, if there are signs of beginning inflammation, apply iced compresses. These should be used without intermission. If it seems likely that the wound has been contaminated with dirt or other agent likely to carry tetanus germs, injections of tetanus antitoxin should be given. Secondary infection may be averted best by the use of Dakin's solution or a solution of chloramine (De Lapersonne). Occasionally, after an infection has begun, the use of a suitable vaccine or of diphtheria antitoxin succeeds in aborting it.

If in spite of the treatment the inflammation progresses, we do an enucleation (*secondary enucleation*), as soon as we see that the eye is lost so far as sight is concerned. How do we recognize this? The future of the eye depends not so much on the changes taking place in the anterior chamber as upon the exudation in the vitreous cavity, which leads to blindness and subsequently to shriveling of the eye. But exudation in the vitreous is just the thing that we cannot see directly, but can only infer, and we infer it from two facts, viz.: (1) Steadily diminishing function of the retina—this, because the latter is always implicated when there is exudation in the vitreous. In this case on repeatedly testing the light perception and the visual field we shall find both suffering greater and greater impairment until finally the eye has lost all perception of light. (2) The gradually increasing softness of the eye, due to the shrinking of the vitreous exudate. When, therefore, these two symptoms have made their appearance the time for enucleation has arrived, and we should not delay too long about it either (not more than two or three weeks from the date of the injury), since otherwise we might be caught unawares by an outbreak of sympathetic inflammation in the other eye.

If there is a *foreign body* in the eye, we must in the very first place endeavor to remove it either by instruments introduced into the eye or by a magnet (see §§ 940 and 978). For this it is requisite to know, at least approximately, the situation of the foreign body (except in the case of fragments of iron, which the magnet withdraws even from an unknown depth).

If there is no prospect of our being able to remove the foreign body, we may, as a matter of experiment, wait a while to see if possibly it will be tolerated by the eye without inflammation. This is especially apt to be the case when a foreign body is imbedded in the lens; the latter becomes cloudy, and consequently, when all the inflammatory symptoms have passed off, can be removed by a cataract operation along with the foreign body contained in it. As soon as plastic irido-cyclitis has set in, it is advisable not to make any more attempts at removing the foreign body; absolutely the only indication in this case is enucleation.

The removal of a foreign body is often fraught with difficulty and may be associated with so much traumatism that the eye is lost at once. If not, it may be lost at a variable time later through detachment of the retina, irido-cyclitis, or panophthalmitis. But many eyes retain their sight permanently or for a long time. The translator has seen a case in which, except for a traumatic cataract, the eye was normal 26 years after the extraction of a piece of iron from the retina.

861. Compensation for Injuries.—When the sight is permanently impaired as the result of an injury the injured man suffers an impairment of his earning capacity. In most countries, a compensation has been fixed for such cases by the laws regulating accident insurance whenever the injury has happened in an industry in which insurance is obligatory. In order to determine the amount of indemnity the opinion of an expert physician is required. Such a physician has to decide: 1. Whether the disabilities alleged to exist by the injured man are actually present or not, and which of them are to be ascribed to the accident. 2. What degree of disability the person in question has suffered in consequence of them.

To decide the first point we must first determine by precise examination all the changes that are objectively perceptible, and then examine the function of the eye by the visual tests. If objective changes can be proved to exist, it is then to be determined which of them were produced by the alleged injury. It is generally easy to do this if one sees the injured man soon after the accident; if for instance, the physician who has to give the opinion is the same as the one who treated the injured man right after the accident. But very frequently the physician has to give an opinion on a case which he sees for the first time months after the injury. Then he must ask himself whether the changes present did not exist before the accident or, in case they followed it, whether they were not perhaps produced by disease which may have chanced to set in soon after the accident, but was independent of it. The *history* is often of no service; on the one hand, we must be prepared for statements which are intentionally wrong; on the other hand, the patients often act in good fath when they erroneously refer their eye disease to a preceding injury. Thus the entrance of dust into the eye is alleged as a cause of the most various eye diseases, even when the changes are old but had not been noticed by the patient until the occasion of his getting the dust in. The patient rubs his eye when the dust enters it and then discovers that when he closes one eye he sees little or nothing with the other. This, he thinks, has just occurred at the time and hence he regards it as the

direct result of the slight accident. Once a young girl came to Fuchs's clinic on account of some external affection of the eyes. The eyes were examined with the ophthalmoscope and in one of them was discovered an extensive old chorioiditis. When the tests of vision, which were thereupon made, showed that this eye was almost blind, the girl could not be dissuaded from believing that the blindness had developed as the result of the ophthalmoscopic examination.

It may also happen, on the contrary, that the disease has been produced by an accident of which the patient has been unaware. This happens especially in industries in which dust or small foreign bodies fly into the workmen's eyes so often that they no longer take any account of them. But the consequence of such an inconsiderable injury may be an ulcer; or a traumatic cataract may develop long after a small metallic particle has penetrated into the eye, and that, without any notice having been taken of it.

Although the patient's testimony is thus likely to be unreliable, yet if we put our questions carefully enough we can often gain from it valuable information—information which may be quite different from that which the patient intends to convey. Often some unwitting statement indicates which way the truth lies. Moreover, from the patient's statements we can usually get an impression of his mental attitude and of his truthfulness, accuracy, and good faith.

We next turn to the *objective findings*, from which we conclude, first, whether the changes present are such as could be produced by an injury at all, and second, whether the alleged accident was just the sort of one that would be apt to produce such changes. Scars in the cornea produced by incised wounds look different from those caused by ulcers; the results of a contusion of the eye (rupture of the sclera, iridodialysis, luxation of the lens, etc.), again, are different from those of incised or punctured wounds. However, it is not to be forgotten that the accident may also entail indirect consequences which produce objective changes quite different from those produced by the injury itself, as, for instance, when an ulcus serpens develops from an erosion of the cornea.

If we find changes which may have developed either as the result of injury or spontaneously, we shall in most cases be able to judge about how long these changes have existed, and from this decide whether they are to be brought into relation with the accident, which has occurred at a determinate time in the past. Thus, old scars of the cornea look different from recent ones; if an eye has been affected with diminution of its visual power for quite a long while it often squints, etc.

Often not only careful examination but also great experience is required in order to judge accurately regarding changes when they are rather old, and this is still more so when we are dealing with those cases in which *no objective changes* can be discovered at all. These cases are divided into two groups. To the first group belong those cases in which anatomical changes are actually present but escape detection. Such changes may be so minute that they are not demonstrable with our present appliances, for example, in the case of an amblyopia produced by a lesion of the fovea due to dazzling from strong light or produced by a small hæmorrhage which has already become absorbed. Or, the changes may lie behind the eye, for example, in the optic tracts as the result of fracture of the base of the skull. Careful examination of the function and prolonged observation of the case will often suffice to clear the matter up. But the real test of an expert are those cases in which it is a question of purely *functional disorders*. Such are the cases in which the function of the eyes is affected simply by shock, or the psychic commotion that accompanies the injury. These cases are known by the name of *traumatic neurosis*, and, so far as their essential nature is concerned, belong to hysteria; hence the disturbances of sight that are present must be characterized as hysterical amblyopia and hysterical asthenopia, produced by an injury. The symptoms of these conditions are described in §245. If we find them in a workman who has been injured only a little while and has not yet had any medical examination of his eyes, we shall be able to make the above diagnosis. But it is otherwise when we have to do with patients, especially those who have been through a railroad accident, who make claims for big damages and have already been examined a number of times, particularly so if they belong to the cultivated classes. Then there is the danger that the symptoms have been suggested to the patients by the repeated examinations or that they become more and

more conversant with the symptoms, about which they have been questioned, and then intentionally simulate them. Since in default of objective finding we are altogether dependent on the patient's statements, it is in many cases absolutely impossible to detect a clever simulant. Cases of this sort are constantly accumulating and require the greatest precaution.

862. The second thing that the expert has to do is to determine the disability that the injured man has incurred. For this purpose the function of both eyes must be carefully determined by visual tests. In doing this we must have in mind the possibility of exaggeration. With some experience it is not difficult to estimate the degree of visual disturbance that ought to correspond to the objective changes.

Both eyes may have been injured by the accident, or in case only one was affected by it, the other may have been already bad. In both cases the total visual power is diminished, and it will be easy to determine for what kind of work the visual power that is left should still about suffice. Much more difficult is it to gauge the working capacity when one eye has been rendered blind but the other is normal. Then the total visual power is also normal, but the special advantages that belong to *binocular vision,* namely the binocular field of vision and binocular perception of depth, are lost. The monocular field of vision is smaller than the binocular because it does not extend so far toward the side of the blind eye (see Fig. 114). But this will be found to be troublesome in only a few callings, and especially so since the patient by turning the head to the blind side and by abduction of the seeing eye soon learns how to get rid of the limitation in the visual field produced by the nose. Of more importance is the loss of binocular vision. A slight diminution in the visual acuity of one eye does not interfere with binocular vision, but a great one takes it away altogether. Where we are to draw the line here is a matter that varies greatly in individual cases, being dependent not only on the degree of visual acuity, but also on many other factors, such as the visual field and refraction of the worse eye, the relations of the eye muscles, etc.

Abolition of binocular vision may also occur when the injury in one eye is but slight, and yet a latent disturbance of equilibrium of the eye muscles (a heterophoria) was previously present. This latent disturbance now becomes manifest, i.e., is transformed into a squint, and, by this, binocular vision is abolished (see § 337).

In many cases loss of binocular vision is not important. This is the case not only in such occupations as labor in the fields, but also in fine work, like writing; even the watchmaker who does the most minute work with a magnifying glass does not require binocular vision. But many other kinds of work do require a precise and certain appreciation of distances, and even such an ordinary thing as splitting wood may be extremely dangerous to the fingers of the workman if he does not have a correct judgment of depth. It must be noted, however, that the necessity for binocular vision in such cases has been somewhat exaggerated. In chopping, hammering, wheeling a barrow along a narrow path, etc. the workman is concerned not so much with depth and distance as with direction, and this can be determined as well with one eye as with two (Fergus). In this or some other way, practice enables one to make up for deficiencies in depth-perception, and thus it happens that there are persons, who, because of unilateral blindness or because of squint, have had nothing but monocular vision since youth, and who yet are as serviceable workmen as those who have normal sight.

The disability produced by an injury affects both the *working capacity* and the *earning capacity.* The two are not quite the same; thus the man with simply one blind eye has theoretically the same impairment of working capacity as the man in whom one eye has been enucleated, but his earning capacity is greater, because it is harder for a palpably one-eyed man to get a situation. This very distinction, in fact, is recognized in some of our states.

The *diminution in earning capacity* may be expressed as a fraction of the total normal earning capacity before the accident. This fraction, E, is determined by three

factors. These are: F, the functional capacity of a man's various organs expressed as a percentage of the normal; T, his technical training and ability; C, his competing ability, i. e. his ability and opportunity to apply his functional capacity and technical skill in making money. Various formulas, more or less complex have been deduced to determine E from F, T and C (Magnus, Würdemann, Holt), but there is as yet no agreement in this regard. Such computations are useful in enabling an expert to form a fair estimate of the amount of loss that a person has experienced in his earning capacity and in stating this loss to a jury in precise percentages. But as regards workmen's compensation the law fixes the indemnity in a much simpler fashion. The laws in this regard vary, but in this country, generally speaking, T is replaced by the weekly wage which the workman was getting before the accident and which naturally varies with his technical ability; F is replaced by the number of weeks for which payment is made, which number varies with the degree of organic loss; and C is not recognized at all or only indirectly. Thus the state of New York rules that for the permanent loss of one eye, a man shall receive two-thirds of his weekly wage for 128 weeks and for the loss of both eyes the same compensation for life. For a minor degree of loss he should receive two-thirds of the difference between his wages at the time of the injury and his wages afterward.

In foreign countries the loss of one eye is regarded as reducing the earning power by 25 to 33 per cent. (in some countries only 10 per cent.) In some countries loss of binocular vision has been considered as equivalent to the loss of one eye.

CHAPTER XXXI

DISEASES OF THE ORBIT

I. Changes of Position of Eye in Orbit

863. Changes of Position in General; Exophthalmus.—Changes of position of the eye in the orbit include changes of direction and exophthalmus and enophthalmus. Changes of direction—strabismus—although sometimes of mechanical origin, i. e., due to processes occurring in the orbit, are usually due to a disorder of motility and are hence considered in Chapter XVII.

Exophthalmus consists in an undue protrusion of the eye from the orbit. On account of the great individual variations in the position of the eyeball, small degrees of exophthalmus can be diagnosticated with certainty only when they are limited to one eye, so that, by making the comparison with the other eye, a guide to the diagnosis is secured. Higher degrees of exophthalmus attract our notice at once. The protrusion of the eyeball may be so great that the lids can no longer keep the eye back in the orbit, and thus it prolapses in front of the lids—*luxatio bulbi*. The protrusion is either directed straight forward, or is associated with a lateral displacement of the eye.

For measuring the degree of exophthalmus, instruments have been constructed, which are called *exophthalmometers* or statometers. For these can be substituted a thin flat trial-frame so placed that the plane of the instrument is parallel with the frontal plane. Each eye in succession is made to sight an object straight ahead and while it is doing so, the distance is measured between the plane of the frame and the external angular process, also between the former and the apex of the cornea. A comparison of the measurements on the two sides will show how far each eye protrudes in front of its own orbit, and how far one is in advance of its fellow.

Exophthalmus is *caused* either by an increase in the volume of the orbital tissue or by a diminution in the capacity of the orbit. The former is much the more frequent, (see however, § 53). The main conditions producing it will be given in other sections of this chapter. But exophthalmus can also be produced by a diminution of the tone of the recti muscles, which draw the eye backward. This is the case in paralysis or in division (tenotomy) of them. Such exophthalmus is sometimes apparent only; e. g., after tenotomy the eye may seem to protrude simply because the lid is retracted by the receding tendon (see § 983).

A transient exophthalmus is produced by distention of the orbital blood-vessels, e. g., under the stress of emotion (Arlt, Donders) or from the hyperæmia of menstruation, (Cohn).

The *consequences* of exophthalmus when it is of high degree are extremely disastrous to the eye: 1. The farther the eye comes forward, the more it

pushes the lids apart. The palpebral fissure is therefore more widely open and more of the eyeball is visible in it than usual. In slight cases of exophthalmus the dilatation of the palpebral fissure is often more conspicuous than the actual protrusion of the eyeball. The fact of the eyeball's being more exposed results in symptoms of irritation upon the part of the conjunctiva, such as redness of the ocular conjunctiva and epiphora. As the protrusion of the eyeball grows greater, the closure of the lids becomes imperfect (lagophthalmus) and then the cornea begins to suffer, because keratitis e lagophthalmo develops. This is the most dreaded sequel of exophthalmus. 2. The pressure which the eyeball exerts upon the lids from behind leads to their eversion; ectropion of the lower lid develops. 3. The mobility of the eyeball diminishes in proportion as the protrusion increases, owing to the marked stretching of the recti muscles and of the optic nerve. 4. Vision is affected in various ways. In the cases in which, together with the protrusion of the eye, a lateral displacement of the organ is present, diplopia makes its appearance. Later on, the vision of the protruding eye may be abolished altogether by keratitis or by disease of the optic nerve. The latter, as long as the protrusion of the eye is but slight, is subject to no undue tension, its normal S-shaped curve (§ 48) being simply straightened out; it is not until the eye is caused to protrude pretty far that the optic nerve is put on the stretch. If this stretching takes place gradually, the nerve fibres often accommodate themselves to it in a wonderful fashion, so that they preserve their conductivity, and vision is maintained intact; but if the exophthalmus increases rapidly, the optic nerve owing to the traction soon loses its conducting power.

The *treatment* consists essentially in the removal of the cause. The symptomatic treatment is mainly that of lagophthalmus (see § 442).

864. Exophthalmic Goitre.—A condition specially marked by exothalmus is exophthalmic goitre (Grave's or Basedow's disease). In this the exophthalmus is bilateral; the eyes are pushed straight forward, sometimes but little, sometimes to such an extent that they can not any longer be perfectly covered by the lids. Even when the exophthalmus is great, there is little or no limitation of mobility of the eye. It is obvious that when the exophthalmus is considerable, it becomes noticeable at the first glance; but even when the protrusion is slight the peculiar appearance of the eyes strikes one at once. This appearance is caused by the fact that the upper lids are raised unusually high. The eyes look as if forced wide open, and give the patient an expression of astonishment or fear (Dalrymple's sign). When the eyes are depressed, the upper lids do not descend properly with the eyeball, but remain elevated, so that a broad portion of the sclera is visible above the cornea (Von Graefe's symptom). Winking takes place less frequently (Stellwag's symptom), and hence desiccation of the deficiently covered cornea is favored. Convergence of the eyes is hindered (Moebius' symptom). Eversion of the upper lid is sometimes difficult (Gifford). The eyeball itself, as long as the cornea has not yet suffered harm, is normal and the visual power is good. Sometimes there is pulsation of the retinal arteries.

The exophthalmus has been attributed to (*a*) engorgement of the orbital vessels; (*b*) irritation of the sympathetic causing tonic contraction of the unstriated muscle fibres running in the orbit from the equator of the eye to the orbital septum (Landström); (*c*) to accumulation of fat in the orbit with œdema of the ocular muscles (Foster Moore); (*d*) to suprarenal insufficiency (Shapiro, and Marine).

Dalrymple's, Von Graefe's, and probably Gifford's signs are referable to spastic contraction of the musculus tarsalis superior due to irritation of the sympathetic.

When a stethoscope is placed over the closed lids, there is occasionally heard, besides the constant hum due to contraction of the orbicularis, a murmur, said to be systolic in rhythm and hence regarded as vascular in origin (Snellen and others). But according to Sattler it, too, is due to muscular contraction.

The two other main symptoms of Basedow's disease are the *swelling of the thyroid gland* and the acceleration of the heart's action (*tachycardia*). The former is distinguished from ordinary goitre by the fact that when the hand is placed upon the thyroid gland it feels the strong movement of pulsation in the arterial vessels, which is communicated to the entire gland. So also the carotids are found to be dilated and strongly pulsating. The intensity of the heart beat is increased, the frequency of the pulse is regularly over a hundred and is increased at once very considerably by the slightest bodily exertion or mental excitement. The physical examination of the heart, apart from the enlargement of the left heart, gives a normal result. Vibratory tremor and increased secretion of sweat are almost always present. The general state of the patients is disordered in that they are for the most part very excitable, and suffer from the symptoms of anæmia or chlorosis. Not infrequently rapid emaciation is present, even when the appetite remains good.

Basedow's disease was first described as a special form of disease by English physicians, and particularly by Parry and afterward by Graves. These authors, however, did not recognize the exophthalmus as being one of the essential symptoms of the disease; this was first done by Basedow, who in 1840 did the pioneer work in demonstrating the whole triad of symptoms of the disease.

In marked cases no disease is easier to diagnosticate than this; even from a distance we can tell what the patient's trouble is. But, on the other hand, there are numerous cases in which some of the symptoms are less pronounced or are even wanting altogether, so that the diagnosis becomes difficult. Taking only the exophthalmus into account, we find that it may be slight, absent altogether, or confined to one eye. When exophthalmus is absent, Von Graefe's symptom is still sometimes present, so that it is not to be regarded as due simply to the exophthalmus; but it, too, is not constant, and in the same case may be sometimes present, sometimes wanting.

The disease principally attacks women, beginning at the time of puberty, and extending up to the appearance of the menopause. Its *cause* is assumed to be a poisoning of the system by the excessive or perverted action of the diseased thyroid gland.

Course and Treatment.—Exophthalmic goitre generally develops quite gradually. Palpitation usually sets in first, then the thyroid dilatation, and last of all, the exophthalmus. As a general thing it takes months or even years before all the symptoms of the disease are distinctly marked. Then it usually remains at the same point for years, after which it subsides again very gradually and not without leaving a tendency to subsequent recur-

rences. In many cases, however, the disease is not recovered from, but remains until the patient's death, nay, more, it may even—by the exhaustion or the complications which it produces—be itself the cause of death. As a general thing the disease runs a severer course in men and in elderly people than in women and in the young. It is a source of danger to the eyes from the fact that when the exophthalmus is of a pretty high degree, closure of the lids is imperfectly performed and keratitis e lagophthalmo develops. In this way blindness of one or even of both eyes may be produced.

Precisely as the separate symptoms of Basedow's disease may show great variations in the extent of their development, so also may the course of the disease vary exceedingly. Although as a rule this is very chronic, yet cases are known in which the disease had a most acute onset. Trousseau tells of a woman in whom the symptoms of the disease developed in the course of a single night, which she had spent in tears, lamenting her father's death. The subsequent progress of the disease, too, may be so speedy that within a few weeks it leads to recovery or to death.

Treatment need be considered here only in so far as it relates to the eyes. The exophthalmus requires treatment only when it leads to imperfect closure of the lids and thus endangers the cornea. In this case we must, by bandaging the eyes at night, provide for their being covered during sleep. If the bandage turns out to be insufficient, we must perform tarsorrhaphy, by which the palpebral fissure is permanently closed in its outer portion.

Foster Moore by removing a quantity of œdematous fat from the orbit through an incision in the lower fornix relieved exophthalmus which was so great as to threaten destruction of the eye.

865. Enophthalmus.—The opposite condition to exophthalmus, namely, the recession of the eyeball into the orbit—enophthalmus—occurs:

1. In decrease of the orbital fat consequent upon extreme emaciation. In Asiatic cholera this condition develops within a few hours, owing to the enormous loss of water from the tissues (Von Graefe). With regard, however, to enophthalmus of this kind, see § 53. 2. In diminution of the orbital contents due to operations in which a part of them is removed (e. g., in extirpation of an orbital tumor). 3. In paralysis of the sympathetic. 4. After injuries (*enophthalmus traumaticus*). In most cases of this kind the traumatism does not affect the eyeball itself, but the margin of the orbit. It is only exceptionally the case that the enophthalmus occurs right after the injury. Usually it does not develop until after several weeks have elapsed. The anatomical changes causing it differ in the different cases. The most frequent condition is a cicatricial contraction of the orbital tissue or an indirect fracture of one of the orbital walls with depression of the fragment of bone outward so that the volume of the orbit is enlarged, and the eyeball is pushed back by the pressure of the external air. In one case the eyeball itself was luxated into the antrum of Highmore by a blow from a cow's horn. 5. In cases of intermittent exophthalmus (see § 881). 6. After spontaneous subsidence of a pulsating exophthalmus (Bronner). 7. In neurotic atrophy of the face. 8. As a congenital anomaly of position. 9. In many cases of congenital paralysis of the external rectus the eye when adducted sinks back into the orbit, generally because the internus and an inextensible band replacing the externus pull on the eye like a pair of reins. See page 320.

An enophthalmus is often simulated, the eye appearing to recede simply because covered more than usual by the lids. This happens in ptosis, true or false, after advancement (§ 66), and with spasm of the musculus tarsalis.

II. Inflammations of the Orbit

(*a*) *Inflammations of the Bony Wall and of the Periosteum of the Orbit.*

866. Symptoms and Course.—Periostitis of the bones of the orbit is not rare, particularly at the orbital margin. Here, too, it is easiest to diagnosticate. A hard swelling is felt immovably attached to the bone and causing the margin of the orbit to appear thicker and misshapen—a fact which is particularly striking when comparison is made with the sharp margin of the orbit on the other side. In view of the great tendency toward œdematous swelling possessed by the lids and the conjunctiva, it is quite conceivable that such a swelling may be present to a greater extent upon one side than upon the other; still, it is usually easy to feel the tumefaction of the periosteum through the soft swelling of the lids. Moreover, the affected spot is distinguished by its greater sensitiveness to pressure.

If the periostitis is situated, not at the margin, but in the depth of the orbit, the diagnosis is much more difficult. We have at first simply the signs of a painful inflammation in the deeper parts of the orbit. That this inflammation starts from the periosteum is frequently not apparent until an abscess forms and breaks through to the outside, in which case we then come down upon the diseased bone with the sound.

The *course* of periostitis leads in favorable cases to complete resorption of the periosteal exudate or to the formation of a permanent deposit of bone (especially in syphilitic periostitis); it is more unfavorable when the periostitis goes on to suppuration which is followed by caries and necrosis of the bone.

When a periosteal abscess develops at the margin of the orbit, the skin over the affected spot first becomes reddened, then becomes thinned by the pus, and finally is perforated. A fistula is thus produced, through which the sound passes down to bare and roughened bone. Afterward there develops at the site of the fistula the funnel-shaped indrawn depression that is characteristic of bone disease. The discharge of pus from the fistula keeps up until all the diseased bone that has died has been eliminated, a process for which often several years are required. Then the fistula heals and leaves an indrawn, funnel-shaped scar attached to the margin of the orbit. Through this can be felt the defect in the margin of the orbit left by the necrosis. Other frequent sequelæ are ectropion of the affected lid and even lagophthalmus. These two conditions result partly because the lid becomes attached to the margin of the orbit and is drawn up strongly toward it, partly because a portion of the skin of the lid has been destroyed in consequence of the prolonged suppuration (Fig. 249).

When the periostitis which has gone on to the formation of an abscess is situated in the depth of the orbit, the disease runs its course with the symptoms of retrobulbar phlegmon, which will be described later on. The process is then much more severe and of longer duration since it takes a good while for the pus to make its way from the depth of the orbit to the surface. These deep suppurations, moreover, may be dangerous to life, if they are transmitted to the cranial cavity and give rise to meningitis or

abscess of the brain. In this respect the periostitides of the roof of the orbit are particularly to be dreaded, because here the cranial cavity is separated from the focus of pus in the orbit by only a very thin lamella of bone.

Chronic periostitis, particularly when syphilitic, may set up a gradually increasing thickening of the bones of the orbit, by which the orbital cavity is progressively contracted. The consequence is exophthalmus and compression of the nerves which enter the orbit, so that neuralgiæ and paralyses are produced. This complex of symptoms is like that seen in *leontiasis ossea.* This consists in a progressive thickening of the bones of the whole face, a process in which the bones of the orbit share, so that in this case, too, the symptoms of contraction of the orbit with simultaneous thickening of its walls are produced.

867. Etiology.—The causes of periostitis are: 1. Injuries. This traumatic periostitis is found most frequently at the margin of the orbit, because this is the part most exposed to injuries. 2. Dyscrasiæ, particularly scrofula (tuberculosis) and syphilis. These periostitides are likewise localized more frequently at the margin than in the depth of the orbit, because in this case, too, injuries play a part as exciting causes. Injuries of a comparatively trifling nature, such as bumps or falls, which in healthy persons would remain without further ill result, may in people affected with dyscrasiæ start up protracted specific inflammation. Scrofulous (tuberculous) periostitis occurs chiefly in children and principally affects the superior external and inferior external margins of the orbit, which are the parts most exposed to knocks; it leads, as a rule, to caries. Syphilitic periostitis, on the contrary, is ordinarily met with in adults and only exceptionally in children (those with hereditary syphilis). It belongs to the third (gummatous) stage of syphilis, and for the most part appears as a chronic affection under the form of periosteal thickening, more rarely as an acute affection with suppuration following it. 3. Suppuration in the accessory cavities of the nose (§ 886).

868. Treatment.—Treatment must, above all, have regard to the *etiological* factor. In this respect the best results are obtained in syphilitic periostitis, which usually subsides rapidly under a promptly initiated treatment with mercury and iodide of potassium. As *local* treatment moist hot compresses are applied, which in the beginning favor resolution, but in the later stages accelerate the softening of the abscess that is in process of development. As soon as there are signs that suppuration has taken place, there should be no delay about making the incision, so that the pus which has accumulated beneath the periosteum may not detach the latter still more extensively from the bone. Particularly in the case of deep-seated periosteal abscesses is early incision indicated, even though no fluctuation is yet perceptible, in order to prevent the transfer of the suppuration to the brain. After the abscess has been opened, a drainage tube or a strip of iodoform gauze is placed in the wound, so as to keep it open for the exit of the pus. If caries or necrosis succeeds periostitis, they are to be treated according to the general rules of surgery. Ectropion and lagophthalmus, which may develop subsequently, likewise call for relief by operation. This relief must be given without delay when the cornea is in danger because insuffi-

ciently covered. In other cases it is better to defer operative interference until the process in the bone has entirely healed, as otherwise the success of the lid operation might be jeopardized by the renewed formation of abscesses and fistulæ.

(b) *Inflammations of the Cellular Tissue of the Orbit*

869. Symptoms.—Inflammation of the orbital cellular tissue—*orbital cellulitis*—manifests itself by a marked œdema of the lids and conjunctiva (chemosis). An associated symptom, and the most important of all, is the protrusion of the eyeball, caused by the swelling of the tissues behind it. Because of this protrusion the eyeball's motility is impaired, and actual paralysis of the eye muscles may be superadded. The sight is often reduced or may even be entirely abolished owing to an inflammation of the optic nerve, which may be purely retrobulbar or may extend to the intra-ocular end of the nerve, in which case the ophthalmoscope shows an inflammation of the nerve-head. The gland behind the ear is swollen. At the same time there are violent pain, fever, and not infrequently also cerebral symptoms, such as headache, vomiting, stupor, retardation of the pulse, etc.

Cellulitis of the orbit may subside by absorption of the exudate that has been deposited, and in favorable cases everything may return to the normal. But often a slight degree of exophthalmus, or paralyses of the eye muscles, or atrophy of the optic nerve may be left. In other cases, however, the inflammation goes on to suppuration, the condition being then called *orbital phlegmon* (retrobulbar phlegmon, retrobulbar abscess). When the symptoms have reached their acme, the skin of the lids at a certain spot grows red, then shows a yellow discoloration, and finally is perforated by a discharge of pus. After the discharge of the pus, which is present in large quantity, the inflammatory symptoms in most cases rapidly abate and the opening soon heals again. The sight may suffer permanent diminution or be altogether annihilated, if the optic nerve has been implicated either through inflammation or through thrombosis of its vessels. Detachment of the retina and even suppuration of the eyeball (panophthalmitis) also occasionally occur in retrobulbar phlegmon. If the suppuration is carried over from the orbit to the cranial cavity, it leads to a fatal issue through purulent meningitis or abscess of the brain.

870. Etiology.—Retrobulbar phlegmon may originate from the following *causes*: 1. Injuries, when the body causing the traumatism enters the orbit and carries infectious germs into the tissues. Those injuries in which a foreign body is left in the orbit are particularly dangerous. Operations, too, such as enucleation, may cause orbital suppuration if not performed aseptically. 2. Transfer of inflammation from the wall of the orbit or parts in the vicinity of the latter, such as the accessory cavities of the nose, to the orbital cellular tissue. 3. Erysipelas; the inflammation being transmitted from the skin to the deeper parts. 4. Metastases in pyæmia, typhus, scarlet fever, smallpox, purulent meningitis, influenza, etc.

The conditions most often causing inflammation of the orbit are empyemas of the cavities adjoining it, and in this regard the ethmoid cells are of more significance than the

maxillary and frontal sinuses, since the former are separated from the orbit by a partition of bone no thicker than paper (cf. § 51 and Fig. 387). Hence in every orbital cellulitis not due to external injury we should always make a rhinoscopic examination.

In other cases there is a primary inflammation of the bony wall of the orbit and as the periosteal pus forces its way out from the deeper parts it sets up an orbital cellulitis. More difficult to recognize is the condition in which the diseased bone is situated far from the orbital cavity, as, for instance, in caries of the petrous bone or in suppurative periostitis of the alveolar process of the superior maxilla. The latter is the case in dental periostitis or after the extraction of diseased teeth. The path that the inflammation takes in these cases is either along the anterior surface of the upper jaw or through the maxillary antrum, in which the diseased tooth has already set up an empyema.

Orbital phlegmons may develop in pharyngitis and in suppurative parotitis by transmission of inflammation from behind forward; and by transmission from before backward in panophthalmitis, in abscess of the lids, and in dacryocystitis acuta, when, as exceptionally happens, perforation of the wall of the lacrimal sac takes place backward instead of forward.

Orbital phlegmons may lead to thrombosis of the cavernous sinus; although the converse process may also happen—i. e., the thrombosis starting from a thrombosed cavernous sinus may extend until abscess in the orbit is formed too.

871. Treatment.—When we have an orbital cellulitis to treat, we must first of all try to remove its cause. Under this head belong the antiseptic treatment of wounds of the orbit, the provision for the free escape of secretion from wounds, the removal of foreign bodies from the orbit, the treatment of suppurating accessory cavities, such as the ethmoid cells, etc. The inflammation itself is combated by moist hot compresses, to which may be added the application of leeches to the temple, cathartics, diaphoresis, etc. If an abscess develops, the indication is to open it as early as possible in order to prevent the suppuration from spreading to the brain. We introduce a sharp-pointed scalpel at the spot where we suppose the abscess to be, and do not hesitate to plunge it deep in. If the phlegmon starts from the bone or from the accessory cavities, the pus at the outset of the disease still lies beneath the periosteum. Then this must be split along the wall of the orbit and be stripped up until the pus is reached. The situation of the abscess is inferred from the way in which the eyeball is displaced. If, for instance, the latter is pushed forward and downward, the abscess is to be sought for in the upper part of the orbit. Even when we do not succeed in getting out any pus by our incision, because no abscess cavity of any size has yet formed, yet the relief of tension in the tissues, produced by the incision, and the profuse bleeding exert a favorable effect.

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872. Thrombosis of the Cavernous Sinus.—Symptoms similar to those which present themselves in the beginning of a retrobulbar phlegmon accompany *thrombosis of the cavernous sinus.* The lids and the conjunctiva swell up with œdema, and the eyeball protrudes and becomes difficult to move. The veins of the retina are seen, upon ophthalmoscopic examination, to be enormously distended. At the same time there is a doughy œdema in the mastoid region. These symptoms are referable to the fact that the veins of the orbit discharge the greater part of their blood through the ophthalmic veins into the cavernous sinus; hence thrombosis of the latter produces stasis in the veins of the orbit, and especially if the thrombotic process is continued on from the sinus into these veins. The stasis leads to protrusion of the eyeball and also to venous hyperæmia of the retina. The œdema of the mastoid region depends upon the fact that in this region an emissary vein (the emissarium mastoideum) empties into the transverse

sinus, and hence when the thrombosis is carried along from the cavernous to the transverse sinus, the mastoid region also shares in the venous stasis. When this œdema is present (which, to be sure, is not always the case), it forms an important differential sign between sinus thrombosis and retrobulbar phlegmon in which latter it is absent. A further difference is that sinus thrombosis frequently passes over to the other side, so that the same complex of symptoms develops there also, while, on the contrary, a bilateral orbital cellulitis would be one of the greatest rarities. Finally, sinus thrombosis is associated with very severe cerebral symptoms, terminated at last by death.

In occlusion of the sinus what we are dealing with is either a simple thrombosis produced by marasmus or a thrombosis due to infection. The latter usually originates from a focus of pus situated in the vicinity—e. g., from a phlegmon of the orbit, which gives rise to a thrombotic process in the superior or inferior ophthalmic vein, this process being then carried over into the cavernous sinus; or most frequently from a caries of the petrous bone, the thrombosis extending from the petrosal sinus to the cavernous sinus. Suppurative processes starting from the posterior ethmoid cells, maxillary antrum, or buccal cavity (teeth or tonsils) also may lead to sinus thrombosis. Finally, thrombosis of the sinus may be set up by erysipelas and may also occur metastatically in pyæmia and infectious diseases.

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873. **Tenonitis.**—Tenon's capsule may be implicated in inflammation of the eyeball, so that an inflammatory œdema develops in the capsule itself and in the adjoining cellular tissue of the orbit, and the eyeball is thus pushed forward. Hence a slight degree of exophthalmus is sometimes found in severe cases of irido-cyclitis (particularly after injuries). This is quite regularly the case and to a much higher degree in panophthalmitis, in which exophthalmus is one of the most constant and striking symptoms. After the subsidence of the inflammation, extensive adhesions are formed between Tenon's capsule and the eyeball—a fact of which we can convince ourselves if an enucleation is subsequently performed. Exudation into Tenon's space also occurs after it has been laid open by injuries, and particularly after squint operations, when infection of the wound has taken place from dirty instruments.

There is also a primary *serous tenonitis*. The symptoms of this rare disease can be best gathered from the description of the following case described by Fuchs: It concerned a woman of fifty-eight years of age, otherwise healthy, in whom the disease had begun, without known cause, six days before she came under observation. The skin in the vicinity of the lids, and still more the lids themselves were very swollen and œdematous, so that the eyes could be opened to only a very small extent indeed by spontaneous effort. The eye was prominent and almost immovable. The conjunctiva of the lids was but moderately, that of the eyeball, on the other hand, quite markedly, injected, and the latter was so greatly swollen as to form a thick protuberance projecting from all sides over the cornea. The secretion was not increased. The cornea and also the deep portions of the eye were healthy, and vision was normal, except that there was diplopia due to the impaired mobility of the eyes. The disease was associated with moderate pain, and particularly with a sense of pressure and tension of the eyes. An infusion of jaborandi was given in order to produce rapid absorption of the exudate by means of profuse diaphoresis. Under this treatment the œdema and the protrusion of the eye gradually went down, so that in four weeks the condition of the eyes had become once more perfectly normal.

The causes of tenonitis are obscure; gout, rheumatism, and refrigeration are the etiological factors that have been held accountable for it. Some cases (among them one case of primary purulent tenonitis) have been observed as a result of influenza. The disease tends to relapse, but leaves no permanent bad results behind.

Tenonitis like orbital cellulitis, is characterized both by œdema of the lid and the conjunctiva and by protrusion of the eyeball. The distinction between the two affections is to be made from the amount of protrusion of the eye compared with that of the chemosis. If the latter is very pronounced and the exophthalmus is slight, a tenonitis must be assumed to exist; while in retrobulbar cellulitis even when the protrusion of the

eye is marked, the chemosis is often not very great and may even be confined to the area occupied by the interpalpebral fissure. Frequently, however, it is impossible to distinguish certainly between the two sets of cases, which in fact are not sharply differentiated even anatomically. There is little doubt that many cases which were formerly called tenonitis would now be regarded as orbital cellulitis.

III. Injuries

874. Injuries of the Soft Parts.—Injuries of the orbit affect either the soft parts alone or the bones as well. Injuries of the *soft parts* originate, as a rule, from the penetration of a foreign body into the orbit, the lids and the eyeball being, of course, very frequently implicated at the same time. The direct consequence of the injury is an extravasation of blood into the tissues of the orbit. If this is considerable it causes exophthalmus, and also, since the blood oozes slowly forward, it comes into view beneath the conjunctiva and the lids under the form of an ecchymosis. Paralysis of the ocular muscles, too, may be produced by the injury, and so also may lesions of the optic nerve, the latter entailing as their direct result partial or complete blindness. It also happens sometimes that the eyeball is driven out of the orbit by the foreign body which has penetrated into the latter, and is thus found lying in front of the lids (*luxatio bulbi traumatica*). This is most apt to happen when the body that causes the injury enters from the outer side, since here the wall of the orbit recedes the farthest—as far back as the plane of the equator of the eyeball or even much farther (§53). There are parts of the world where such injuries are purposely inflicted in brawls, the eye being pried out of its socket by the thumb, which is thrust into the orbit from the outer side. Insane patients have sometimes enucleated one or both of their own eyes in this way. Furthermore the eye may be dislocated or torn out by injury (pressure of the forceps) at the time of birth. Ordinarily the luxated eyeball is lost, but cases are known in which after reposition the eye healed again in its place and retained its visual power.

875. Injuries of the Bones.—Injuries of the bone are most frequently produced by gun shot (especially a shot in the temple in attempted suicide) or the action of a contusing force (e. g., by a blow or a fall) upon the margin of the orbit. They are easy to diagnosticate when they affect the margin of the orbit itself. A fracture in this locality is recognized by the unevenness, sensitiveness to pressure, and in marked cases the crepitation.

Deep fractures of the orbit without fracture in the margin may be produced by deeply penetrating foreign bodies, but may also be caused apart from these and indirectly (by contre-coup). This is the case, for example, after a fall upon the head, the dropping of a heavy weight upon it, etc. Such fractures when deeply situated can only be suspected from the fact that they are accompained by orbital hæmorrhage, which manifests itself by a suddenly developing exophthalmus and the appearance later on of ecchymosis of the conjunctiva and the lids. A further diagnostic point would be supplied if directly after the injury partial or total blindness were determined to exist along with a normal appearance of the eyeball. This condition would necessarily be referred to an lnjury of the optic nerve in the canalis opticus, produced by fracture of the wall of the latter (Hölder and Berlin; see § 822) or by hæmorrhage into the nerve sheath. Similar symptoms also at times accompany fractures of the base of the skull, except that in this case the exophthalmus is wanting and the ecchymosis of the conjunctiva and lids sets in still later, since the blood takes a longer time to push its way so far forward.

Spontaneous hæmorrhages into the orbit are of extremely rare occurrence, taking place as a consequence of whooping-cough or in persons who in general are predisposed to hæmorrhages, e. g., in infantile scurvy in which they produce exophthalmus (Stephenson).

876. Emphysema of the Orbit.—When in consequence of the fracture the orbit is made to communicate with the neighboring cavities, emphysema may develop in the orbit. Wherever the air has accumulated in the anterior portions of the orbit, it can be felt through the lids; but air that has come out behind the eyeball makes its presence manifest by exophthalmus. Whether such an exophthalmus is caused by exuded blood or by air can be determined by noting that in the latter case the protruded eye can be pushed back into the orbit again with the finger since the air is displaced by the pressure. On the other hand, the exophthalmus is increased by straining during the acts of coughing, blowing the nose, etc., owing to the fact that fresh supplies of air are forced into the orbit.

Emphysema of the orbit and lids develops after a contusion affecting the eye. By the force that acts on it, the eyeball is driven back into the orbit, and the orbital fat is made to recede to one side. But it meets with a firm resistance in the wall of the orbit everywhere except on the nasal wall which is formed of the thin lamina papyracea. This is pushed in, and thus one of the ethmoid cells is put in communication with the cellular tissue of the orbit.[1] The mere presence of such a communication between the orbital tissue and a pneumatic cavity does not suffice to produce emphysema; to effect this, air must be driven through the fracture into the orbital tissue. Ordinarily this occurs from blowing the nose, in doing which strong expiration is made while the nasal orifice is kept shut and consequently the air in the nasal fossa is put under high pressure. Thus is explained the circumstance that emphysema often does not develop until several hours after the injury and then quite suddenly when the patient blows his nose. Violent blowing of the nose in some rare cases has produced emphysema even when there was no antecedent injury. [This happens in men in whom the lamina papyracea has particularly thin spots which are forced open by the heightened pressure of the air.

The air which passes through the fracture in the lamina papyracea gets first into the orbital tissue (*orbital* emphysema). The eyeball is driven forward and so is the tarso-orbital fascia which is squeezed against the skin of the lids so that the lids in their whole extent are stretched tight like a drum and protrude, for which reason these cases are usually regarded as an emphysema of the lids. Owing to the entrance of air behind the septum orbitale (see § 4) the lids are squeezed together and the palpebral fissure is narrowed, while in exophthalmus of other kinds the lids are pressed apart by the eye. If the pressure under which the air enters the orbit is particularly high and in addition the tarso-orbital fascia is weak (as is the case in old age) the air may break through the fascia and then gets under the skin of the lids—*orbito-palpebral* emphysema. We can then feel the air beneath the thin skin of the lids much better than we can when it is beneath the tightly-stretched fascia (page 427). Moreover, the swelling extends to the free border of the lid, while in the other case it stops at the convex border of the tarsus where the fascia is attached (Heerfordt). A purely *palpebral* emphysema without implication of the orbit is extremely rare. It occurs when the point at which the air enters the tissue lies in front of the septum orbitale, e. g., in case of injury of the lacrimal sac.

877. Results and Treatment of Injuries.—The injury, if no important organs have been destroyed, may heal after the resorption of the extravasated blood with a restitutio ad integrum. In other cases disturbances of

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[1] If the contusion has resulted in a rupture of the sclera, emphysema does not develop, because the eyeball being collapsed does not push the orbital fat aside with enough force to produce this result. Emphysema and rupture of the sclera, therefore, although both are consequences of a contusion affecting the eye, are mutually incompatible conditions.

motility of the eyeball remain, owing to its adhesion to the neighboring structures, or as a result of paralysis of the ocular muscles. Moreover, if the optic nerve has been injured, permanent blindness of the eye may ensue. Still worse are the cases in which the injury results in a phlegmon of the orbit, in which case it may even end fatally.

The *treatment* of a recent injury varies according to circumstances. If the wound is very lacerated and dirty, the indication is to remove the shreds of tissue that are likely to die and to cleanse the wound; furthermore, if a foreign body of any considerable size is lodged in the orbit, this must be removed at once, and the wound kept open with a drainage tube or strips of gauze. If, on the other hand, the lips of the wound are in good condition or have actually perhaps already united, we do not even wash them off, but simply place a dry sterile dressing over them. If in this case there is a foreign body in the orbit, we put off making any attempts at extracting it until we find whether or not it is going to remain without causing trouble. Projectiles, in particular, often do so remain. If there is a considerable hæmorrhage into the orbit or an emphysema, a pressure bandage contributes to the rapid absorption of the blood or the air. As soon as the symptoms of deep suppuration have set in, we must proceed as in the case of a retrobulbar phlegmon.

IV. Tumors of the Orbit

878. Tumors of the orbit—in the widest sense of the word—have in common one very important symptom, namely exophthalmus. If this is present the main thing to determine is whether an inflammation or tumor growth causes it. Such a determination is easy, as a rule, since inflammatory exophthalmus starts up acutely and with inflammatory symptoms (swelling of the lids and conjunctiva, pain), while exophthalmus due to tumor growth develops gradually and without signs of irritation. Yet not infrequently diagnostic errors do occur especially in the case of certain growths which simulate tumors closely but which on the operating table are found to be the products of a chronic inflammation. These inflammatory pseudoplasms may be of tuberculous, syphilitic, or unknown origin.

To determine the site of the tumor, we must consider the nature of the protrusion: whether it is directed straight forward, or whether there is also a lateral displacement of the eyeball. With the same object in view we test the mobility of the eye in all directions. Then we try to palpate the tumor itself, so as to be able to tell its size, form, consistency, mobility, etc. If the tumor lies deep in the orbit, we endeavor to push the little finger as deeply as possible between the margin of the orbit and the eyeball (under narcosis, if necessary), so as to get at the tumor. Finally, we determine the visual acuity and examine the fundus with the ophthalmoscope thus ascertaining whether and in what way the optic nerve has been injured.

Tumors of the orbit include the rarely occurring tumors of the lacrimal gland (§ 525). These do not in general cause exophthalmus, but a swelling perceptible with the finger under the upper outer margin of the orbit. Sometimes the gland is dislocated downward so as to appear as a swelling under the upper retrotarsal fold.

879. Cysts.—The most frequent of these are the *dermoid* cysts, which are congenital, but often develop to a greater size after birth. They generally lie in the anterior part of the orbit, and usually in its upper and outer or upper and inner angle. On account of their superficial situation, they do not displace the eyeball, but push forward the skin of the lids, through which they can readily be felt as round, movable tumors as big as a bean or walnut. Upon extirpating them one can convince himself that they are, as a general thing, unilocular cysts with pultaceous or sebiform contents; sometimes processes from them extend pretty deep into the orbit, and thus render complete removal difficult. The only harm that dermoid cysts produce is the disfigurement caused by them, and this, too, is the reason why, quite often, we proceed to extirpate them. In doing this, we must go to work very carefully so as to dissect out the cysts, as far as may be, unopened. If the cyst wall, which often is thin, breaks prematurely, part may easily be left behind and cause recurrences.

Fig. 386.—Cephalocele Orbitæ Anterior.

Histological examination of dermoid cysts shows that their wall possesses essentially the structure of the external skin—that is, it consists of a substratum of connective tissue, the cutis, which supports an epithelial lining like that of the external skin, and which not infrequently contains hair follicles and glands (sebaceous and sweat glands). The contents of the cyst are mostly like porridge or sebum, and are formed of the exfoliated epithelial cells and of the secretion of the glands contained in its walls; in many cases hair, and in very rare cases teeth have been found in them. Sometimes the contents of the cyst are converted into an oily or honey-like liquid (oil cysts and honey cysts or meliceris); or they may even become like serum. The anatomical structure of these cysts ranges them in the category of dermoid cysts—i. e., of those which we regard as originating from an invagination of the external germinal layer, which then develops afterward into a cyst (Remak). Some of the cysts with serous contents may perhaps have originated from an analogous invagination of the nasal mucous membrane (Panas). Dermoid cysts should not be confounded with dermoids of the corneal margin. The two varieties of tumor differ essentially in structure (see § 511), and clinically in the fact that the first are deeply placed cavities, the second, superficially situated, flat, expanded, wart-like structures.

Another form of tumor with which dermoid cysts might be occasionally confounded is the *hernia cerebri* (cephalocele). That form of it which requires consideration here, the cephalocele orbitæ anterior, projects into the orbit between the ethmoid and frontal bones (Fig. 386). At this spot the dura is wanting in the space occupied by the hernia, being adherent to the periosteum at the margin of the gap in the bone. The sac protruding from the gap is formed of arachnoid and pia mater, and contains remains of brain substance or, when the ventricle extends as far as the sac, is lined with ciliated epithelium (Stadfelt). Clinically, therefore, the cephalocele of the orbit usually represents a tumor which is situated in the upper and inner angle of the

orbit, is covered by normal skin, is distinctly fluctant, and has existed since birth. Since dermoid cysts also are congenital and frequently occupy the same spot, an error in diagnosis might be readily made. Such an error might possibly lead to the worst consequences, if extirpation of the meningocele, which may be followed by meningitis should be thereupon performed. It is hence important to know how such a mistake can be avoided. The signs which principally distinguish a cephalocele from the dermoid cysts are as follows: 1. A meningocele is immovably attached to the bone. Not infrequently we are able to feel with our finger the opening in the bone through which the cephalocele communicates with the cranial cavity (hernial orifice). 2. A cephalocele sometimes shows the pulsatory and respiratory oscillations which are communicated to it from the brain. 3. A cephalocele can be diminished in size by pressure with the fingers, since its fluid contents are in part pushed back into the cranial cavity. At the same time symptoms of increased cerebral pressure, like vertigo, nausea, deviation of the eyes, convulsions, etc., may make their appearance. 4. In order to be perfectly certain, we make an exploratory puncture of the cyst. In doing this we must proceed under rigid aseptic precautions, so as not to excite inflammation of the cyst and consequent meningitis. The diagnosis becomes more difficult, or is even impossible, when the communication between the meningocele and the cranial cavity (the subdural space) is obliterated; but in this case removal of the tumor is not associated with danger of any sort.

Besides those already given, the only *cysts* of the orbit requiring mention are those formed by *entozoa* (cysticercus and echinococcus), the *congenital cysts* of the lower lid in microphthalmus (page 729) and *blood-cysts* developing from extravasations of blood.

880. Vascular Tumors.—To vascular tumors, in the widest sense, belong vascular dilatations (aneurysms) and new growths consisting of vessels (angiomata). Both the one and the other occur, though rarely, in the orbit. Of hæmangiomata we here meet with two forms that also occur in the lids (*telangiectasis* and cavernous tumor). The former is congenital, and is originally seated in the lids, from which it may gradually extend into the orbit. The diagnosis, hence, is easy, since on the lids the tumor is obvious. Its treatment when in the orbit is the same as when on the lids. *Cavernous tumors*, in contradistinction to those just named, usually develop first within the orbit and grow slowly, pushing the eye farther and farther before them. As long as they lie wholly in the depth of the orbit, the correct diagnosis can be made only from the variable volume which these tumors possess. We can diminish their size by pressing the eye back into the orbit, and, on the other hand, they swell in the acts of crying, straining, etc., or when the patient bends far over. They may also enlarge during the menstrual period (Birch-Hirschfeld). When the vascular tumors grow big and extend farther forward they gleam with bluish lustre through the skin of the lids, and dilated blood-vessels appear in the latter; in this case the diagnosis is, of course, easy. When we see that these tumors are endangering the eye by causing its progressive protrusion, we must remove them. Extirpation with the knife is principally adapted to those cases in which the tumor is sharply limited and is inclosed in a fibrous capsule; for the case of a more diffuse vascular tumor electrolytic treatment is indicated (see § 922).

881. Pulsating Exophthalmus.—Under this term is denoted the following complex of symptoms: The eye is protruded; the blood-vessels of the conjunctiva and the lids and often of the surrounding parts, too, are dilated. If the hand is placed upon the tumor, distinct pulsation of the eyeball itself and of the surrounding parts is felt; and if the ear is applied to it, blowing murmurs and a continuous whi ring and rumbling

sound are heard. The patient also hears the same sounds; he has a constant rumbling in his head, as if he were standing near a waterfall, and he is often more disturbed by this than by anything else. The eye can be pushed back into the orbit with the hand. A special feature distinguishing the disease is the fact that compression of the carotid of the same side as the exophthalmus diminishes both the pulsation and the sounds or causes them to disappear altogether. The vision is in many cases impaired or abolished, and that, as the ophthalmoscope shows, by optic neuritis; a conspicuous feature that is brought to light at the same time being the enormous dilatation of the retinal vessels. At times there are violent pains in the orbit and impairment of the hearing. There is not infrequently paralysis of the third and sixth nerves.

It has been proved by a number of autopsies that the most frequent cause of this complex of symptoms is an arterio-venous aneurysm resulting from rupture of the carotid into the cavernous sinus (cf. Fig. 142). Through the spot where this rupture has taken place the blood of the carotid is discharged under a high pressure into the cavernous sinus and the veins of the orbit, which empty into it, so that these veins are very greatly dilated and are set pulsating. The rupture of the carotid is most frequently caused by traumatism, and particularly by severe injuries of the skull with fracture of the base; rarely a spontaneous rupture occurs from degeneration of the vessel wall.

In rare cases the aneurysm subsides spontaneously; otherwise it persists, and may produce death with cerebral symptoms or by hæmorrhage from the dilated vessels. The treatment is self-evident, from the fact that the symptoms disappear as soon as the carotid is compressed upon the affected side. Hence we try first compression by digital or instrumental pressure upon the carotid every day for as long a time as can be borne. This is combined with rest in bed and the use of the iodides. If this procedure after being continued for some time, is unsuccessful, ligation of the carotid is indicated—an operation by which many cases are relieved at least for a time, although death or blindness may occur within a comparatively short time after operation. Ligation of the superior or inferior ophthalmic vein or of the angular vein is comparatively harmless and quite frequently successful.

There are cases of *intermittent exophthalmus*, which appear only at intervals, and in fact mainly when the head is depressed, while in the erect position some enophthalmus may be present. As in such cases the exophthalmus increases when pressure is made upon the jugular vein, and as sometimes dilated veins are visible even upon external examination, it is assumed that the exophthalmus is caused by varicose veins in the orbit, which, however, in contradistinction to what occurs in pulsating exophthalmus, are not in communication with an artery.

882. Other Tumors.—Besides the tumors enumerated above, the following have been observed in the orbit, as rarities: Teratoma, angioma lipomatodes, lymphangioma, neuroma simplex, neuroma plexiforme, leiomyoma, cylindroma, endothelioma, psammoma, enchondroma, and osteoma. The *osteomata* start from the bony wall of the orbit, and most often from the frontal bone. They may, however, also and, according to some most frequently, develop in one of the cavities adjoining the orbit, particularly the frontal sinus, and enter the orbit itself afterward through erosion of the orbital wall. They are usually sessile, being attached to the bone by a broad base, but rarely are pediculated, in which case it occasionally happens that they necrose spontaneously and are then eliminated. Most osteomata are as hard as ivory, so that chisel and saw can make scarcely an impression on them (exostosis eburnea); but there are also osteomata of spongy or partly cartilaginous structure. They grow very slowly, and in time displace the eye from the orbit and destroy the sight by compressing the optic nerve. They frequently also cause death. For this reason they call for removal by operation, which because of the great hardness of the growth is often very difficult, and, moreover, is not devoid of danger since in its performance the cranial cavity must be opened. Hence we often abstain from taking the growth out radically, and satisfy ourselves with removing only so much of it as projects into the orbit. When the eye has been pushed out of the orbit and rendered blind by a large osteoma, it is sometimes better to relieve the

patient of his troubles by enucleating the useless eye, rather than expose him to the dangers of an extirpation of the growth.

An enlargement of the lacrimal and parotid glands on both sides occurs in Mikulicz's disease (see § 525).

883. Malignant Tumors.—The most common primary tumors of the orbit are sarcomata. These may start from bone, periosteum, orbital muscles or connective tissue, the lacrimal gland, and even the optic nerve and its sheaths. (For the special features of these latter, see page 775). Orbital sarcomata are usually rounded, pretty soft, and sharply defined, because they are inclosed in an envelope of connective tissue. Primary carcinomata of the orbit are very rare; they spring from the lacrimal gland.

The secondary formation of tumors in the orbit occurs when tumors of the eye (sarcomata, gliomata) perforate posteriorly into the orbit. So too, carcinomata of the lids or conjunctiva, if not removed early, grow into the orbit. Neoplasms may also grow into the orbit from the neighboring accessory nasal sinuses or even from the cranial cavity. Finally metastases of malignant tumors occur—rarely, however—in the orbit.

Tumors, usually bilateral, and running a malignant course are lymphosarcomata, the leucæmic and pseudoleucæmic tumors, and the *chloromata.* The last-named are greenish tumors occuring in young persons, especially young children, marked by early blood changes (diminution of erythrocytes, increase of leucocytes with diminution of the polymorphonuclear cells and increase of myelocytes) and by a rapidly fatal course with metastases in various organs. Benzol internally and x-ray and radium have been tried (Bedell).

If malignant tumors are not removed early, they push the eye more and more out of the orbit, afterward destroy it, and finally fill the entire orbit, from whose anterior opening they project as large, ulcerated, readily bleeding masses. Still later they pass over to structures adjacent to the orbit, and especially to the brain; the neighboring lymphatic glands swell up, and metastases form in the internal organs. The patient succumbs from exhaustion or from a transmission of the growth to vital organs. To this course a stop can be put only by as early and as radical a removal of the growth as possible. Small encapsulated sarcomata can be cleanly enucleated with preservation of the rest of the contents of the orbit. Large tumors, particularly if they are not sharply circumscribed, demand the removal of the entire contents of the orbit, in doing which the eye, even when it still retains its ability to see, must be sacrificed. After removal a prophylactic application of radium may be made. (For methods of operating see §§ 998, 999.)

884. Congenital Anomalies of the Orbit.—Congenital *cysts* include the dermoid cysts (§ 879) and the cysts found in connection with microphthalmus (page 729).

Cyclopia is a condition in which the two orbits and their contents are replaced by a single orbit and a single median eye. The anomaly is probably occasioned not by a fusion of two embryonic organs, but by a failure of a primitive single organ to differentiate (Stockard, Hill). The anomaly seems to be due to toxic influences.

The eye may be absent altogether (*anophthalmus*) or be quite rudimentary in development (*microphthalmus*) (cf. page 729).

V. Disease of the Cavities Adjoining the Orbit

885. Character.—The accessory cavities of the nose, namely the maxillary antrum, the frontal sinus, the ethmoid cells, and the sphenoid sinus, may implicate in their diseases the orbit and the optic nerve. This is most likely to occur in disease emanating from the ethmoid cells, for the latter are separated from the orbit only by the thin lamina papyracea which, more-

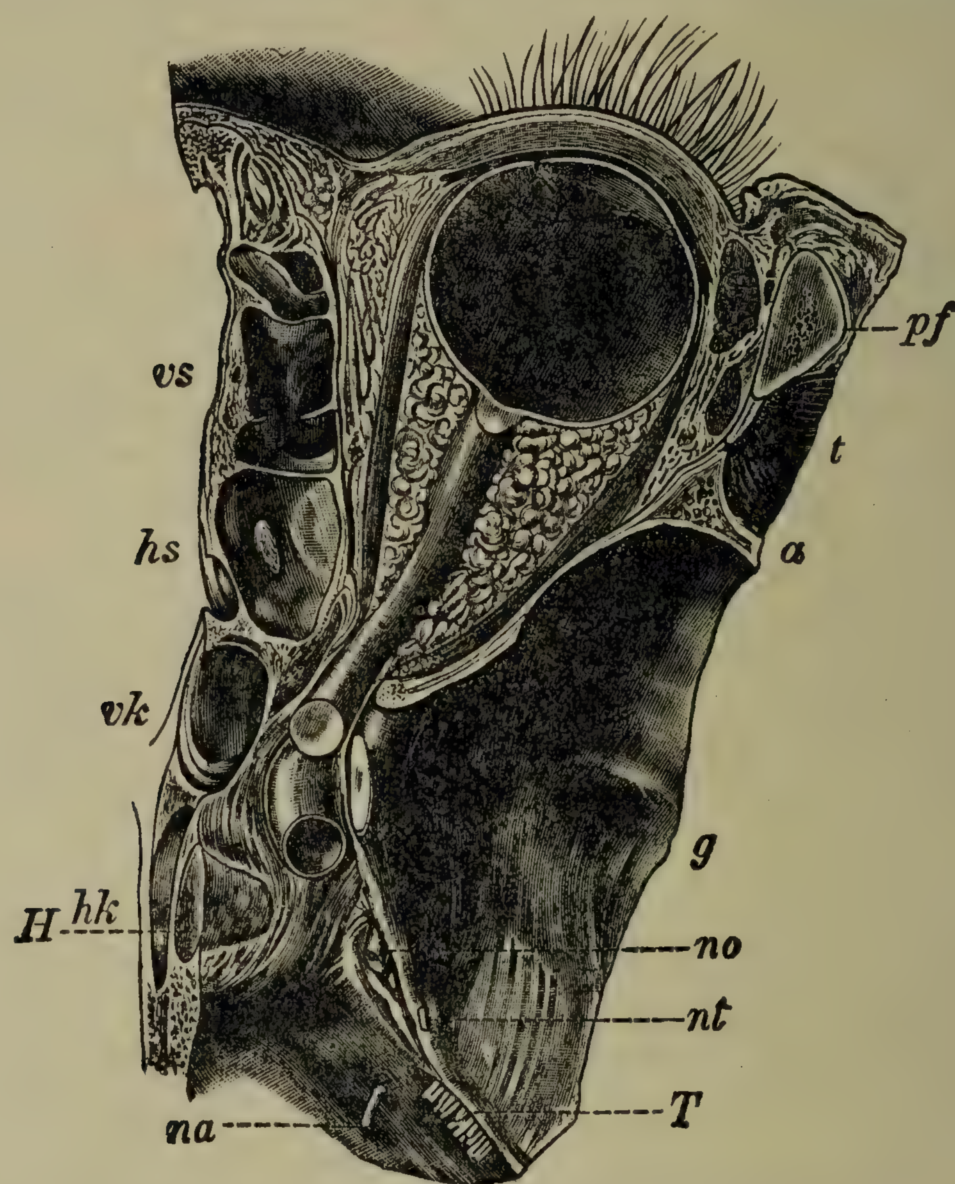


Fig. 387.—Horizontal Section Through the Right Orbit.

Adjoining the internal wall of the orbit and separated from it by the lamina papyracea are the cells of the anterior and posterior labyrinth of the ethmoid bone, *vs* and *hs*. Right behind this is the cavity of the sphenoid bone, which in this case is divided into two segments by a projecting ridge of bone. The anterior division, *vk*, adjoins the optic canal at the inner side of the latter, and also extends beneath it. Over the posterior division, *hk*, lies the hypophysis, *H*, which fills up the sella turcica, and upon which rests the chiasm, which in this preparation has been removed along with the brain. At the outer side of the orbit, between the eyeball and the frontal process of the zygoma, *pf*, are two lobes of the lacrimal gland, the lower border of which was just met by the section. Behind the frontal process of the zygoma in the temporal fossa is the cross section of the temporal muscle, *t*, and behind this the cross section of the great wing of the sphenoid, *a*, succeeding which is the middle fossa of the skull, *g*. At the internal and posterior border of the latter the tentorium was detached along its insertion when the brain was taken out. Along the site of this insertion the nerves of the eye muscles pass through the dura, the furthest forward being the oculomotorius, *no*, while the trochlearis, *nt*, is somewhat further back. To the inner side of the latter and hence within the posterior fossa of the skull, the abducens, *na*, goes down into the dura. Furthest back is the trunk of the trigeminus, *T*, passing from the cranial cavity through a slit, which lies between the upper edge of the pyramid of the temporal bone and the attachment of the tentorium. The optic nerve where it passes from the optic canal into the cranial cavity has been cut off so that the internal carotid which lies beneath it is visible.

over, sometimes has gaps in it. The most posterior ethmoid cell not infrequently extends into the small wing of the sphenoid and then comes into closer relation with the optic nerve because it now borders on the optic canal; and it may even extend so far beyond the median line that it actually

adjoins the optic canal of the opposite side. Otherwise the wall of the optic canal is formed in part by the sphenoid sinus (Fig. 387, *vk*) and involvement of this not infrequently affects the eye.

Inflammatory infections of the above named cavities, called by the name of *sinuitis*, or less properly sinusitis, are generally the result of an inflammation of the nasal mucous membrane. This may be either a catarrhal inflammation (coryza) or produced by infectious diseases, whether acute (the most frequent example of this being influenza) or chronic (tuberculosis, syphilis). Furthermore, injuries, new growths, and, in the case of the maxillary antrum, diseased teeth may cause the inflammation. The inflammation is associated with purulent secretion or empyema (called open empyema if the secretion can escape through the natural orifice of the cavity, closed empyema if the ostium of the cavity is shut off). In the latter case the secretion accumulates in the cavity and distends it; at the same time the contents of the cavity which at first are purulent gradually become mucous and at length watery (dropsy of the cavity). In infants in whom there is no maxillary antrum to speak of, acute osteomyelitis of the lower jaw may set up inflammations about the eye often with exophthalmus (Marx).

Inflammation of the accessory cavities often produces *symptoms* which are very striking. If for instance, after a coryza there is marked frontal headache and the forehead above the eyebrows is sensitive to percussion, or the skin in this region actually becomes somewhat swollen, we should at once think of an inflammation of the frontal sinus. But often the inflammation of the accessory cavities runs a course which is pretty much void of symptoms, and particularly so when it is one of the deep seated cavities that is concerned. Hence a diagnosis to be certain always requires a rhinoscopic examination. This often requires some preliminary operative procedure (removal of some portions of the turbinal bodies, opening of the wall of the sinuses). At the same time x-ray plates of the sinuses should be taken.

886. Transfer to Orbit.—The inflammation of the cavities may at any stage pass over to the orbit. In the *acute* stage an orbital cellulitis (see § 869) most frequently develops.

With marked headache and some fever the lids and the conjunctiva swell up, the eye protrudes and becomes less movable, and frequently there is added a paralysis of the eye muscles or an inflammation of the optic nerve. These threatening symptoms soon abate, but the paralysis of the eye muscles or the inflammatory atrophy of the optic nerve may remain permanently. A rarer sequel of such accumulations of pus in the accessory sinuses is suppuration in the orbit occurring under the guise either of subperiosteal abscess or of a retrobulbar phlegmon with its destructive consequences. Acute coryza with accumulation of secretion especially in the ethmoid cells may occasion an acute retrobulbar neuritis with suddenly developing temporary or sometimes permanent blindness (see § 815).

Chronic empyemata of the accessory cavities may produce sluggish inflammations of the orbital structures, and, since such empyemata often run their course without causing any symptoms, the diagnosis sometimes encounters great difficulties.

Dropsy of the accessory cavities is not so apt to produce inflammation of the orbit as it is to cause by distention of the walls of the cavities an encroachment on the orbit and hence a displacement of the eyeball.

Thus in dilatation of the frontal sinus we see the frontal bone protruding above the margin of the orbit and find the eyeball displaced forward and downward. In distention of the ethmoid cells we get a protrusion of the inner wall of the orbit upon which we can feel through the soft parts the thin lamina papyracea crepitate under the pressure of the fingers.

In not a few cases disease of the optic nerve and uveitis may be produced not by pressure but by absorption of *toxic* matter from a chronically inflamed sinus. In such cases we may find the causal organism in the blood, or complement-fixation tests may indicate its activity.

Dilatation of the accessory cavities may also be produced by new growths like polypi, osteomata, or malignant neoplasms.

Chronic disease of the sinuses (especially of the sphenoidal sinus) may produce inflammation or atrophy of the *optic nerve*. The first evidence of this is often an enlargement of the blind spot (Van Der Hoeve's symptom, § 815). This may be accompanied or followed by the development of a central scotoma, at first relative, later absolute. Such a scotoma indicates the onset of a chronic retrobulbar neuritis with the usual primary implication of the papillo-macular bundle. With these visual symptoms there may be moderate pain in the depth of the orbit or supraorbital neuralgia. By progressive enlargement of the scotoma partial or complete blindness may be produced, with a corresponding atrophy of the nerve.

In explaining these symptoms we must recollect that in the optic canal the dural sheath of the optic nerve is transformed into the periosteum of the canal. The nerve, therefore, is here enveloped only by the delicate pial sheath which in most places is applied to the periosteum and on the upper side is actually adherent to it. This anatomical relation facilitates the direct transfer of inflammation from the cavities in the vicinity of the canal to the optic nerve itself. This inflammation first affects the pial sheath of the nerve, and we should therefore expect that the peripheral bundles which are next to the sheath and which are thought to supply the periphery of the retina would be first diseased. But the first part actually affected is regularly the papillo-macular bundle which here lies in the centre of the nerve. We explain this fact by assuming that the fibres of this bundle are particularly vulnerable, and connect this vulnerability with its especially exquisite and delicate function.

The supra-orbital neuralgia occasionally observed is attributable to pressure or irritation exerted on the first branch of the trigeminus which runs close to the lateral surface of the body of the sphenoid.

887. Treatment.—The sinuses involved must be washed out or drained and otherwise adequately treated. If there is toxæmia from absorption of bacterial matter from the sinus, it may be necessary to supplement this local treatment by the use of vaccines.

PART VI

OPERATIONS ON THE EYE

OPERATIONS

CHAPTER XXXII

EYE OPERATIONS IN GENERAL

888. Preparation of the Patient for Operation.—In anticipation of any serious operation, the patient should be put in the best possible *physical condition.* Any source of possible infection in the teeth, tonsils, accessory sinuses or digestive tract should be looked for, and the condition appropriately treated. The urine should be examined, particularly for sugar, and if the latter is present treatment instituted to remedy as far as possible the diabetic condition before venturing an operation. In patients with a gouty tendency pre-operative treatment with cincophene diminishes the likelihood of post-operative irido-cyclitis (Wilmer). In cases in which there is reason to anticipate post-operative hæmorrhage (see § 898), either because of high blood pressure or because an expulsive hæmorrhage has already taken place in one eye, venesection with free catharsis before the operation (Maddox) and the performance of the operation itself in the semi-recumbent instead of a flat posture (Fuchs) are measures that will probably avert the danger. Calcium lactate (gm. 0.20 three times a day), may also be given to reduce a tendency to hæmorrhage (Wilmer). Before all serious operations the bowels should be thoroughly emptied. Nervous patients may require to be quieted with bromides combined, if necessary, with a hypodermic of morphine. But the latter must not be given without determining the patient's idiosyncrasy as in some cases it causes nausea and restlessness.

We must make sure also that the patient's *conjunctival sac* is in proper condition for the operation, i. e., that it does not harbor virulent germs that can not be satisfactorily removed or neutralized by the usual pre-operative cleansing. In all cases, therefore, in which the eyeball is to be opened we should take smears and cultures from the conjunctival sac, and if dangerous germs (particularly streptococci and pneumococci) are found should postpone operation until these germs have disappeared.

To effect this, Elschnig applies mercury oxycyanide every half hour, and if this does not suffice applies one-per-cent nitrate of silver once a day. Others apply a bandage before operating, and if this causes secretion to develop in the eye, postpone operation until the conjunctiva is in healthier condition.

It is particularly important not to operate in the presence of disease of the *lacrimal sac*. If such disease is present, the sac should be extirpated one or two weeks before the operation. If this cannot be done or if in old dacryocystitis there is no abnormal secretion from the sac we may seal the canaliculi by searing them with the galvano-cautery or by ligating them (§ 907). These latter precautions may also be taken if the nose presents any condition (ozæna, ethmoiditis) likely to give rise to infection (Lancaster).

Artificial Pre-operative Leucocytosis.—In the belief that post-operative infection can be prevented if before operation we excite active leucocytosis with consequent formation of protective bodies, Bell, two hours before the operation, drops a 1-per-cent solution of silver nitrate into each eye. This produces an intense irritation so that when the patient comes to the operating table the eye looks red and inflamed and there is a moderate secretion of mucus. This is washed out with normal salt solution. This procedure of Bell's is analogous to the practice of those general surgeons who by preference operate on an appendicitis in the acute stage rather than wait for the inflammation to subside. The good results obtained by this practice in eye operations seem well substantiated by statistics, and the translator believes that it is based on sound theory.

889. Antisepsis and Asepsis.—As in other operations, so in operations on the eye and its adnexa, we must use antiseptic precautions in preparing for them and maintain rigid asepsis throughout their performance and during the after-treatment.

In this regard we must distinguish between operations in which the eyeball itself is opened and those in which simply the conjunctiva or Tenon's capsule is divided. Operations of the former kind require the strictest precautions; in those of the second class, provided the rules of ordinary cleanliness are observed, infection practically never occurs. In the pre-aseptic days the translator observed a series of several hundred such operations done on office patients with no precautions whatever, and in not a single case was there any inflammatory reaction.

890. Preparations Regarding the Operator and the Instruments.—In eye operations the hands of the operator rarely come into contact with the wound; nevertheless, they should be very thoroughly cleansed and then disinfected with a solution of corrosive sublimate (1:2000). To avoid infection by drops falling from the mouth, the operator and his assistants should wear a wire frame covered with sterile gauze in front of the mouth and for a like reason should have the hair covered with a sterile cap and wear sterile gowns. Even more important is the disinfection of the instruments. This is best effected by boiling in a 1-per-cent solution of sodium carbonate in which they do not rust. On removal from this solution they should be passed through sterile water or wiped off to remove the excess of alkali. For minor operations (removal of foreign bodies from the cornea, removal of chalazia) instruments may be sterilized by passing them through an alcohol flame or by immersion in alcohol.

891. Preparation of the Field of Operation.—To prevent infection of the wound by the adjacent parts, the vicinity of the eye and especially the skin of the lids and the edges of the lids should be thoroughly washed with soap and sponged off afterward with a 1:5000 bichloride solution. The lids are then everted, and the conjunctival sac and the exposed tarsal surfaces are thoroughly irrigated with either normal salt solution or with a bichloride solution. The exposed tarsal surfaces and the edges of the lids are gently wiped off with a cotton swab dipped in the cleansing solution. The field of operation is then shut off from the adjoining parts (eyebrow, nose, mouth) by a gauze pad with a hole cut in just big enough to allow of the surgeon's manipulations.

There is much difference of opinion as to the respective advantages of irrigation with a simple detergent solution (normal salt) and solutions of bichloride. In a thoroughgoing

way the latter is applied by Herbert and Elliot. Fifteen minutes before operating they irrigate the everted lids with 1:3000 mercury bichloride solution delivered from a container three feet above the patient's head, and at the same time move the lids vertically, so that the solution shall reach all the folds of the conjunctiva. This irrigation is done for a minute at least and may be repeated if the conjunctiva does not give a satisfactory reaction (secretion of grayish mucus, cloudiness of membrane). Then epinephrine (adrenaline) and cocaine are instilled, and the eye is irrigated with normal salt solution. In addition Elliot wipes off the conjunctiva with sterile cotton. He and other operators also trim off the lashes, because it is practically impossible to sterilize these, and they may come into contact with the instruments during the operation. It can not be denied that, so far as the prevention of infection is concerned, the method of Herbert and Elliot greatly surpasses the less energetic procedures. In a series of several thousand cases thus operated on not one case of suppuration and very few cases of non-suppurative inflammation occurred (Lancaster).

As the conjunctiva even after careful cleansing often still contains germs, we might think that the infection of recent wounds would necessarily be very frequent. Fortunately, however, at the present time infection of wounds is only of exceptional occurrence, so that evidently the germs of the conjunctival sac are not to be greatly dreaded (cf. § 94).

In operations performed through the skin (operations on the lids, dacryocystectomy, tenotomy of the inferior oblique) it is sufficient to paint the field of operation with tincture of iodine without preliminary washing.

892. Posture of Patient; Illumination.—Operations are usually done with the patient in the recumbent position. When there is reason to apprehend post-operative hæmorrhage the semi-recumbent or sitting position is better, and some operators of great experience have preferred to operate in this way generally.

Usually a good daylight is the most satisfactory illumination. For certain exigencies and regularly in certain operations like discission of the lens capsule a strong artificial light concentrated on the spot by a condensing lens held by an assistant is required.

893. Anæsthesia.—To effect local anæsthesia a 4-per-cent solution of cocaine is instilled several times into the conjunctival sac at intervals of one or two minutes. The solution should be freshly prepared and should be sterilized by boiling. Cocaine dries the cornea by diminishing the frequency of winking. Hence, we direct the patient to keep the eyes shut after the instillation and from time to time during the operation we irrigate the eye with normal salt solution, so as to moisten the surface and wash off impurities. But apart from its drying effect, cocaine directly injures the epithelial cells of the cornea, and hence, should never be used in great excess (§ 567) Accordingly, in cases in which we have to use an anæsthetic repeatedly and also in cases in which we wish to avoid mydriasis, we resort to *holocaine* (phenacaine) which does not affect the pupil and affects the cornea to a less degree than cocaine. Holocaine in 1-per-cent solution is the best agent for producing surface anæsthesia as in removing foreign bodies from the cornea, cauterizing or incising corneal ulcers, applying the tonometer, etc. Cocaine anæsthesia begins to take effect in the conjunctiva and cornea in a few minutes and in these parts its action persists for ten minutes or more. It affects the deeper parts later, and hence in operations like iridectomy the excision of the iris is best put off

till about fifteen minutes after the first cocaine instillation. Some think that holocaine in 2-per-cent solution acts better as an anæsthetic for these deeper parts. But in operations involving the deeper parts—operations on the eye muscles and iridectomy or trephining—more profound anæsthesia is secured by *subconjunctival injections* of a 1 or 2-per-cent solution of *procaine* (some prefer for this purpose a 1-per-cent of cocaine even though the latter occasionally produces toxic symptoms). Or after the wound has been made, cocaine may be dropped directly on the still sensitive deeper parts (iris, eye muscles). Before cataract operations procaine may be injected about the orbicularis to induce nerve-block and so prevent a disastrous squeezing (Barraquer). In performing enucleation, procaine is injected into the neighborhood of the ciliary ganglion through a long cannula plunged 4.5 cm. in, at the temporal side of the optic nerve and nearly to the apex of the orbit. In operations performed through the skin, (e. g. in lid operations, dacryocystectomy, etc.) procaine injections may also be used, but a better method is the *infiltration anæsthesia* of Schleich. In this a 0.2-per-cent. solution of sodium chloride to which 1:10,000 or 2:10,000 of cocaine are added, is injected intra-cutaneously, thus raising a bleb within the limits of which there is anæsthesia. This anæsthetic area may be enlarged by repeating the injections at different points of its periphery. Infiltration anæsthesia in the course of the retrotarsal fold renders operation on the tarsus, e. g., the expression of trachoma, painless (Guttmann).

To solutions used for subconjunctival or subcutaneous injection a little *adrenaline* may advantageously be added, since it diminishes the tendency to toxic effect. Adrenaline is also used if we have to operate on highly injected eyes. If these are previously blanched by adrenaline the cocaine acts better and the congesting effect of holocaine is offset. Adrenaline, moreover, is a useful adjunct to cocaine in muscle operations, since here it checks the bleeding which in the tiny field of operation interferes greatly with the view. On the other hand, it is better not to use it in operating on a pterygium or other vascular growths, which may be so contracted by it that the normal outlines are obscured. In case of very great sensitiveness of the eyeball (severe irido-cyclitis, acute glaucoma) cocaine even with the assistance of adrenaline cannot make the eye insensitive enough for security in the performance of the operation. In that case *general narcosis* with chloroform or ether is requisite, and it is likewise usually employed in major operations such as enucleation, etc., and for children. Even in major operations it can generally be replaced by local anæsthesia, properly applied.

894. Fixation of Eye.—In regard to *operations upon the eyeball itself* the following principles hold good:

The separation of the lids is effected by means of lid specula (blepharostats or elevators). There are some which hold both lids open at once, keeping them apart by the elastic force of a spring (spring specula; Fig. 388, *a*, *b*, *c*); others which are designed for one lid only and must be held with the hand (Desmarre's lid retractor, Fig. 388, *d*). The eyeball itself is fixed by grasping a fold of conjunctiva and underlying tissue close to the

corneal margin with a toothed forceps (Waldau's fixation forceps, Fig. 388, *e*) and holding it in place. In most operations (iridectomy, cataract extraction, muscle operations) the fixing forceps is applied at a point of the circumference of the cornea opposite to the operation wound; but it may be placed elsewhere according to circumstances. The conjunctiva even near the cornea sometimes tears out or does not afford a good hold. Hence, Angelucci followed by many operators fixes and controls the eyeball by firmly grasping the tendon of the superior rectus with the fixation forceps. Elsch-

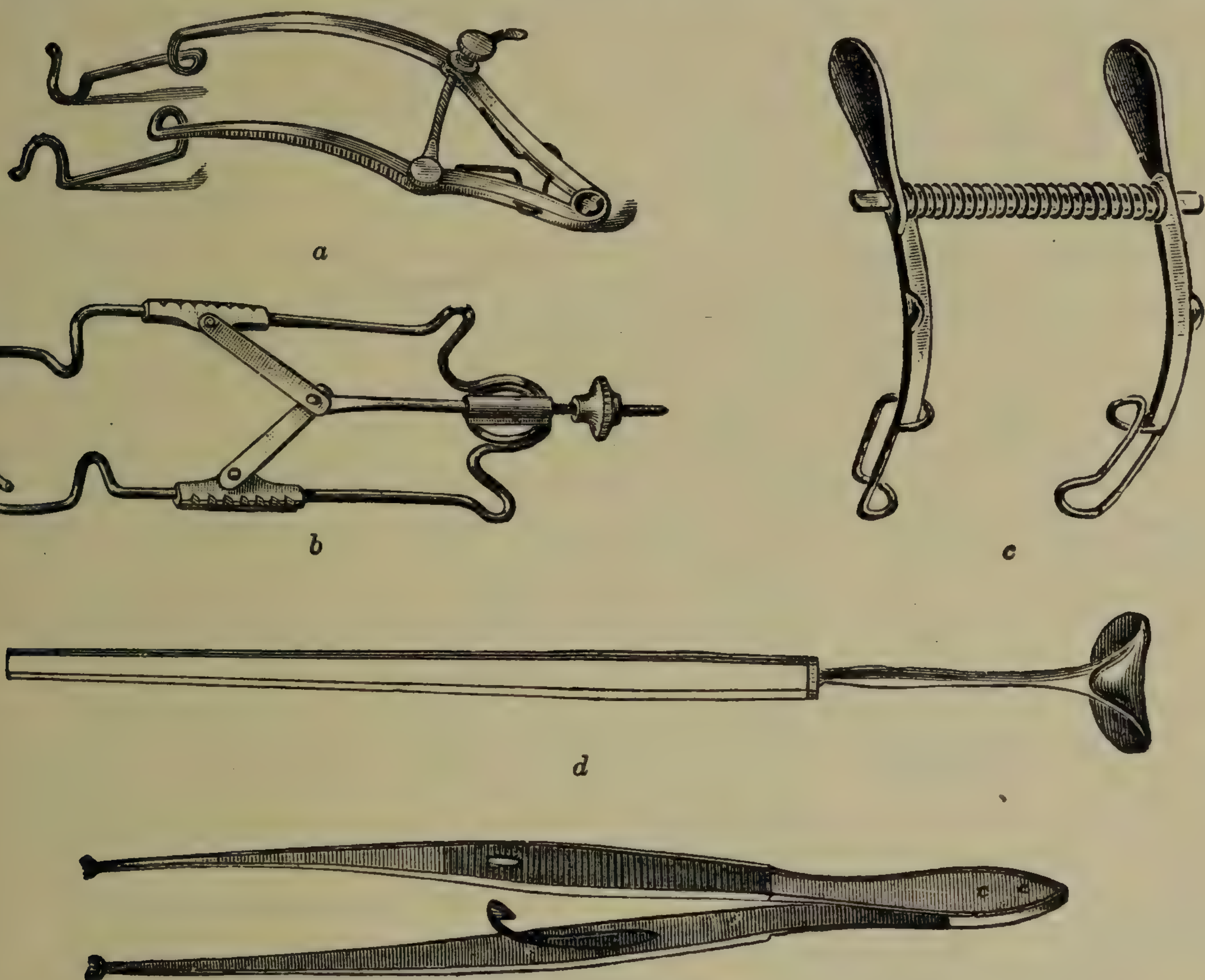


FIG. 388.—INSTRUMENTS FOR OPENING THE EYE AND KEEPING IT IN PLACE.

a, *b*, *c*, various forms of spring specula for holding the eye open in operations. *d*, Desmarre's elevator or lid retractor, for holding the lids apart in examinations of the eye and in operations. *e*, fixation forceps for holding the eyeball steady in operations.

nig, Maddox, and others pass a stitch through the tendon of the superior rectus, and control the eye with this.

Since the metal arms of the speculum exert a pressure on the lids that is unpleasant to the patient, we may, if we have a skilled assistant, get him to hold the lids apart with his fingers. This or the use of the lid retractor is particularly advisable in cases in which success depends on our not exerting any pressure on the eyeball (e. g., in order to avoid escape of vitreous), and we then too abstain as far as possible from grasping the eyeball with the fixation forceps.

895. Incisions.—The incision which lays the eyeball open is made, as a rule, within the limits of the anterior chamber. As this latter is bounded by the cornea and at its periphery by the most anterior portion of the sclera, the section may lie either in the cornea or in the sclera. We therefore distinguish sections with respect to—

(*a*) Their *position*, into corneal and scleral. These differ from each other mainly in the following points: 1. In scleral incisions there is more tendency to prolapse of the iris than in incisions of the cornea (for explanation of this see § 896). 2. The sclera is covered by conjunctiva, and a wound in the sclera can therefore be provided with a conjunctival flap—a thing which is not usually possible in corneal sections. 3. Scleral wounds are less apt to become infected than those in the cornea, because of the slighter tendency that the sclera exhibits toward purulent inflammations. Hence, before the introduction of antiseptic methods, scleral incisions gave better results than did those in the cornea. At present, when infection is avoided in every possible way, this distinction is no longer of so very much weight.

(*b*) In *shape*, incisions may be linear or curved. The former lie in a great circle of a sphere, and hence form upon the surface of the eyeball a line which is the shortest that can be made to connect the terminal points of the section (*eaf*, Fig. 389). The curved or flap incisions correspond to a small circle of a sphere. Between the largest curved section (*edf*, Fig. 389) and the linear section an infinite number of sections (*ecf*, *ebf*) may be conceived to exist, constituting the transition forms between the two. These are curved sections of varying altitude; the linear section forms their inferior limit, and may therefore be regarded as a curved section whose altitude = 0. Most of the sections in general use are curved ones, with an arch of greater or less altitude. An example of a pure linear section is the Guthrie-Saemisch incision (§ 941). A flap incision, having the ends of its section at the same distance apart as a linear incision, will make a much longer wound than the latter, and, by the lifting up of the flap, the wound can be made to gape more.

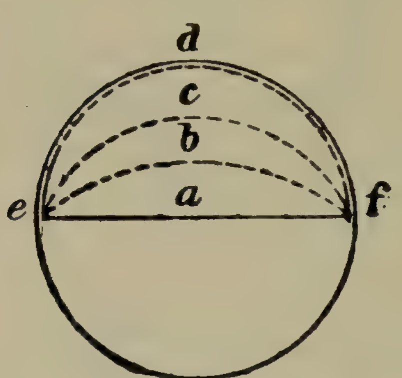


FIG. 389.—DIFFERENT FORMS OF SECTIONS IN THE CORNEA.

In *making* the section, care must be taken that the knife is withdrawn from the wound slowly, so that the aqueous may escape as gradually as possible. In this way we avoid the evil results which too rapid escape of the aqueous often entails, such as extensive prolapse of the iris, subluxation of the lens, prolapse of the vitreous, and intra-ocular hæmorrhage. Slow escape of the aqueous is particularly important if an operation is done when there is increase of tension.

In judging of the size and position of any particular form of section, not only the *outer and visible wound* but also the *internal* one must be taken into consideration. That the latter is of a different size, shape, and position from the external wound arises from the fact that in most methods of performing the section the knife divides the tunics of the eye obliquely (Fig. 395, *I* and *P*). This is particularly the case with the wounds made by the keratome. Even if at first we plunge the keratome in perpendicularly, we must still, as soon as its point has entered the anterior chamber, change its position so that it shall be pushed along parallel with the iris, as otherwise we should get into the iris and lens. The inner orifice of the section (Fig. 436, *i i*) therefore lies nearer the centre of the cornea than does its outer orifice (Fig. 436, *a a*). For this reason sections whose outer orifice lies in the sclera, and which hence are usually regarded as

scleral sections, nevertheless by their inner segment belong to the cornea (*I*, Fig. 395). A further fact contributing to this relation between the external and internal sections is that the sclera overlaps the cornea externally, and the latter therefore in its inner layers extends farther toward the periphery than can be seen from the outside. Hence, even sections that are fairly perpendicular, like those made in performing cataract extraction with a Graefe knife (Fig. 279), are in great part located in corneal tissue. This fact that the internal wound is less peripherally situated than the external, must be taken into account in making the section. If, for example, we desire to excise the iris up to a certain spot, we must not perforate the cornea at a point directly opposite the latter, but must place the corneal section farther toward the periphery, so that the internal wound may lie at the spot at which the iris is to be cut off.

The internal wound is also of less length than the external (Fig. 436, *a a* and *i i*). This fact also must be taken into consideration, particularly in cataract operations, in which care must be taken that not only the external but also the internal wound shall be large enough to allow the cataract to pass through.

The *oblique course* of the wound through the tunics of the eye, furthermore, affects the tendency of the wound to gape. It was said above that flap wounds gape more than linear wounds; but the tendency toward gaping depends still more upon whether the wound traverses the tunics of the eye perpendicularly or obliquely. The former is more particularly the case in sections made with Graefe's knife, in which the knife passes through the cornea or sclera from within outward; the latter is the case in sections made with the lance knife. Sections of the former sort gape, owing to the elastic retraction of the edges of the wound. Wounds, on the other hand, which are made with the lance knife, and pass obliquely through the tunics of the eye, do not gape, because the lips of the wound close upon one another like a valve. The closure is effected by the intra-ocular pressure. This latter is exerted to the same extent on every point of the internal surface of the eyeball. It presses as strongly upon the posterior lip of the wound (*a*, Fig. 395) as upon the anterior (*b*), and hence pushes the former against the latter. To this valve-like closure of the wound is to be attributed the fact that the aqueous does not escape after paracentesis of the cornea if the lance knife is withdrawn from the wound cautiously, and without pressure or rotation. The wound must be made to gape before the aqueous (or, in simple linear extraction the soft masses of lens matter) can be expelled. It would be a mistake to try to effect this by pressure either upon the centre of the cornea or upon the sclera, as in so doing we should only increase the intra-ocular pressure by that which is exerted from the outside, and thus push the posterior lip of the wound still more strongly against the anterior. Only upon very strong pressure, by which the lips of the wound would be shoved past one another, would the wound gape open. The proper procedure, rather, is to depress the peripheral lip of the wound (*c*, Fig. 395) with a scoop, and thus open the valve.

In *small children* quietude of behavior after the operation is not to be expected, and hence the large sections, such as those made for iridectomy or cataract extraction, have their healing interfered with. Accordingly, for small children we ought to choose only those methods of operating, such as discission, which produce very small wounds.

In order to avoid post-operative infection many try to *close in operation wounds with conjunctiva* wherever they can. Thus in performing discission, whether of soft or membranous cataracts (see §§ 961 and 964), they enter through the conjunctiva beyond the corneo-scleral margin and then through the sclera at the periphery of the anterior chamber, instead of going through the cornea directly (Byers). So, too, when doing a posterior sclerotomy (§ 976), especially one for removing foreign bodies from the vitreous chamber, we fashion a quadrangular conjunctival flap in such a way that the cuts in the conjunctiva are distant from the incision to be made in the sclera. This flap is reflected, the scleral incision made, and the flap then replaced and sutured so as to cover the wound in the sclera. In a quite similar way a conjunctival flap is used by Kuhnt after keratectomy for partial staphyloma of the cornea (§ 943), by Lagrange for covering in the wound in sclerectomy, by Elliot for covering in the artificial corneal fistula made by the trephine (§ 959), and by Van Lint and others for covering in the corneal incision of a cataract extraction (§ 967).

896. **Reposition and Prolapse of Iris.**—In completing the operation the greatest attention must be paid to having *the iris in proper position.* Under no circumstances should the iris be left incarcerated in the wound (except in iridotasis). In cases in which an iridectomy has not been made, incarceration takes place because either the out-gushing aqueous or the lens that is pushed out by the pressure forces the iris into the wound. If an iridectomy is made, the iris, which is grasped by the forceps in front of the wound (Fig. 393, *aa*), is cut off flush with the latter. When this is done, of course the part of the iris that lay in the tract of the wound at the moment of the excision is left behind in the wound. If during the excision some traction is made on the iris, the portion of the iris that still lies inside of the anterior chamber is put on the stretch. This tension ceases when the iris is excised, so that the iris that is left behind can contract and thus the portion lying in the channel of the wound can retract into the anterior chamber. This retraction takes place particularly well if a contraction of the sphincter pupillæ aids it, although the sphincter has naturally lost much of its strength on account of the excision of a piece involving a solution of continuity. Very frequently, however, these agencies do not suffice to draw the iris back from the wound into the anterior chamber. If then, the iris is not pushed back by the operator into the interior of the eye, it remains in the wound.

Incarceration of the iris in the wound is manifested by different signs, according to the extent to which the iris is protruded. If the iris has pushed its way through the wound to the outside, it becomes visible as a dark swelling or nodule either in the middle of the section, or, if some of the iris has been excised, at one or both ends of it (*i*, Fig. 391). The condition when the iris does not protrude from the wound, but is merely jammed in between the internal lips of the latter, is recognized by the displacement of the pupil. The latter in case no iris has been excised is pear-shaped with its apex directed toward the scar (Fig. 392), just as after perforating ulcers of the cornea with inclusion of the iris (Figs. 239 and 268). After operations of this sort, in which the iris has been excised, the boundary between the pupil and the coloboma is marked by two projecting angles, forming what are called the angles of the sphincter (Fig. 390). These represent the spot where the margin of the pupil passes into the lateral limits ("limbs") of the coloboma. When the iris is free, the angles of the sphincter are directly opposite one another, and, moreover, lie in the circular line which the margin of the pupil would form if still intact ("the angles of the sphincter are low down," Fig. 390). But if the iris is incarcerated in the wound, the corresponding limb of the coloboma is thereby shortened, and the angle of the sphincter looks as if drawn up ("the angle of the sphincter is higher," Fig. 391, a_1). The angle of the sphincter may be pushed up so far that it is not visible at all and if this is the case with both sphincter angles the entire pupil is markedly displaced toward the margin of the cornea (Fig. 394).

Inclusion of the iris in the wound is accompanied by *evil consequences* of many kinds. The healing of the wound is interfered with by inflammatory irritation, and is protracted. The cicatrix is less solid and regular in its formation, and may become ectatic, and later on the inclusion of the iris

may give rise to increase of tension, to inflammation, and even to sympathetic disease of the other eye. To avert these results, every attempt must be made, after completing the operation, to release the iris from its condition of incarceration, and to put it in the proper position. This is done by entering the wound with a spatula, and stroking the iris with it back into the anterior chamber. Should this attempt be unsuccessful, or should the

Fig. 390

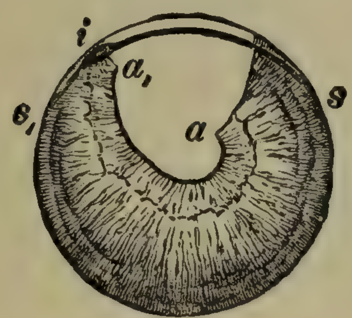


Fig. 391

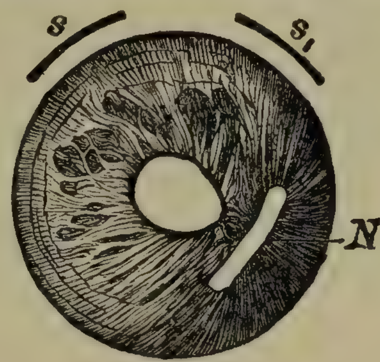


Fig. 392

Fig. 390.—Normal Position of the Iris. Magnified 2 × 1. The flap incision for the extraction, S, S_1, lies in the sclera. The angles of the sphincter, a and a_1, are both low down.

Fig. 391.—Incarceration of the Iris in the Wound (after a Corneal Flap Extraction). Magnified 2 × 1. The flap incision for the extraction, s, s_1, lies in the cornea. The iris is visible as a dark nodule, i, in the wound, and the angle, a_1, of the sphincter on the same side appears drawn up in comparison with that of the other side, which is in its proper position.

Fig. 392.—Incarceration of the Iris (after Linear Extraction). Magnified 2 × 1. The pupil is drawn toward the cicatrix, N.

Fig. 393

Fig. 394

Fig. 393.—Position of the Iris During Iridectomy. Magnified 2 × 1. The forceps has grasped the iris near the pupillary margin and has drawn it out of the wound, a, a. The portion of the iris lying outside of the eye is tent-shaped. The pupillary margin forms the two sides of the entrance to the tent, and between can be seen the posterior aspect of the iris—the retinal pigment of the latter—lining the inside of the tent.

Fig. 394.—Inclusion of Both Limbs of the Coloboma in the Scar. Magnified 2 × 1. The operation scar is solid and smooth and nowhere shows any included iris. The inclusion of the iris is recognized only from the drawing up of the pupil. The angles of the sphincter are not to be seen on either side, but the pupillary portion of the iris extends like a garland from one side to the other above, and the fibres of the ciliary portion are put greatly on the stretch in a vertical direction.

iris after replacement again prolapse into the wound, the incarcerated portion of the iris must be pulled out, grasped, and excised.

The firm closure of the wounds made with a lance knife diminishes the danger of prolapse of the iris. How, then, does *prolapse of the iris occur* at all? When the cornea has been perforated at any spot the aqueous flows from every direction toward this point, because here the ocular tension has sunk to nothing (i. e., has become equal to that of the external air). The fluid of the anterior chamber can flow toward the opening without obstruction; but the fluid of the posterior chamber, to get at the opening, must first pass through the pupil. Let us assume that the opening lies at the nasal margin of the cornea (I, Fig. 395). In this case the liquid from the temporal portion

of the posterior chamber would flow toward the opening directly through the pup because this is its shortest way. The case is otherwise with the nasal portion of th chamber, which lies immediately opposite the opening. Here going through the pup means taking a roundabout way, which is the more circuitous the farther the openin lies toward the periphery. The aqueous will therefore tend to take the shortest wa and rush straight forward toward the opening, pushing the iris before it. In the lan guage of physics, the posterior surface of the iris is subjected to the pressure of tha portion of the aqueous which has not yet escaped (*d*, Fig. 395). On the other hand, th pressure upon the anterior surface of the iris has become zero, and the iris consequentl is pushed against and into the opening. This is the way in which a prolapse of the iri originates, the prolapse being nothing but a sac consisting of iris and filled with th liquid of the posterior chamber.

The *danger of a prolapse of the iris* occurring is greater—1. The greater the rapid ity with which the aqueous escapes, because then proportionately less time is allowe the liquid of the posterior chamber to make the circuit by way of the pupil. Henc the rule that in making the sectio the aqueous should be allowed to flo off as slowly as possible. 2. Th higher the ocular tension, becaus then the difference between the pres sure in the anterior chamber whic has been emptied and the posterio chamber which is full becomes s much the greater. When an iridec tomy is made in glaucoma, a con siderable extent of the iris usually a once protrudes from the wounc 3. The further the wound lies towar the periphery, for then the rout which the aqueous has to take through the pupil is just so much the more circuitou and there is a proportionate increase in the depth of the posterior chamber at the spo corresponding to the section and in the quantity of aqueous that acts to propel the iri forward. 4. According to the size and shape of the aperture, since these conditions likewis have an effect in producing prolapse of the iris. It is evident that the opening must be of certain size for the iris to enter it at all. Again, if the aperture is circular—as is the case, fo example, after perforation of an ulcer of the cornea has taken place—prolapse of the iris wi assuredly not fail to develop. On the other hand, wounds with a valvular closure such as those made with the lance knife, display a comparatively slight liability t inclusion of the iris. We therefore try to anticipate the occurrence of perforation of a ulcer by making paracentesis with the lance knife, in order to avoid prolapse of the iri and the anterior synechia that results from it.

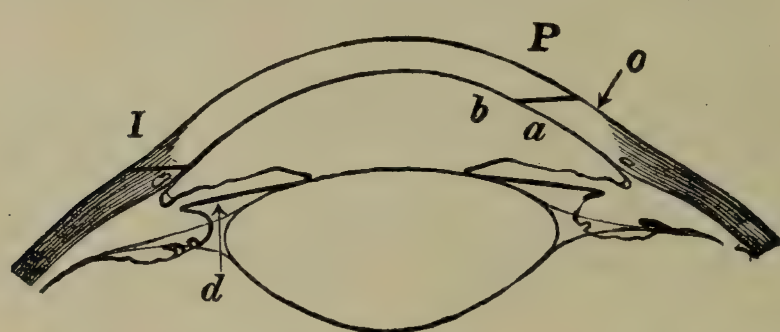


FIG. 395.—OBLIQUE COURSE OF THE SECTION THROUGH THE TUNICS OF THE EYE (SCHEMATIC). *I*, section for iridectomy, lying externally in the sclera, internally in the cornea. *P*, section for paracentesis, *a*, peripheral; *b*, central lip of the wound.

Prolapse of the iris may occur not only during the operation but *afterward* also We may, for example, have succeeded in avoiding the development of prolapse durin the operation, or have removed it if it has developed; and yet on the next day, whe we change the dressing, we may find the iris prolapsed into the wound. This occurrenc is due to the fact that the recently agglutinated wound has reopened, and the momen this took place the same conditions favoring prolapse of the iris were supplied as at th instant of the operation itself.

Prolapse of the iris and its incarceration in the operation wound must be avoide at any cost. If we are dealing with wounds in which there is but little tendency t prolapse, it is sufficient to replace carefully any iris that may have prolapsed durin the operation. But if the section is of such a character that the iris is apt to be pushe into it (as, for instance, is the case with a large scleral section), the reposition of th iris affords no security against prolapse; this may, and very often will, take place sub sequently. In these cases the only resource is excision of the iris. How does this aver prolapse? Is it perhaps because all the iris is removed that could possibly prolaps into the wound? If that were the case, the iris would have to be excised through th

entire extent of the wound—that is, often for a considerable width. But this is not by any means necessary. As a matter of fact, *iridectomy prevents prolapse* of the iris because it puts the posterior into direct communication with the anterior chamber at the site of the wound, so that the fluid which rea cumulates in the posterior chamber can flow directly toward the opening of the wound without forcing the iris before it. For this purpose a narrow iridectomy or even a very small opening in the periphery of the iris (button-hole iridectomy; see § 968) is all that is necessary.

897. Escape of Aqueous and Vitreous.—To avoid prolapse of the iris (§ 896) and hæmorrhage (§ 898), the escape of aqueous which follows an incision into the anterior chamber should be made as gradual as possible. In certain operations, as in discission (§§ 961, 964) no aqueous escapes. Escape of *vitreous* presupposes a breach in the sclera or in the diaphragm separating the aqueous and vitreous chambers. It occurs, therefore, particularly in cataract operations.

An escape of *aqueous* after the cornea has been opened presupposes a proportionate contraction upon the part of the capsule of the eyeball. If the walls of the eyeball were perfectly rigid like a metallic capsule, for instance, not a drop of liquid would escape from the orifice which had been made; a counter-opening would have to be made in another spot before it could do so. In addition to the elastic contraction of the tunics of the eye, the pressure of the external ocular muscles, and also the pressure of the lids upon the eye, contribute to reduce the volume of the capsule of the eyeball. Another factor contributing to the same result is added where the diaphragm formed by the lens and zonula is yielding enough to push forward after the escape of the aqueous. In old persons, the capsule of whose eyeball is rigid and whose eyes lie deep in the socket, so that the lids and muscles have but little power over them, the cornea after the escape of the aqueous (especially if the lens is removed at the same time) is often pushed in by the external atmospheric pressure (*collapsus corneæ*). This occurrence is favored by the diminished thickness of the cornea in old age, and also by the use, during the operation, of cocaine, which reduces the ocular tension. Collapse of the cornea was formerly looked upon as an evil event, because it prevents the precise apposition of the lips of the wound, and it was supposed that suppuration of the wound was thus produced. We now know that the process of healing is in no respect affected by a collapse of the cornea. The collapse disappears as soon as the aqueous reaccumulates, which is generally the case as early as a few minutes after the operation. If this is not the case, the collapse may be relieved by introducing warm sterile salt solution into the anterior chamber. This can be done by means of a bulb irrigator, a slender-tipped syringe, or any like device, introduced between the lips of the wound. The same manœuvre is called for in case of the much more serious collapse of the eyeball caused by great *escape of vitreous*. When the cornea on account of its elasticity tends to resume its shape after collapse has taken place, a negative pressure develops in the anterior chamber precisely as when the rubber ball of a syringe, after being compressed with the hand, is allowed to expand again. *Air* may be sucked in by means of this negative pressure, so that an air bubble enters the anterior chamber. This does no sort of harm to the eye. Blood may also be sucked in (see below).

898. Hæmorrhage After Operations.—*Hæmorrhage* into the *anterior chamber* occurs in those operations that cause injury to vascular tissues like the sclera and iris. If the iris is healthy, it hardly bleeds at all upon being cut through, since its vessels close very rapidly from contraction of their walls. But in those cases in which an operation is done upon a diseased iris (as in iritis, glaucoma, and atrophy of the iris), copious bleeding often takes place from the iris, so that the whole anterior chamber fills with

blood. General disturbances of metabolism also tend to provoke bleeding. Thus in a large number of cases of cataract extraction it was found that hæmorrhage into the anterior chamber occurred in 29 per cent. of the diabetic patients, in 13 per cent. of those affected with albuminuria, and only 4 per cent. of those having neither albumin nor sugar in the urine (Wheeler). The bleeding is disagreeable, because it prevents the operator from inspecting the interior of the eye; but in otherwise healthy eyes it generally produces no other disadvantage, as the blood is absorbed again within a few days. But in eyes where the iris is diseased not only is the hæmorrhage more extensive, but in addition the blood takes a longer time—sometimes in fact months—to disappear by resorption; for, it is precisely in such cases as these that the whole metabolism of the eye is seriously affected.

Hæmorrhage into the anterior chamber may occur when a cornea re-expands after having been collapsed and as it does so sucks out blood from the divided vessels of the iris, so that the chamber is filled with blood. This is particularly apt to take place when the cavity of the chambers is separated from the cavity of the vitreous by a more solid diaphragm than usual (exudation membranes), which is not able to advance adequately after the escape of the aqueous. Particularly profuse hæmorrhage is thus encountered in those iridectomies and iridotomies which are made in eyes with an old irido-cyclitis. The blood in this case is unpleasant for two reasons: first, because it is very slow in being absorbed; and, second, because it may in part become organized and close the new-made pupil up again. To prevent this hæmorrhage ex vacuo, a pressure bandage should be applied to the eye in such cases as soon as possible after the pupil has been formed (Fuchs). This bandage by external pressure diminishes the volume of the capsule of the eyeball, and presses the vitreous against the cornea.

Hæmorrhage from divided vessels should not be confounded with those intra-ocular hæmorrhages which are caused by the sudden and very great reduction of ocular tension in consequence of the operation—either from escape of the aqueous or removal of the lens—so that now blood pours in greater quantity into the vessels of the uvea and retina and makes them rupture. Such hæmorrhages are especially to be anticipated when operations are done in the presence of increase of tension, in which case the vessel walls are often degenerated. In fact, in glaucoma small retinal hæmorrhages after an iridectomy are the rule (see § 948). In rare cases, blood escapes from the vessels in such quantities that it pours out into the vitreous or beneath the chorioid. In the latter case the chorioid is detached from the sclera by the blood, and, indeed, when the hæmorrhage is very great it may even happen that the contents of the eyeball are expelled from the wound by the blood (*expulsive hæmorrhage*) and then the blood itself oozes from the wound. Such an eye, of course, is lost.

899. Dressing.—After the operation an aseptic dressing is applied. In the case of operations on the eye itself we proceed as follows. To prevent the agglutination of the lids by the secretion which usually forms under a dressing, we put sterile vaselin (with or without argyrol or collargol) into the conjunctival sac; and to prevent adhesion of the lashes to the dressing, smear lids and lashes with vaselin or boric-acid ointment. We then close the lids and place upon them first a pledget of sterilized gauze, and upon this lay a dressing of cotton, which is held in place by strips of zinc-oxide or isinglass

plaster. (The translator prefers the latter as being easier to remove and less disagreeable to a sensitive skin). Over the whole is applied a bandage. In operations for cataract, etc., a light latticed frame of wire (Fig. 396) is fastened over the dressing to prevent the patient from getting at his eye with his finger and doing harm by making the wound burst open. Snellen uses for this purpose a plate of aluminum, shaped like a mussel shell and

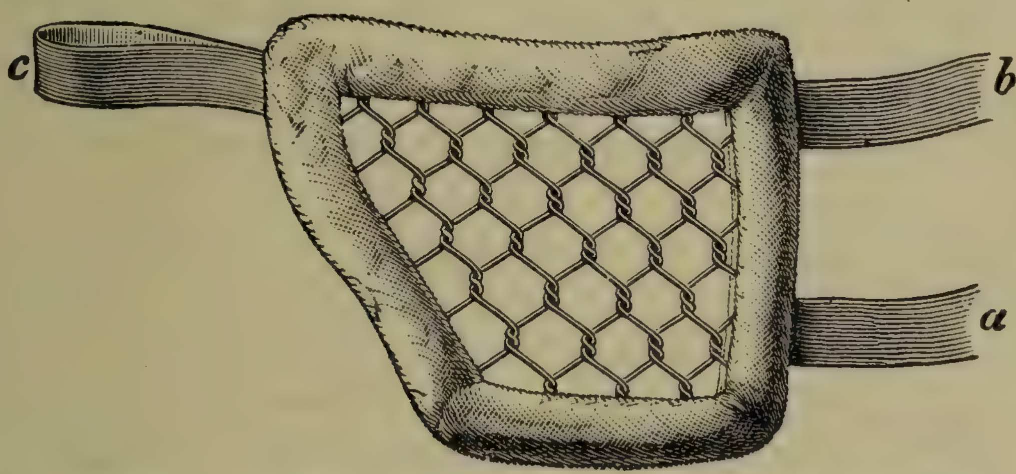


Fig. 396.—Latticed Frame Used for Dressing the Left Eye.

This is padded on its edges so as not to exert any pressure. From the two temporal extremities pass the bands, *a* and *b*, which are carried respectively below and above the left ear and across the occiput to the right side of the head. Here the two bands are tied together after one of them has been passed through the loop *c*.

fastened over the eye with strips of plaster. The same end is accomplished very satisfactorily by Ring's light pasteboard mask (Fig. 397). This completely covers both eyes, but, as a window can readily be cut in it, either eye may be left open when desired.

Some prefer to *dispense with a dresssing* altogether after operations, believing that it does not really prevent movements of the lids or the eye that would interfere with the healing of the wound, and that it does favor the damming up of secretions and the multiplication of germs in the conjunctival sac. And, as a matter of fact, if the conjunctiva is predisposed to inflammation, bandaging the eye will cause a profuse accumulation of secretion in the sac, so that the dressing has to be removed, and the eye irrigated several times a day with sterile salt solution and argyrol or protargol. So far as statistics go, the results of the open method seem to be as good as when a dressing is employed. Yet the consensus of opinion among the best and most experienced operators is that the dressing should be used in all operations in which the eyeball is opened (cataract, iridectomy). It is also the custom with most to bandage the eyes for several (two to ten) days after an advancement, the intention here being not so much to protect the eye from infection as to keep it from seeing and hence from moving about and so making traction upon the sutures. If this is the intention, both eyes ought to be bandaged, since if this is not done the uncovered eye will move about freely, and in that case the covered eye will necessarily move too.

Fig. 397.—Ring's Mask.

Even if it is thought wise to omit a dressing in the kind of cases just cited, the eye ought to be protected by a mask from the risk of accidental blows, especially at night.

The open method is suitable for tenotomies. Here, contrary to what obtains in the case of advancement, we wish to have the eyes move under the modifying influence of binocular vision and to be subjected to the fusion impulse which will help them to assume the proper position. As the danger of infection in these cases is very slight and after the first few hours when the external wound is sealed is practically nil, a bandage is used for at most the day of the operation. After this both eyes are left open, and the patient is encouraged to use them for distant vision.

Most prefer to *inspect the eye* twenty-four hours after the operation, to ascertain the character of the wound and particularly to see whether a prolapse of the iris is present or not; also in the case of an advancement or tenotomy to determine whether the position of the eyes is correct. If the condition is found to be satisfactory the dressing may then be left undisturbed for forty-eight hours, although many still prefer to examine the eye daily.

900. After-treatment.—The after-treatment of an operation in which the eyeball has been opened must be mainly directed to the prevention of anything that might interfere with the rapid and permanent closure of the wound. For this purpose the eye operated upon is bandaged, in order to put a stop to the movements of the lids; and in operations of any magnitude the eye not operated upon should also be kept shut for the first two or three days. Furthermore, the patient ought to avoid all physical exertion, as by this the ocular tension is increased, and the recently agglutinated wound might be forced open again. Hence, after major operations (iridectomy or cataract extraction), the patient is made to stay flat on his back in bed for the first day or two, and on the following days is permitted to be up in an arm-chair. For the first few days he is allowed only soft diet, so as to obviate the exertion of chewing. For other precautions to be observed in post-operative treatment, see § 113.

In old people, especially if they are drinkers, delirium not infrequently occurs, particularly when both eyes are bandaged. In this case the eye that has not been operated upon must be opened at once. Sometimes the delirium can be relieved only by getting the patients out of bed or sending them home. Drinkers, moreover, must not be deprived of their alcohol (see page 113). Atropine must be stopped, and hyoscine (0.3 mgr.) or chloral and bromides may be given (De Schweinitz, Posey).

Old people suffering from marasmus are apt, if they lie quiet upon their backs for several days after an operation, to get hypostases in the lungs, which may cause the patient's death. Hence, persons that are weak from old age ought to be taken out of bed very soon—if necessary, directly after the operation. There are other accidents, too, by which the course of the healing may be interfered with. As these can not usually be foreseen, it is usually advisable not to operate upon both eyes at one sitting; for from the operation and after-treatment in the case of one eye we learn what we are to expect when operating upon the second.

901. Normal Healing of Wounds.—When these precautions are observed the course of healing regularly takes place as follows: The edges of the wound become agglutinated soon after the operation, and the anterior chamber is restored. It very often happens that the recently agglutinated wound can not at once withstand the pressure of the accumulated aqueous, and in the course of the first day opens once or twice and allows the aqueous to escape before a permanent closure of the wound has taken place. The edges of the wound then heal by indirect union, so that a fine linear cicatrix is formed. If this lies in the cornea, it remains visible always

as a narrow gray line, while cicatrices in the sclera are as a usual thing scarcely discoverable after some time has elapsed. It always takes quite a long time for a cicatrix to become sufficiently firm to be able to resist external injuries. Until this has occurred (that is, for several weeks or months, according to the size of the wound), the patient must refrain from all severe physical exertion, avoid making any pressure upon the eye, etc.

902. Abnormalities in Healing.—Variations from the course of healing just described not infrequently occur. Such are—

1. *Irregular Healing.*—The *closure of the wound may be delayed.* Still more frequently it happens that the wound, after it has already been closed, is *burst open again* by an external injury, such as pressure of the hand upon the eye, coughing, sneezing, spasmodic tremor in sleep, and the like. This "bursting of the wound" is ordinarily followed by extravasation of blood into the anterior chamber. The iris also may be swept into the wound and incarcerated there, or inflammation (irido-cyclitis) may set in. Another way in which healing of the wound may be interfered with consists in the fact that the edges of the wound *do not adhere to one another directly* but are united by an interposed cicatricial mass of recent formation. This is most frequently the case when direct contact of the edges of the wound is prevented by the presence of an incarcerated iris or lens capsule or conjunctiva, but it may also occur when there is an increase of tension by means of which the wound is made to gape and is thus kept open. In these cases the scar that is formed is less firm, and in fact a small portion of it may remain entirely open, so that the aqueous keeps oozing through it beneath the conjunctiva and makes the latter œdematous (*cystoid cicatrization*, Fig. 279). The scars which have not sufficient firmness frequently become ectatic. This has as its immediate consequence an irregular bulging of the adjoining parts of the cornea, so that the results of the operation, so far as vision is concerned, are impaired by the presence of high astigmatism. Later on, cystoid or ectatic scars may give rise to elevation of tension or to inflammation.

As a result of imperfect closure or secondary rupture of the wound, allowing the aqueous to leak or sometimes as a result of detachment of the chorioid (§ 760), *the anterior chamber may remain obliterated* for days.

2. *Suppuration of the Wound.*—This usually begins in one or two days after the operation, rarely later. If the germs have made their way into the lips of the wound itself, the inflammation shows itself by a yellow infiltration of the edges of the wound; but, if the germs have been introduced into the deeper parts—anterior chamber or vitreous,—the inflammation begins with an exudation, which is poured out into the interior of the eye and which is first fibrinous, but very soon becomes purulent. It is only in the rarest cases that such an inflammation is arrested so quickly that the eye retains some visual power. The rule is that the eye is destroyed either by suppuration of the cornea or by a severe plastic irido-cyclitis or by an actual panophthalmitis. The outcome is atrophy, or, if panophthalmitis is superadded, phthisis of the eyeball. The lighter cases may sometimes be checked by cauterizing the wound with the cautery or carbolic acid or by repeated paracentesis of the cornea with injection of formalin or other anti-

septic into the anterior chamber (De Schweinitz). The subcutaneous injection of vaccines, and particularly of diphtheria antitoxin (Hansell and Ring) has saved some cases. Suppuration of the wound is most apt to set in after cataract extractions, and formerly was the most frequent cause of blindness in an eye which had been operated upon for cataract. The surest preventive is rigid antisepsis and asepsis.

3. *Non-Suppurative Inflammation of the Uvea.*—Iritis and irido-cyclitis occur very frequently after operations in which the eyeball has been opened. In most cases there is simply a slight iritis, which does no harm beyond that induced by the persistence of a few posterior synechiæ. But in the severe cases the inflammation leads to occlusion of the pupil, and either necessitates a secondary operation, or actually terminates in incurable blindness due to atrophy of the eyeball. In cases of the latter sort there is also a danger of sympathetic disease of the other eye. Slight inflammations of the iris may be produced by infection of a particularly mild character (see § 98); but in other cases they are to be regarded as purely traumatic, being caused by the way in which the iris has been grasped and pulled upon or by the fact that portions of the lens which are left behind, or similar substances, produce mechanical or chemical irritation of the iris. Severe inflammations depend either upon infection or upon a lighting up of old inflammation, as when an operation is done in an eye which was formerly the seat of an irido-cyclitis.

(For *striate opacity* of the cornea after operations, see § 589).

CHAPTER XXXIII

OPERATIONS ON THE CONJUNCTIVA AND LACRIMAL ORGANS

I. Dilatation of the Tear Passages

903. Dilatation of the canaliculi and the nasal duct is effected either with a syringe (Fig. 398, *h*) or with sounds.

Syringing the Duct.—Before using the syringe, the punctum and canaliculus should be dilated with the conical sound (Fig. 398, *c*). A little cocaine-adrenaline solution is dropped on the punctum, and the tip of the sound introduced with gradually increasing pressure. When well engaged it is turned horizontally inward and pushed gently toward the sac. Immediately afterward the tip of the syringe, properly bent, is introduced, gently shoved into the sac, and revolved so as to enter the tear duct. Then ten drops of a 2-per-cent solution of cocaine with a little adrenaline added are injected. The cocaine and adrenaline not only make the passages insensitive, but also shrink up the mucous membrane and so make the passages wider. When the passages are well opened by this means we may inject disinfectant solutions (1-4,000 sublimate solution, 3-per-cent solution of boric acid), which can afterward be replaced by astringent solutions. Or, even better, we may instil 30-per-cent argyrol just before the injection of the cocaine-adrenaline. When the latter has made its way through to the nose the argyrol will generally follow. (It is better not to inject the argyrol, as argyrosis may be produced in this way). If there is obstruction in the nose itself adrenaline may be applied to the inferior turbinate before the syringing. Syringing may be done twice a week or oftener, the patient in the intervals using instillations of adrenaline followed by argyrol.

904. Slitting of Canaliculus.—This is done as a preliminary to the use of sounds. We dilate the lower canaliculus with a probe, and syringe in a little adrenaline-cocaine solution as directed in the preceding section, then we introduce a Weber's knife (see Fig. 398, *f*) into the dilated canaliculus, in such a way that the edge of the knife looks up and a little backward. By quickly raising the knife, the probe-pointed extremity of which rests in the lacrimal sac, the canaliculus is slit open, and is thus converted into an open groove, looking upward and a little backward.

As Bowman showed, the conduction of tears is not injuriously affected by the slitting of the canaliculus. If this operation is performed with Weber's knife in the way described above, if affects only the external two-thirds of the canaliculus; the inner third of the latter lies beneath (behind) the caruncle, and remains unopened. Usually, the divided canaliculus closes up again the following day, so that we have to introduce the conical sound and thus tear apart the slightly agglutinated edges of the wound. If in this way readhesion is prevented at the outset, the cut surfaces become clothed with epithelium, so that afterward adhesion is no longer to be apprehended.

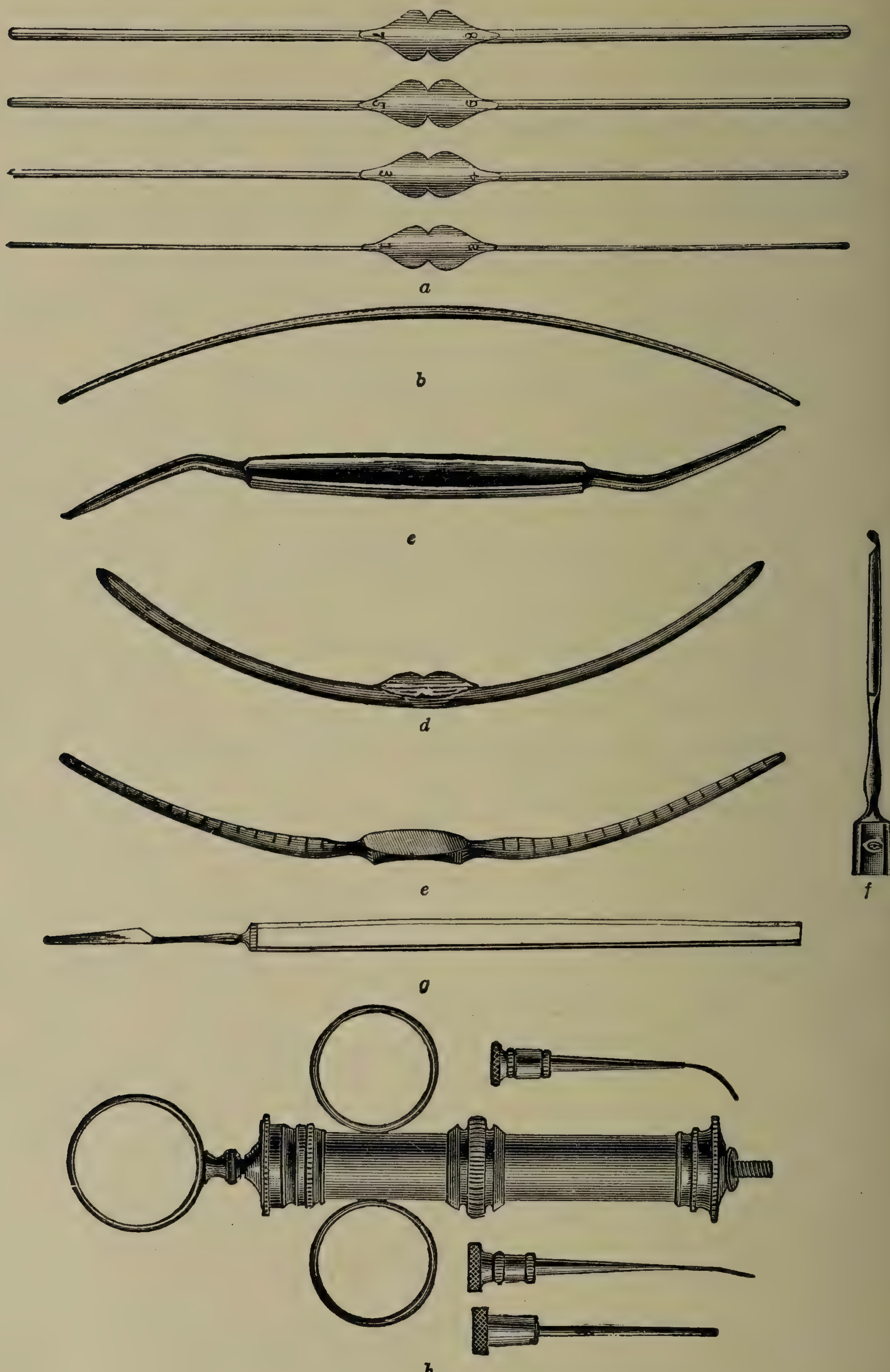


Fig. 398.—Instruments for Lacrimal Stricture.

a, Bowman's sounds. *b*, Ziegler's needle dilator (now discarded by Ziegler). *c*, Ziegler's dilator now made in six different sizes—1.5 tc 4 mm.—and of slightly different shape from that shown. *d*, Theobald's probe. *e*, Weber's thick sound for graduated dilatation. *f*, Weber's canaliculus knife for slitting the canaliculus. *g*, Stilling's knife, for division of lacrimal strictures. *h*, Anel's syringe.

905. Sounding the Duct.—In the way usually followed, this operation is preceded by slitting of the canaliculus (§ 904). We use then either Bowman's sounds, (Fig. 398, *a*), which are to be had in different sizes, numbered from one to eight or Theobald's probes, which are larger (Fig. 3, *d;* see also § 906).

The sound is first introduced through the inferior canaliculus, after this has been slit open, and is passed in the direction of the canaliculus until it strikes against the inner wall of the lacrimal sac. Then we tilt the sound so as to be about perpendicular, lifting its free extremity until its point, which is in the lacrimal sac, points to the furrow between the ala of the nose and the cheek, this giving the position of the inferior orifice of the nasal duct. The sound, being thus placed in an upright position, is now slowly and cautiously pushed downward until it rests upon the floor of the nasal fossa. In so doing we necessarily pass the contracted point, the favorite seat of which is either the point where the lacrimal sac enters the nasal duct, or the inferior extremity of the latter—the former spot, because it is normally the narrowest point in the lacrimal passages; the latter, because it is affected sooner than is the rest of the lacrimal tract by diseases of the nasal nucous membrane. We begin with the slenderest sounds. If we cannot pass the contracted spot with one of these, we must not push the sound forcibly forward, but must keep trying again and again, on succeeding days, to introduce the sound, until at length we succeed in carrying it down into the nose. The sound after its introduction is allowed to remain for about a quarter of an hour. We repeat the sounding every day, or every other day, gradually passing to larger and larger sounds, until at length the nasal duct is readily pervious and the epiphora has ceased. Even then the sounding should not be at once discontinued, as in that case the spots which have been dilated would soon close up again, owing to renewed contraction of the cicatrices. The sounding must therefore be repeated again and again at longer intervals (of a week to a month).

We might pass the sound through the *upper canaliculus*. This is rather the easier way because we do not then have to turn the sound so much in order to get it into the duct. But we prefer the lower route because the upper canaliculus is narrower so that with sounds of any size we easily cause a laceration of the mucous membrane, which may cause a subsequent obliteration of the passage.

Beginners in sounding frequently make the mistake of tilting the sound before its point has got into the lacrimal sac. We then feel an obstacle opposing the downward progress of the sound, and if we should try to overcome this obstacle by force we would make a false passage. That the sound has been tilted up too soon is recognized from the fact that when it is set vertical, the skin beneath the canaliculus is drawn along with it and is thrown into wrinkles. We shall not commit this mistake if we avoid tilting the sound until we distinctly feel through its point the firm resistance offered by the inner osseous wall of the lacrimal sac (the lacrimal bone). The obstacles opposing the passage of the sound in the nasal duct may be actual constrictions of the duct but are often nothing but projecting folds in which the sound catches. We accordingly try to push our way forward by sliding the point of the sound sometimes along one, sometimes the other, wall of the lacrimal sac in order to smooth out the folds. Sometimes we can pass a rather thicker sound (No. 3) more readily than we can the thinnest ones; the latter, moreover, are more apt to injure the mucous membrane, so that we get beneath it and make a false passage. Bleeding from the nose after sound-

ing points to an injury of the mucous membrane, as does also the striking of the apex of the sound upon bare bone. The latter, however, can also happen without the mucous membrane's being injured, if, for example, the bone has been already denuded by ulcerative disintegration of the mucous membrane of the nasal duct. In cases of this kind a cure cannot usually be obtained by treatment with sounds.

When finally the sound has been passed all the way through, it is felt to rest upon the floor of the nasal cavity. In most persons the lamina in the middle of the sound then lies upon the inner end of the eyebrow. To ascertain the position of the sound more precisely, we may place on the outside a second sound of the same length in the direction of the lacrimal tract and in such a way that the laminæ of the two sounds are superimposed; the exterior sound then shows at what height the lower extremity of the sound that we have introduced stands.

Treatment by sounds must be kept up until at least No. 4 of Bowman's sounds passes with ease. Then the patient may be instructed how to pass the sound upon himself with the aid of a looking-glass, so that the sounding may be performed from time to time subsequently in order to prevent a recurrence of the contraction.

906. Radical Treatment of Strictures.—The long duration of the treatment by sounds has excited a desire to accomplish the dilatation of the strictures rapidly instead of gradually, and thus shorten the treatment. This can be done either by Weber's or Theobald's method of introducing very thick sounds, (Fig. 398, *d* and *e*), or by Stilling's method of incising the strictures with a knife designed for the purpose, (Fig. 398, *g*), or by a combination of both methods, Green curettes the duct, then dilates with the very largest probes and applies tincture of iodine to the walls of the sac and duct. Much better than these methods seems that of Ziegler, who by using his conical dilators (Fig. 398, *c*) in succession from smallest to largest, is able to dilate the canal at one sitting and that without preliminary slitting of the canaliculus. He has thus been able to effect a radical cure at once, and has, morover, not incurred the difficulties alleged against radical treatment, namely solutions of continuity in the mucuous membrane leading to the formation of fresh cicatrices with recurrence of the stricture.

907. Artificial Closure of the Puncta.—A procedure for shutting off the connection between the lacrimal sac and the conjunctival sac before an operation on the eyeball and thus preventing infection of the wound consists in searing the canaliculi by cauterization. For this purpose a fine galvano-cautery point is carried into the canaliculus The latter is at once obliterated by the eschar and by further action is completely seared. If, however, the cauterization is performed lightly, the permeability of the canaliculus can be restored after the operation wound has healed. Even when we extirpate the tear sac before operating on the eyeball we ought at the same time to sear the canaliculi, since otherwise the latter may become the source of infection. Quackenboss and others accomplish the same end by passing a ligature round the canaliculi before an operation. When the operation wound is healed the ligature can be removed and the lumen of the canaliculus restored.

II Operations on the Lacrimal Sac and Glands

908. Incision of the Lacrimal Sac (Dacryocystotomy).—In *acute dacryocystitis* as soon as fluctuation is apparent we incise the anterior wall of the lacrimal sac, or that portion of the skin beneath which the presence of pus can be made out. A lacrimal fistula is thus artificially produced, through which the contents of the abscess and of the lacrimal sac itself are dis-

charged externally. This is kept open by the introduction of a strip of iodoform gauze every day, until all inflammatory symptoms have disappeared and the secretion that exudes has lost its purulent character. But even then we ought not to allow the fistula to close at once, for we must recollect that a chronic dacryocystitis has preceded the acute attack, and that consequently there is a stricture present in the nasal duct. So long as this is present the fistula will not become permanently closed, and its temporary closure might result in a new attack of acute dacryocystitis. Hence, the permeability of the nasal duct must first be restored by treatment with sounds. When we have succeeded in doing this the fistula usually closes of itself. If this should not be the case, we can effect a closure of the fistula by either refreshing and uniting its edges or by cauterizing them. If the conditions are such that a permanent state of perviousness of the lacrimal channels is unattainable, or if the patient cannot undergo the protracted treatment with sounds, we proceed to extirpate the lacrimal sac (see § 909). To the translator this radical treatment seems the better procedure, and he has even found it of distinct advantage to extirpate the sac in the acute stage of dacryocystitis right after incision.

In *chronic dacryocystitis* incision is done by Petit's method, for the performance of which Arlt has given the following guides: By drawing the lids to the outer side the internal palpebral ligament is put on the stretch, so that it is seen through the skin of the inner angle of the eye as a prominent projection. The point of a sharp scalpel is introduced exactly beneath the centre of the ligament. The back of the knife looks upward and the knife itself is held so that its handle passes through the middle point of an imaginary line drawn from the apex of the nose to the outer margin of the orbit. The knife held in this direction is thrust vertically in, thus penetrating through the skin and the anterior wall of the lacrimal sac. As soon as we feel the point of the knife striking against the posterior wall of the sac (lacrimal bone) we no longer push it forward, but depress its point by raising the handle as high as the forehead. If now the knife is pushed forward, its point enters the upper part of the nasal duct, the wound in the anterior wall of the sac being at the same time enlarged. After withdrawing the knife we enlarge the wound upward and downward so as to have a view of the mucous membrane of the sac throughout its whole extent.

Incision of the sac was formerly done in order to introduce corrosive agents into it and thus destroy it. This procedure is now replaced by that of extirpation of the sac. Incision has been done occasionally as a preliminary to a cataract operation or to an iridectomy, when there has been a dacryocystitis, but the secretion from the sac is not obviously abnormal. The sac in this case may be opened, washed out, and packed off with iodoform gauze directly before the operation on the eyeball (Fuchs). When after the wound in the eyeball heals the gauze is taken out of the sac, the wound in the latter readheres and the sac performs its function as before.

909. Extirpation of the Sac (Dacryocystectomy.)—For this purpose we first inject some cocaine-adrenalin solution beneath the skin over the tear sac, and also through the canaliculi into the tear sac itself. Then we make a curved incision through the skin three mm. to the nasal side of the inner canthus, beginning a little above the internal palpebral ligament and dividing the latter[1] and running two cm. downward and somewhat outward. We

[1] The operation can also be done without dividing the internal palpebral ligament, the cupola of the sac which lies behind the ligament being simply drawn down and dissected out. The extirpation may be facilitated by injecting the sac with paraffin (its lower end being tied off before this is done); or if the sac has already been opened it may be packed with gauze.

distend the lips of the wound with a small spring speculum (Müller's), which not only exposes the field of operation but also checks the bleeding. Then beginning at the temporal side of the tear sac and using a blunt instrument, we dissect the sac out without opening it. When the sac has been isolated completely, it is divided with the scissors close to the point where it passes into the nasal duct, i. e., at the upper end of the bony canal. The extirpation must be very carefully done since, if the smallest bit of mucous membrane is left behind, suppuration will recur from the canaliculi or from a fistula. If, owing to the great friability of the mucous membrane, it is impossible to dissect it out completely, we must currette the remaining portions out with a sharp spoon. Then we sew up the outer wound and by means of a pressure bandage make sure that the walls of the cavity are in apposition. As a rule, primary union occurs and healing is complete in a few days. In Verhoeff's operation, which exposes the parts well and is attended with little bleeding, a curved incision, fifteen mm. long and extending rather more above than below the canthal ligament, is made with its apex about two mm. from the inner canthus and directed toward the eye. The skin flap thus formed is laid back over the nose.

910. Operations Effecting Permanent Drainage.—Since by extirpation of the lacrimal sac the normal drainage of tears is made forever impossible, Toti has proposed as a substitute for this operation that of *dacryocystorrhinostomy*. The medial wall of the sac is removed, and a correspondingly large gap is also made in its bony substratum by the resection of a piece of the lacrimal bone and of the frontal process of the superior maxilla. Thus the lumen of the lacrimal sac is brought into direct connection with the nasal fossa, into which the contents of the sac can now discharge, though passing outside of the nasal duct. The direct result of the operation is good so far as the epiphora is concerned, but it is the belief of the most experienced ophthalmologists that extirpation of the sac is a more satisfactory procedure than this or than *West's operation*, by which the tear sac is opened from the nasal cavity and to such an extent that a permanent communication is effected.

911. Extirpation of the Lacrimal Glands.—Extirpation of the *superior lacrimal gland* is performed through an incision made in the outer part of the previously shaved eyebrow. (It is made here in order that the scar shall be invisible afterward.) This operation is done particularly in cases of degeneration of the gland.

The *inferior lacrimal gland* is extirpated through the conjunctival sac. After everting the upper lid we draw down the retrotarsal fold with the forceps and inject some cocaine solution beneath the temporal half of the fold. Then we cut the latter longitudinally with the scissors as far as the outer commissure. If now we separate the edges of the wound we see the acini of the gland presenting, and can readily dissect them out. Since the acini are grouped about the excretory ducts of the upper gland these ducts are divided at the same time. This explains the marked effect of the operation on the secretion of tears.

III. Expression and Grattage

912. In performing *expression* for trachoma we attempt to remove the granules without destroying the conjunctiva. This may be done either by puncturing the granulations individually with a sharp knife and then squeezing them out (Sattler), or by passing the retrotarsal folds through the blades of Knapp's roller forceps. In the latter instrument each blade carries a fluted roller, and when the lids are everted the conjunctiva is drawn

in between the two rollers, which fit closely into each other and which are rolled over the surface so that the trachoma granules are squeezed out.

Many scarify the retrotarsal folds with a small triple-bladed scarifier before expressing the granulations. Kuhnt uses an "expressor," which has smooth blades and simply squeezes the granules out without making traction on the conjunctiva or tearing it. Others prefer *brossage*, which consists in brushing the everted lids vigorously with horizontal sweeps of a small sterile toothbrush. *Grattage* is brossage with preliminary scarification, the toothbrush being soaked in a 1:500 or 1:2000 sublimate solution. All these operations are quite painful and in children require general anæsthesia. In others they may be performed with local anæsthesia, particularly if, following Guttman's suggestion, we inject a very weak solution of cocaine directly into the retrotarsal folds § 893. The translator's experience, agreeing with that of others, is that expression with the roller forceps is a satisfactory method, which, if properly performed, is rarely attended with undue reaction or untoward symptoms. The expression may be applied not only to the retrotarsal folds but to the tarsus as well. In this case one branch of the forceps is placed on the cutaneous, the other on the tarsal surface of the lids, and the latter is squeezed by the forceps, which is rolled in different directions.

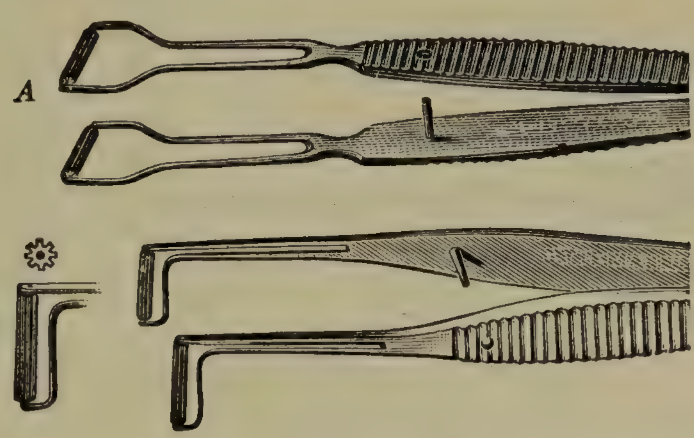


FIG. 399.—ROLLER FORCEPS FOR TRACHOMA. *A*. Knapp's forceps. B. Rust's modification. Similar forceps have been devised by Noyes and others.

IV. OPERATIONS FOR PTERYGIUM AND SUPERFICIAL GROWTHS ON EYE

913. A small pterygium may be ablated by Arlt's method. With a toothed forceps we grasp the pterygium at its neck, where the incurving of its margin permits it to be partially lifted from its bed. Starting from this point, we carefully dissect off the head from the cornea, upon which it lies, keeping strictly to the line of division between the tissue of the pterygium and the cornea. We must be particularly careful to remove thoroughly the gray, gelatinous zone at the apex of the pterygium, scraping it away if necessary. When the whole of the head as far as the limbus has been separated from the cornea we circumscribe this segment by carrying from the upper and lower margins of the neck two converging incisions into the body of the pterygium (the dotted line, Fig. 400). In this way a rhomboidal piece, containing the head and part of the body, is excised and a raw surface remains, one portion of which lies in the cornea, the other in the conjunctiva of the eyeball. This last is covered by uniting the upper and lower margins of the sections by means of one or two interrupted sutures. The wound in

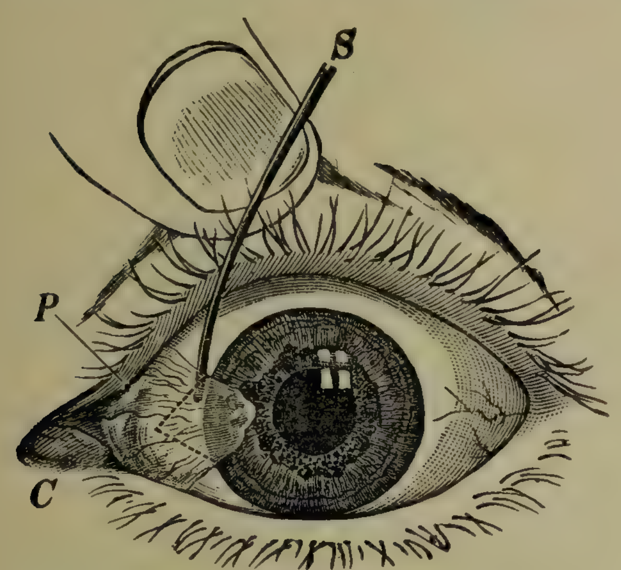


FIG. 400.—OPERATION FOR PTERYGIUM.—Dotted lines indicate incisions.

the cornea heals by a cicatrix forming over it, so that a permanent opacity remains. Care in sewing up the conjunctival wound is of the greatest importance, especially in the neighborhood of the limbus, as otherwise the conjunctiva grows anew over the raw surface of the cornea, and thus the pterygium recurs. However, even after carefully performed operations relapses are not infrequent, and then a second removal is required.

In *detaching* the pterygium a blunt divulsor (Fig. 401) may be used. This is introduced beneath the pterygium and brought like a draw-knife toward the pterygium head. Or, the pterygium may be stripped off clean by a thread passed beneath it and carried in toward the cornea with a sawing motion (Hunter).

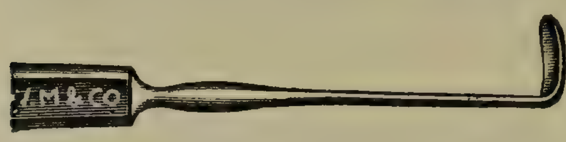


FIG. 401.—PRINCE'S DIVULSOR.

In very broad pterygia it is difficult or impossible to unite the edges of the conjunctival wound by a suture. In that case liberating incisions are made through the adjacent conjunctiva in order to make it easy to draw it down; and, instead of cutting off the tip of the pterygium, it may be sewed into the gaps which have been made by the liberating incisions. This is called *transplanting* the pterygium. *Knapp's* method of transplantation is shown in Fig. 402. *McReynold's* method is somewhat like this,

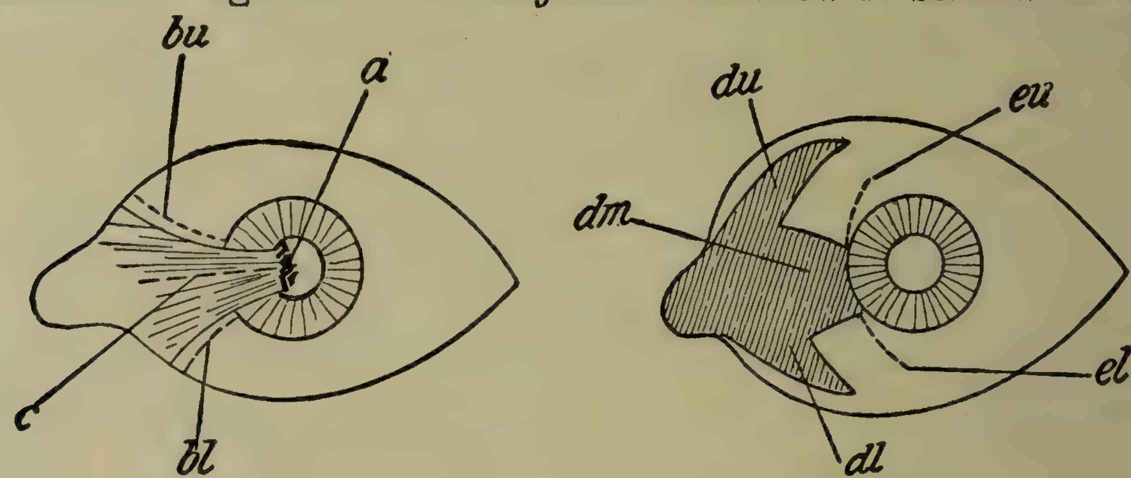


FIG. 402.—KNAPP'S TRANSPLANTATION OF PTERYGIUM. (After Norris and Oliver.)

The head of the pterygium (I, *a*) is dissected off, and the incisions *bu* and *bl* carried along the margins of the pterygium through the conjunctiva. These incisions are prolonged into the upper and the lower fornix, producing in each an angular gap (II, *dl* and *du*). The pterygium is then detached, its head is cut off, and the pterygium split by a horizontal incision (I, *c*) into an upper and a lower half. The tip of the upper half is stitched into the defect *du*, the tip of the lower half into *dl*. The incisions *eu* and *el* are made, delimiting two quadrangular conjunctival flaps, which are undermined and then brought together and stitched so as to cover the defect *dm*. The innermost stitch connecting these flaps is also passed through the base of the pterygium.

but the conjunctiva is divided along the lower edge only of the pterygium, the conjunctiva below the pterygium is undermined, and the pterygium is drawn down into the pocket thus formed and sewed there by a double-armed suture passed through the tip of the pterygium and brought out through the conjunctiva six or seven mm. below the cornea. The upper edge of the pterygium with the conjunctiva still attached to it is thus drawn out smooth and flat. *Beard's* operation is shown in Fig. 403.

Pterygia may also be destroyed by *strangulation.* Ligatures are passed round the neck and body of the growths, tied tight, and left in till they cut their way through. The head of the pterygium then atrophies. To secure the same object the neck of the pterygium may be seared with the *cautery.*

914. Removal of Superficial Tumors.—For removing dermoids and similar growths at the sclero-corneal margin *Beard's* operation (very similar to his pterygium operation) may be used (Fig. 404).

In *Francis'* operation for removing superficial epitheliomata, etc., a broad keratome is used to make an incision in the cornea encircling the

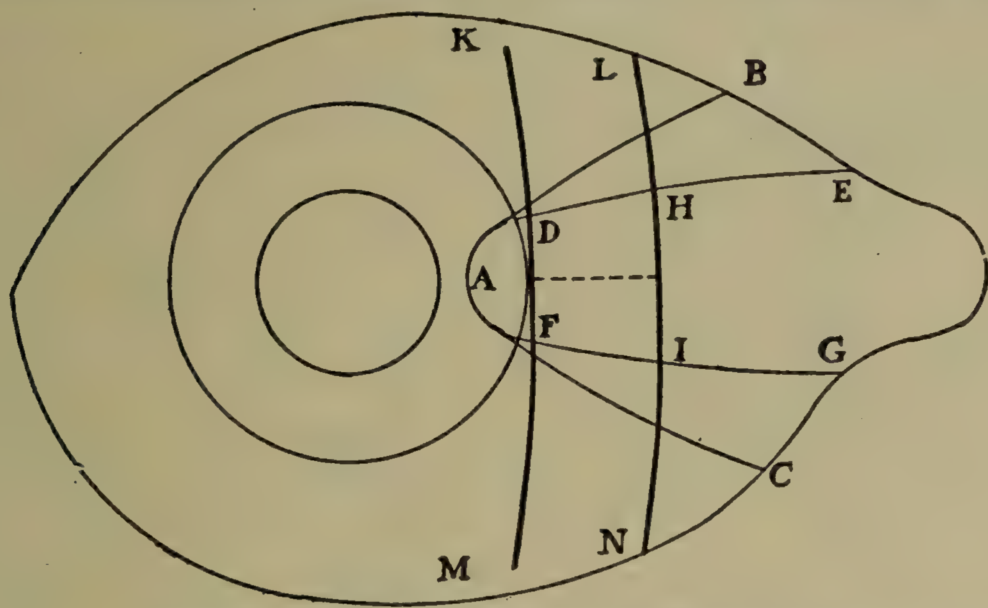


FIG. 403.—BEARD'S OPERATION FOR PTERYGIUM.

BAC, pterygium. Two slightly diverging incisions, *DE* and *FG* made through the upper layer only of the conjunctiva (not through the whole thickness of the pterygium) are carried well to the canthus. An instrument to strip up the pterygium is introduced through incision *FI* and brought out in incision *DH*; then is carried toward the cornea so as to detach the head *DAF*. The latter is cut off, and then the body of the pterygium, so far as it is included between the incisions *DH* and *FI*, is carefully dissected from its bed and pushed back toward the canthus, *DF* retracting to *HI* and leaving a raw surface, *DHFI*. The conjunctiva above *DH* and below *FI* is undermined, and the incisions, *HL*, *DK*, *FM*, and *IN*, made so as to delimit the flaps *KDHL* and *MFIN*. These are drawn together so that *DH* and *FI* meet in the dotted line. They are attached to each other by a single suture and by another suture to the retracted pterygium *EHIG*.

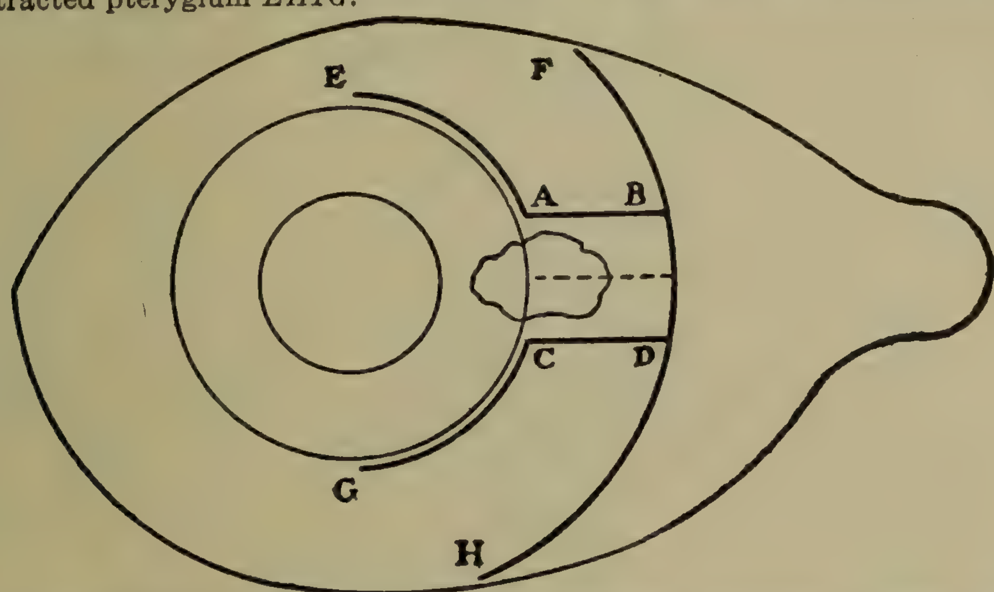


FIG. 404.—BEARD'S OPERATION FOR SUPERFICIAL GROWTHS AT THE LIMBUS.

The incisions, *AB* and *CD*, are made so as to include the whole of the growth. The latter is stripped up and shaved off clean just as in the case of a pterygium (Fig. 403), but instead of being simply shoved aside, is removed in its entirety. There is thus left a raw surface, *BACD*. The conjunctiva above *AB* and below *CD* is undermined, and by incisions, *AE*, *CG*, *BF* and *DH*, concentric with the cornea, the flaps, *EABF* and *GCDH*, are delimited. These are brought together so that *AB* and *CD* meet along the dotted line. A stitch or two serve to keep them in place.

growth. The keratome, directed perpendicularly to the cornea, is first carried through the superficial layers of the latter, then is turned at right angles and shoved along underneath the tumor so as to lift it in its entirety from the sound cornea underneath. The rest of the dissection is done with scissors as far out as necessary. The defect is covered with a conjunctival flap overlapping the cornea.

V. Operations for Symblepharon

915. Cases of *symblepharon anterius* are easily cured. We separate the adhesions between the lid and the eyeball with great care, so as not to cut into the sclera or the tarsus. When the lid has been set free, we must then take care to prevent a readhesion of the raw surfaces, and to make each of these cicatrize by itself. This is effected by repeatedly drawing the lid away from the eyeball, and also by interposing a pledget dipped in oil or smeared with ointment between the lid and the eye.

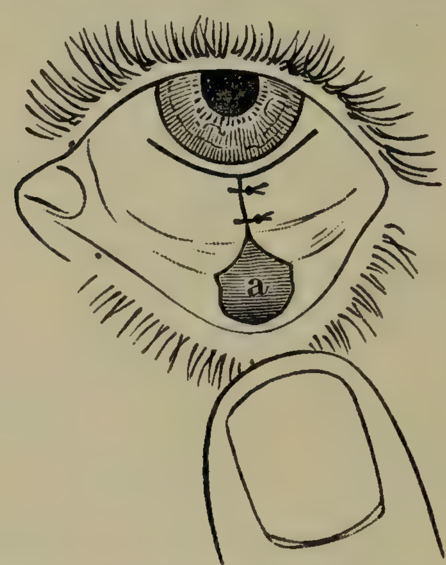


Fig. 405.—Operation for a Symblepharon Posterius (Arlt).

The wound in the palpebral conjunctiva (a) has been left open, that in the bulbar conjunctiva closed by two interrupted sutures. In order to be able to draw the conjunctiva up over the raw surface, a liberating incision has been made along the margin of the cornea, inward and outward from the upper end of the wound.

In *symblepharon posterius* we also begin by separating the adhesions as far back as the fornix. We then see, when we have drawn the lid away from the eyeball, two corresponding raw surfaces, one in the eyeball, the other on the lid (Fig. 405). These two opposed surfaces, which meet at the fornix, would soon reunite, beginning at the fornix, if we did not take care to have one of the raw surfaces covered with conjunctiva, so that a place coated with epithelium lies opposite to the raw surface remaining. The wound

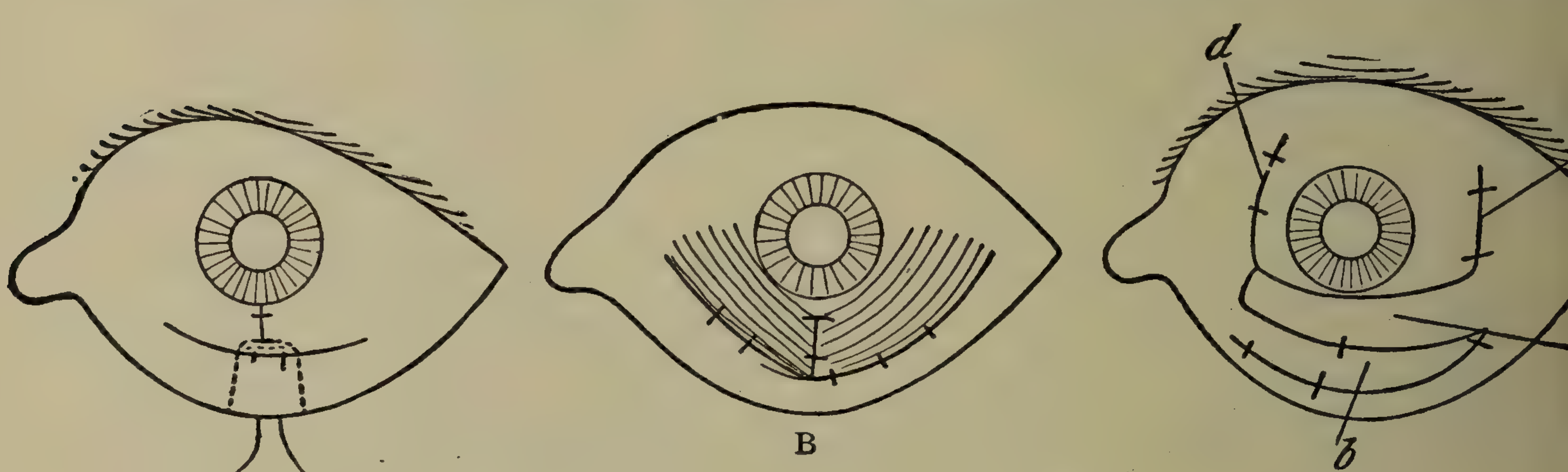


Fig. 406.—Operations for Symblepharon. (After Knapp in Norris and Oliver.)

A. Arlt's second method used when the cornea is encroached upon (pseudo-pterygium). This latter is detached from the cornea and eyeball down to the fornix, and the head of it is stitched into the fornix by a double-armed thread, which is passed through the lid so as to form a loop on the cutaneous surface of the latter. This loop is tied over a roll of gauze or bit of rubber tubing. The raw surface of the conjunctiva is covered in as shown in Fig. 405.

B. Knapp's method for covering the defect with two sliding flaps of conjunctiva. To prevent these flaps from over-riding the cornea their lower borders are fastened down with stitches carried through the submucous tissue of the fornix or through the thickness of the lids as described in A.

C. Teale's method. A sliding quadrangular vertical flap is fashioned out of conjunctiva at c and another one at d. Flap c, is brought down rotated 90°, and stitched in place so as to cover the defects in the bulbar conjunctiva, (at a), flap d covering that in the tarsal conjunctiva (at b). The raw spots left by the removal of these flaps are covered in by drawing the adjacent conjunctiva over them and stitching it in place.

on the eyeball is the one we select for covering over, because the conjunctiva of the eyeball can readily be displaced, while the conjunctiva of

the lid is adherent immovably to the tarsus. We loosen the conjunctiva of the eyeball on both sides of the wound, draw it down over the latter, and unite it by sutures. Especial care must be devoted to uniting the wound in the neighborhood of the fornix. If, after dividing the adhesions, the raw surface upon the eyeball is so large that it cannot possibly be covered by conjunctiva, reunion of the symblepharon will inevitably result.

For these more serious cases other operations have been devised. Himly, in an operation analogous to that for syndactylism, first made a preliminary perforation of the adhesion along the fornix, and carried a lead wire through the canal so formed. The latter, after the wire has been in it a pretty long time, becomes covered with epithelium (like the canal made by piercing the lobule of the ear for ear-rings), so that the symblepharon posterius is changed into a symblepharon anterius, and can be operated upon, like the latter, by simply separating the adhesion. Some have attempted to cover a large loss of substance upon the eyeball, caused by the removal of a symblepharon, by making the conjunctiva that is to be drawn up over it movable by means of liberating incisions, or by fashioning pedicellate flaps out of conjunctiva and attaching them to the wound (Teale, Knapp, Fig. 406). Non-pedicled grafts of mucous membrane (Stellwag, Wolfe) may also be used (cf. § 916).

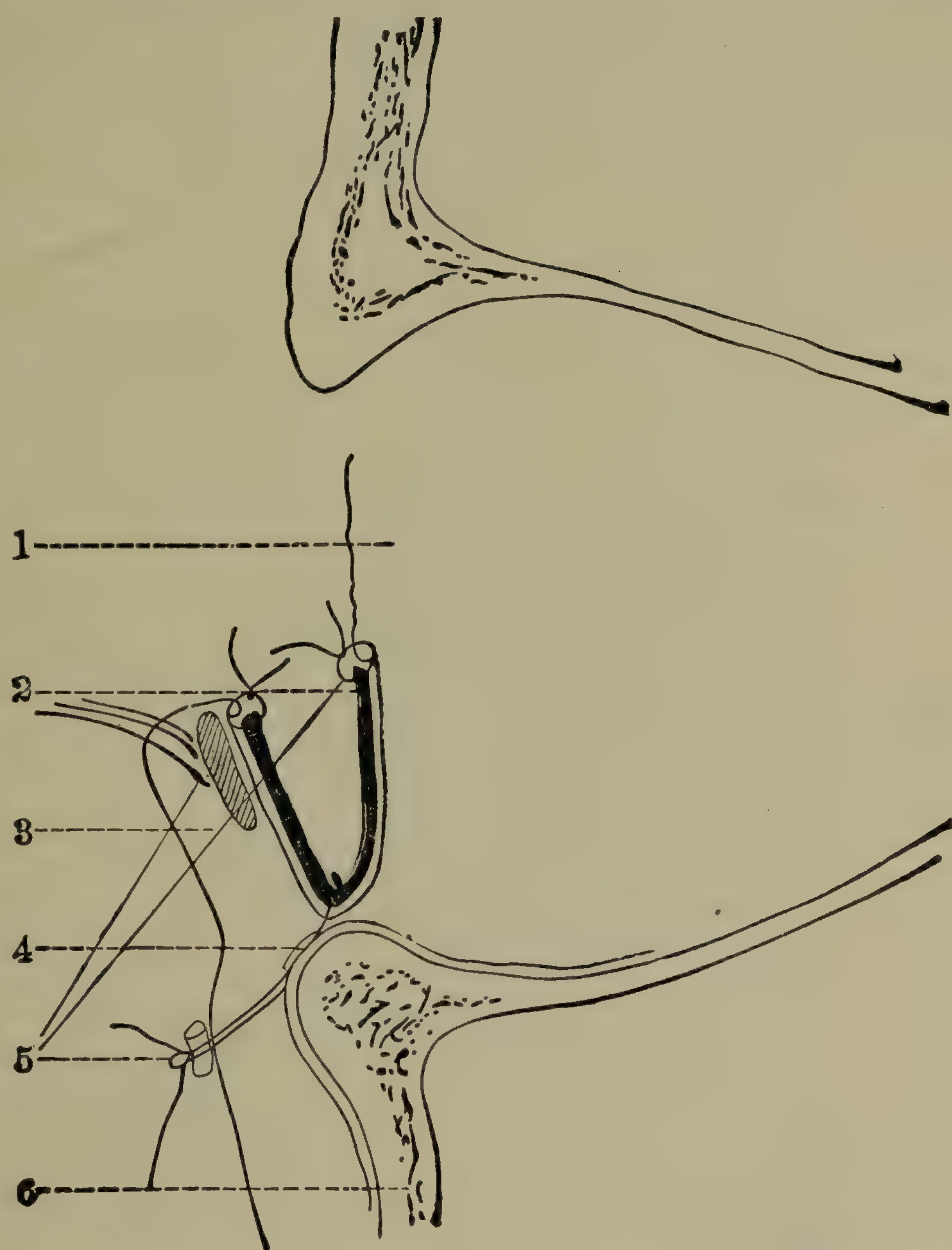


Fig. 407. Formation of New Cul-de-sac (after Weeks).—1, orbital tissue; 2, flap in position; 3, lid; 4, periosteum; 5, sutures; 6, bone of orbit.

916. Formation of New Cul-de-Sac and Eye Socket.—When the symblepharon is such as to obliberate the fornix, a new fornix may be made (especially for the reception of an artificial eye) by inserting a Wolfe graft (§ 934) into the groove made by dissecting out the cicatricial tissue, anchoring the graft down by stitches passed through the periosteum of the orbital margin and the lids (see Fig. 407), and keeping it in place by a guttapercha plate (Weeks). Instead of the Wolfe graft one made of mucous membrane taken from the lip or inner surface of the cheek may be used. This is sewed to the adjoining mucous membrane with silk stitches and anchored to the cul-de-sac as in Weeks's operation by double-armed sutures (best made of silk worm gut) which pierce the lid and are tied down on the exterior surface of the latter (Jobson). In Wheeler's operation (Fig. 408) after a canthotomy extending from outer canthus to orbital margin, the lids (including only the skin, orbicularis, fascia and tarsus) are separated from the under-

lying tissue by a dissection extending to or beyond the orbital margin; all cicatricial and granulation tissue is removed and the tarsus trimmed away; then a dental impression mould of sufficient size round which is wrapped a graft composed of epidermus only, raw

A B C

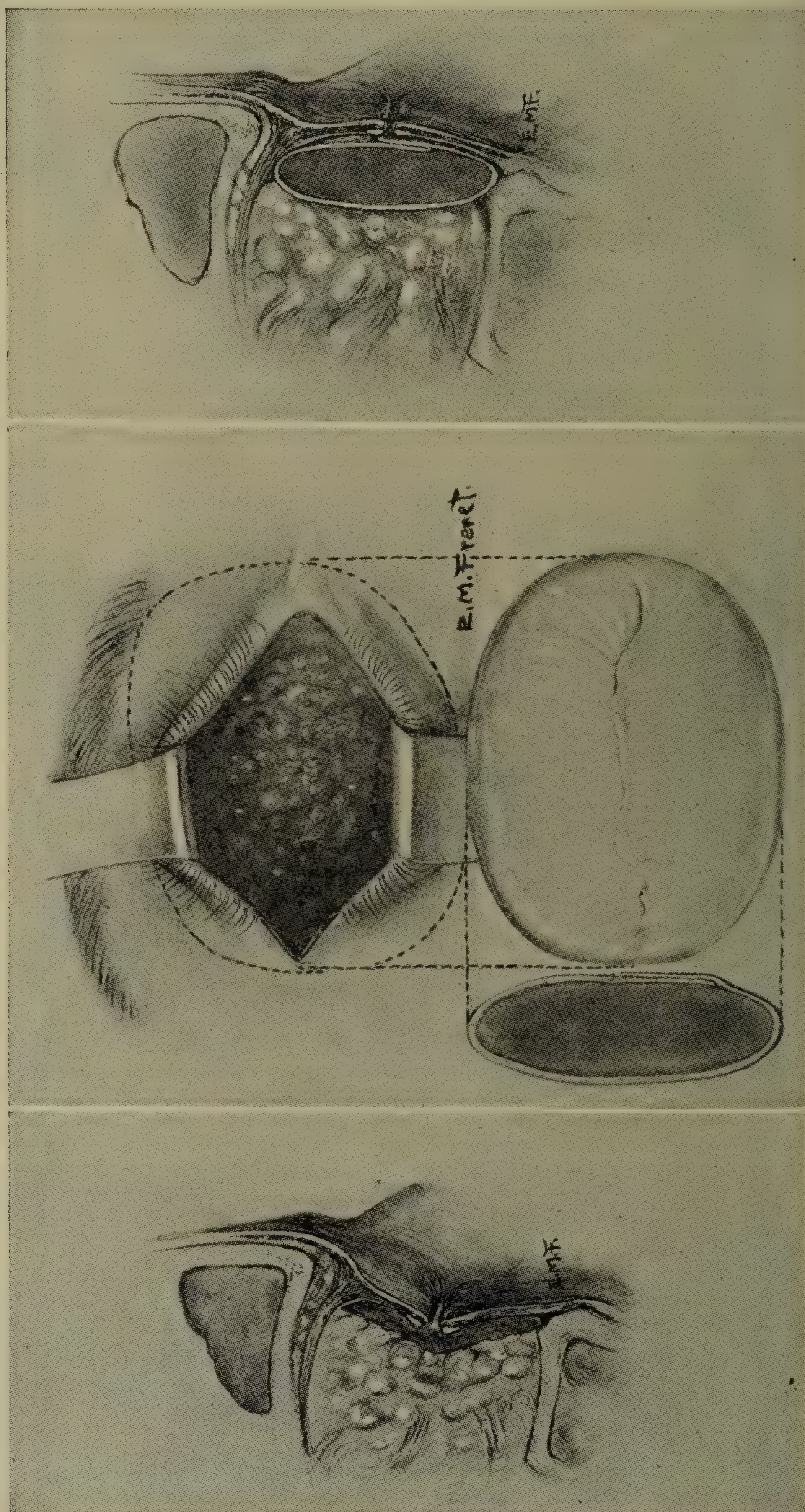


Fig. 408. Restoration of Obliterated Eye Socket (after Wheeler).

A. Cross section through orbit and eyelids to show dissected cavity between eyelids and orbital contents for new socket.
B. Dissection completed. Eyelids retracted to show bed for reception of graft. Orbital margin indicated by curved dotted line.
Below—Cross section and front view of form, completely covered by epidermic graft, ready for introduction to the prepared cavity.
C. Mold and graft placed in new socket.

side out, is inserted. A very firm pressure bandage is applied and is left undisturbed for a week. The form is removed in three weeks. Similar in principle is the operation of Esser and others by which a cul-de-sac contracted by cicatricial processes in the lids is

enlarged by an *epithelial inlay.* For this an incision is made through the skin of the lids (Esser) or better through the conjunctiva (Waldron), and the subconjunctival cicatricial tissue is cut out without, however, removing the conjunctiva itself. In the cavity so formed a dental-compound mold covered with a Thiersch graft, raw side out, is inserted. The mold is removed in two weeks, either through a new incision, or through the original opening which is left wholly or partially unclosed (McKee, Risdon).

Maxwell takes a flap from the cheek to form a floor for the socket (Posey). Verhoeff uses Thiersch grafts held in place by a large glass ball, the tendency of the tissues to contract being overcome by a pressure bandage applied over the ball. If the resulting cavity is too deep, he permanently implants a glass ball deep in the orbit. Zentmayer also has used a glass ball to support Thiersch grafts in the orbit. Majewski to deepen the fornix makes a deep incision in the back part of it, so that the floor and subjacent tissues of the socket being loosened from their posterior attachment can be drawn down and forward by sutures which are passed through them and then carried down and out through the cheek, where they are tied over a roll of plaster. A very contracted socket may be enlarged by progressive dilatation (Coulomb).

917. Transplantation of Conjunctiva.—This may be done (*a*) for rapidly advancing *ulcers* of the cornea. The ulcers are cleansed by scraping the floor and walls, and then a flap, either with a single pedicle or bridge-shaped, is cut from the conjunctiva of the eyeball close to the margin of the cornea. This flap, is then applied to the surface of the ulcer without being attached to it in any special way. In a similar way a fistula of the cornea (§ 545) may be closed in. (*b*) To cover *wounds*, especially operation wounds, of the conjunctiva, cornea, and sclera. In the case of operations on the cornea or sclera, we form the conjunctival flaps first, and in such a way that the incision in the conjunctiva shall be at some distance from the corneal or scleral incision, and then expose the site of the latter by reflecting the flap (see § 895). When the cornea is cut into or cut away at some distance from the limbus, two or more flaps may be made in the conjunctiva, then drawn up over the cornea and sewed together over the wound (conjunctivo-keratoplasty; cf. § 943). A like principle holds in covering in non-operative wounds of the cornea and sclera. De Wecker in such cases covered the entire cornea by dissecting up the conjunctiva all round the cornea, drawing it down over the latter, and holding it in place with a purse-string suture. This sort of flap is very useful in corneal wounds that are likely to become infected (De Schweinitz). It is kept in place for eight days. Francis covers in scleral wounds and ruptures near the corneal margin with a flap of conjunctiva which is made long enough to pass to the centre of the cornea and meet another flap drawn across from the opposite side. Stitches are passed through the lips of the wound and then mattressed through the proximal flap, which is then laid over the cornea and sutured to the distal flap. For covering in scleral wounds elsewhere, conjunctival flaps are used which are fastened down by sutures passed either through the conjunctiva alone or through the episcleral tissue also (not through the entire thickness of the sclera). For covering defects of conjunctiva, flaps are made of various shapes according to the exigencies of the case. Cf. Beard's operations (Fig. 403, 404) and the operations for symblepharon (Figs. 405, 406).

918. Division and Excision of the Conjunctiva.—(*a*) *Peritomy* consists in dividing the conjunctival vessels running over the limbus either by simply making an incision round the cornea through the conjunctiva, or by excising a narrow annular strip of the latter (the latter operation is also called *peridectomy* and *syndectomy*). It is done in order to get rid of a thick pannus and sometimes also for other conditions (episcleritis, herpes, acne rosacea, chronic ulcers of the cornea). In Agnew's operation, which yields good results, the conjunctiva is divided all round close to the limbus, and pushed back for a space of five mm.; then the bared episcleral tissue in this zone is scraped clean away, and the larger vessels incised or cauterized (Beard). Panas and others perform peritomy by destroying the vessels round the cornea with the cautery (*igneous peritomy*).

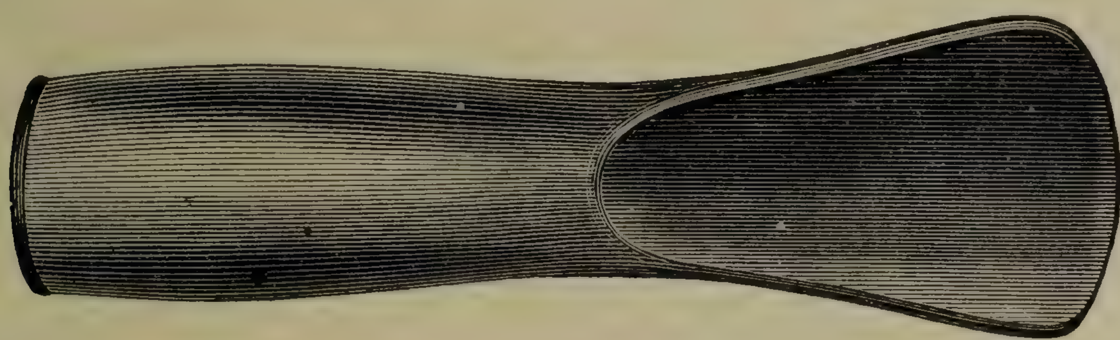
(b) *Excision of the Retrotarsal Fold.*—This operation, advised by Blascovicz, is suitable for cases of trachoma in which the retrotarsal fold is markedly infiltrated, but the tarsus is not involved. The upper lid is everted and the convex margin of the tarsus is grasped with two pairs of forceps. An incision is made in the healthy conjunctiva scleræ, extending from the outer to the inner canthus and close to the line of demarcation between the healthy conjunctiva and the diseased fold. Several sutures are passed through the retracted margin of the scleral conjunctiva, and the latter is undermined. A second incision is made separating the retrotarsal fold from the tarsus, and the fold is then dissected off and removed. The sutures that have been already placed in the scleral conjunctiva are then passed through the upper edge of the tarsus and are tied (Römer). This operation may be combined with excision of the tarsus (see § 929).

CHAPTER XXXIV

OPERATIONS ON THE LIDS

I. Fixation and Splitting of Lids

919. Fixation of Lids.—In many operations it is necessary to have a firm support beneath the lid, upon which the cutting can be done. For this



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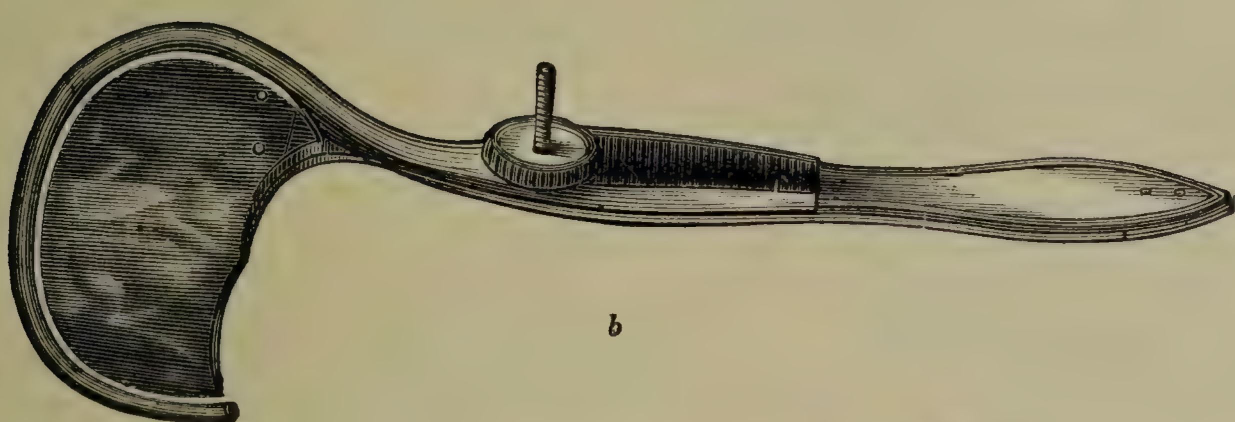

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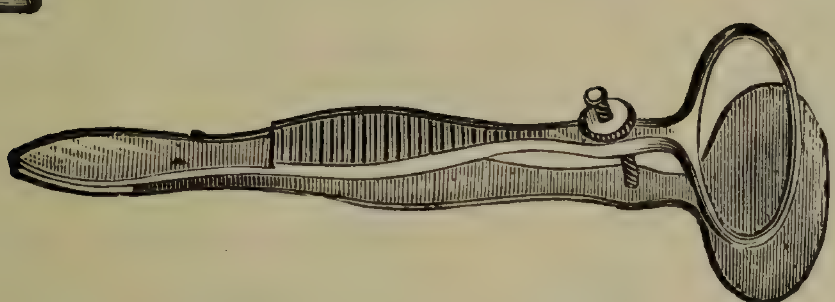

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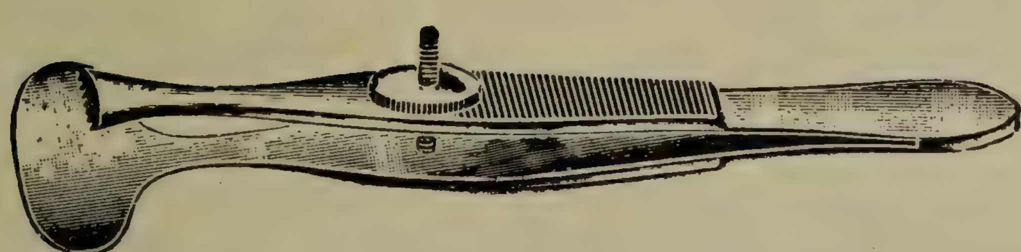

Fig. 409.—Blepharostats for Lid Operations.

a, Jäger's horn plate. *b*, Knapp's lid-clamp. *c*, Desmarre's clamp. The plate in *a*, *b* and *c* is inserted beneath the lid thus affording a resisting surface upon which to cut in all operations upon the lids (operations for trichiasis, entropion, ectropion, ptosis, chalazion, etc.). *b* and *c*, which have a ring that can be screwed down upon the lid, also act as clamps holding the lid firmly in place and preventing hæmorrhage during the operation. Smaller clamps used specially for chalazia are made on the same principle. *d*, Ehrhardt's forceps for double eversion of upper lid in resecting the tarsus and removing the accessory lacrimal gland.

purpose a lamina of horn or shell is used, which is pushed beneath the lid, the lamina being either in the simple form of Jäger's horn plate (Fig. 409 *a*)

or in the form of Knapp's and Desmarre's lid clamps (Fig. 409, *b*, *c*) in which the lid is kept pressed against a plate by a metal ring with a set screw.

920. Splitting the Lid.—In some operations it is necessary to split the lid into two laminæ, the anterior containing the skin with the cilia, the posterior, the tarsus with the conjunctiva. The lid is steadied either by a plate introduced beneath it (§ 919) or (Fig. 410) by being pinched up between the thumb and finger (the pressure in this case controlling the bleeding) and a keratome or scalpel is introduced in the gray line which separates the orifices of the Meibomian glands from the roots of the cilia (*i*, Fig. 2). In doing this it is better to introduce the knife repeatedly rather than to insert it in a single spot and enlarge the incision laterally. When we make this intermarginal incision we get into the loose connective tissue which lies between the tarsus and the orbicularis and which is readily divided.

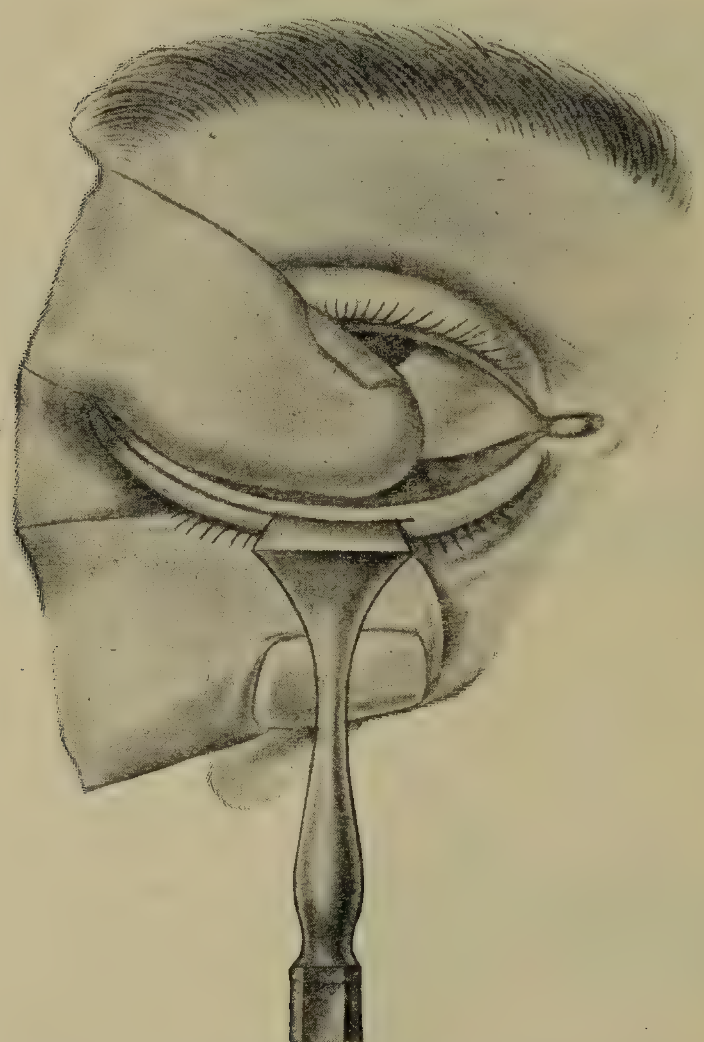

Fig. 410.—Splitting of Lid in Intermarginal Border (after Meller)

II. Removal of Growths on the Lids

921. Operation for Chalazion.—For this, after anæsthetization by procaine injection or infiltration anæsthesia, the lid is everted, and the conjunctiva and the wall of the chalazion lying beneath it are opened by an incision with a sharp-pointed scalpel. After the fluid portion of the contents has escaped, the granulation masses which still remain are removed by curetting. Even then the tumor does not disappear completely, because its resistant capsule remains, although this shrinks up after a short time. If the contents of the chalazion are not completely removed, it is apt to form again, so that the operation has to be repeated. Extirpation (see below) obviates this disadvantage, and is hence preferred to incision by many, including the translator. In operating for chalazion it is usually advantageous to apply a lid clamp (see Fig. 409, *b* and *c*) or a smaller chalazion clamp to stop the bleeding which is generally free enough to interfere with the proper performance of the operation.

Instead of merely opening the chalazia, we may *extirpate* them by dividing the skin over them and then cutting them out of the tarsus. A fenestra is thus made in the tarsus and in the conjunctiva as well. Chalazia, especially of the lower lid, can often advantageously be removed through an incision on the tarsal surface. Extirpation is specially indicated when we are dealing with large chalazia, which project far forward and have a particularly thick capsule.

922. Removal of Lid Tumors.—For vascular tumors we employ *electrolysis.*

The positive pole of a constant-current battery under the form of a plate electrode is placed upon the temple, and then the current is passed through the circuit. The negative pole is armed with a needle which is plunged into the tumor. The current is now turned on, and soon bubbles of gas begin to escape along the needle out of the puncture made by it. The needle is then withdrawn, and the same procedure is repeated at another point of the tumor. Owing to the decomposition of the tissue fluids produced by the galvanic current, the blood coagulates in the vessels, which consequently become obliterated. Several sittings are always required for the complete removal of the tumor.

Other tumors we extirpate according to the usual surgical rules, being careful to remove all the growth and being particularly careful not to cut into cysts. In removing the growth we spare the lid margin if possible and as much of the skin of the lids as we can. In removing malignant tumors we must keep at least a centimetre outside of the visible or palpable border of the neoplasm, and after their removal, the base should be cauterized with the actual cautery and radium used in the open wound. If we have to sacrifice so much of the lid that the eyeball remains uncovered, a substitute for the skin that has been destroyed must be gotten by blepharoplasty (§§ 934, 935). This may have to be done right after the removal of the tumor, but in general in the case of malignant growths it is best to defer plastic operation for some months, as the new skin may cover up a recurrence (New and Benedict). In extensive tumors it is necessary often to remove the eyeball (§ 993), and even the entire contents of the orbit (§ 999).

III. Canthotomy and Canthoplasty

923. The object of these operations is to dilate the palpebral fissure by dividing the outer commissure. The two lids are separated widely with the fingers and drawn toward the nose, so that the outer commissure is put

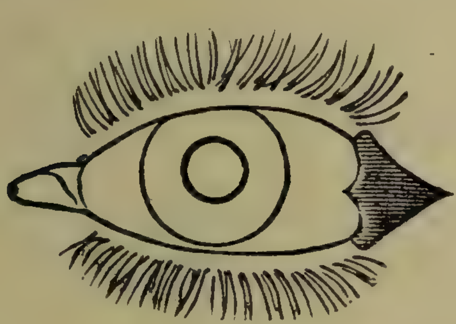

Fig. 411.

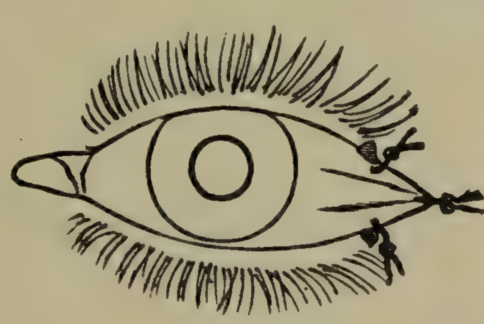

Fig. 412.

upon the stretch, and the blunt-pointed blade of a straight pair of scissors is introduced behind it as far as possible; then, with one snip the skin lying between the blades of the scissors is divided in a horizontal direction; then, upon drawing the lids apart, a rhomboidal wound is exposed to view (Fig. 411). The two outer sides of this wound lie in the skin, the two inner sides in the conjunctiva. In order to make the sides of the commissure draw far enough apart, we divide the canthal ligament. To do this we seize the conjunctival border of the wound with the forceps and drawing on it until the resistance of the fibres (canthal ligament) is distinctly felt, then pass

the closed blades of the scissors into the wound, feel for the ligament and, when it is found, open the scissors blades so as just to include and divide it (Hotz). The operation as thus performed is called *canthotomy.* If nothing further is done the wound soon reunites. This operation is hence used only when all we need is a temporary extension of the palpebral fissure. If a permanent extension of the latter is required (*canthoplasty*) we must cover in the wound surfaces so that they shall not readhere. This is effected as follows. The inner sides of the wound are stitched to the outer by grasping the conjunctiva at the spot where the two borders of the wound come together, and attaching this point to the external angle of the wound by a suture. Then an additional suture, uniting skin and conjunctiva, is passed through the upper and the lower portions of the wound respectively (Fig. 412).

In trachoma it is often difficult, owing to the shrinking of the conjunctiva to draw the latter sufficiently into the skin wound and sew it there. In this case we follow Kuhnt's method and take instead of conjunctiva a narrow *cutaneous flap* which is fashioned from the skin of the lower lid. The flap is cut parallel to the free border of the lid and close to the latter in such a way that its base lies to the outer side of the commissure of the lids so that the flap can be placed in the split commissure without being turned.

The *indications* for canthotomy and canthoplasty are: 1. *Blepharophimosis* and *ankyloblepharon.* In this case a permanent effect is desired from the operation, so that canthoplasty is required.

2. *Blepharospasm,* especially if it gives rise to spastic entropion. Here canthotomy is sufficient. The success of the operation in these cases is attributable not only to the lengthening of the palpebral fissure, but also and mainly to the division of the fibres of the orbicularis, in consequence of which the latter suffer a considerable impairment of strength. If, as is so frequently the case, the spastic entropion is combined with blepharophimosis, we must do a canthoplasty.

3. *Gonorrhœal conjunctivitis,* when the lids are extremely swollen and exert a considerable pressure upon the eye. In this case canthotomy is sufficient. The same thing is true when the enlargement of the palpebral fissure serves as—

4. *A preliminary step* to enable us to remove through the palpebral fissure an eyeball which is very much increased in size, or an orbital tumor.

IV. Tarsorrhaphy

924. Tarsorrhaphy consists in shortening the palpebral fissure by uniting the edges of the lids; it is accordingly the direct opposite of canthotomy. The union of the edges of the palpebral fissure may be effected either at the outer or at the inner angle of the eye (external and internal tarsorrhaphy) or in the middle of the lid (median tarsorrhaphy).

1. *External* (*or Lateral*) *Tarsorrhaphy.*—This according to Fuchs is performed as follows: First, the extent to which it is desired to join the lids together is marked out; then to the same extent the lower lid is split into its two laminæ by an intermarginal incision. From the inner extremity of the section a short incision is carried downward through the skin, thus converting the

anterior lamina of the bisected portion of the lid into a flap (*a*, Fig. 413). The upper and inner borders of this flap are free, while the lower and outer borders are connected with the skin of the lid. The follicles of the cilia, which lie exposed along the posterior border of the upper end of the flap, are removed by a scissors applied flatwise—this being done so that the cilia may afterward fall out. Then the upper lid is denuded by first making the intermarginal section in the same way as upon the lower lid and then ablating the bed of hair follicles thus detached. There is thus produced a raw surface (*b*, Fig. 413), to which it is intended that the skin flap of the lower lid shall adhere by its raw surface. In order that adhesion of the raw surfaces themselves, and not simply of their edges, shall take place, the suture is applied as follows: Both ends of a thread armed with a needle at each end are carried through the upper lid near its free border, the needles being passed from behind forward. In this way the loop of the thread gets to lie upon the conjunctival side of the lid, while the free end comes out upon the raw anterior surface. These ends are then passed through the base of the skin flap below and are tied upon its anterior aspect over a glass bead. By this suture the base of the flap is kept pressed against the raw surface of the tarsus of the upper lid; then, as an additional precaution, the edges of the skin flap are accurately united to the edge of the wound in the upper lid by means of a few fine sutures. The adhesion of the lids obtained by this method is firm enough to withstand even a powerful strain.

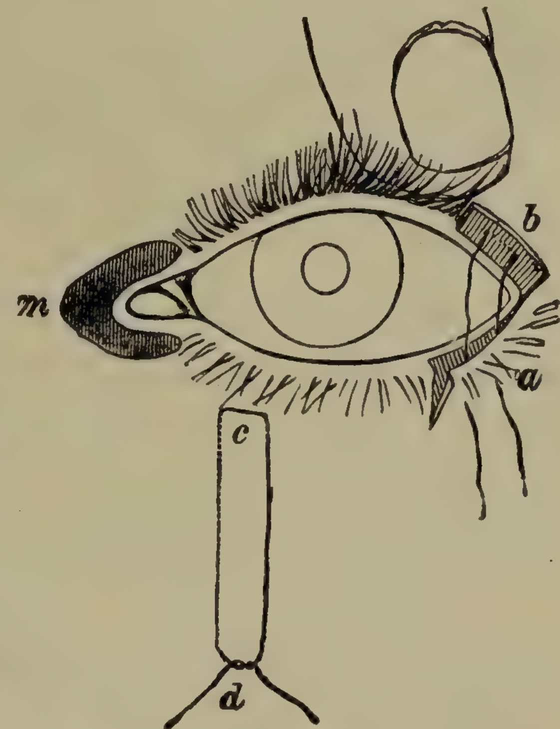

Fig. 413.—Tarsorrhaphy.
A lateral tarsorrhaphy is represented at the external angle of the eye; a median tarsorrhaphy at the internal angle. On the lower lid is shown the position of the loop of thread in Gaillard's suture.

In order to avoid loss of the lashes, Elschnig modifies this operation as follows: After splitting the lid into its two laminæ, he excises a portion of the posterior plate of the upper lid and sews into the defect thus formed a corresponding portion of the posterior plate of the lower lid.

2. *Internal tarsorrhaphy*, as devised by Arlt, is performed by excising with the forceps and scissors a narrow strip of skin from both the lower and the upper lid close to their inner angles. The long and slender wound thus produced should meet in an acute angle at a point on the inside of the inner commissure (*m*, Fig. 413). They are then united to each other through their entire extent by means of interrupted sutures. If a firmer adhesion is desired, this operation, too, like external tarsorrhaphy, may be made with the formation of a small skin flap.

External and internal tarsorrhaphy are indicated—

a. In *ectropion.*—In this the lower lid is raised by attaching it to the upper. Tarsorrhaphy is most effective in senile and paralytic ectropion, and also in slight cases of cicatricial ectropion.

(b) In *lagophthalmus*, because the closure of the palpebral fissure is facilitated by shortening the latter. The form of lagophthalmus that most often gives occasion for the performance of tarsorrhaphy is that which develops in Basedow's disease in consequence of the exophthalmus, inasmuch as here we have no other means of relieving it.

As a rule, external tarsorrhaphy is the operation which is done. Internal tarsorrhaphy is scarcely ever employed except in paralytic ectropion, and is done then only because in this condition the lower lid generally droops more in its inner than in its outer half.

When the lower lid has been in a condition of ectropion for a pretty long time, it is ordinarily found to have become elongated by the stretching it has undergone. In order to make the lid short again, a procedure by which it is at the same time put upon the stretch and pressed against the eyeball, the lower lid, in the performance of tarsorrhaphy, is pared to a greater extent than is the upper. When the elongation is particularly marked, the lid is shortened by excising a triangular piece at its outer extremity. The apex of the triangle is directed downward, and its base corresponds to the free border of the lid. The two sides of the triangular notch are united by means of sutures.

3. *Median tarsorrhaphy* is used when the lids are to be brought together quite completely and for a long time (months or even years) but are ultimately to be separated again. It is hence done in blepharoplasty to keep the lid in position during healing (§§ 932, 934) and in neuroparalytic keratitis when the eye is to be kept covered for a long time (§ 560). For method of performing it see § 932 and Fig. 429.

Median may be combined with external tarsorrhaphy when the latter is done under great tension—e. g., when the attempt is made to bring closer together lids that have become shortened, or when the operation is done in a case of exophthalmus. In this case median tarsorrhaphy may be done simply by inserting sutures without paring the edges of the lids, so that the union shall be only a provisional one. The sutures are left in until they cut their way through, or until the healing of the wound made by the external tarsorrhaphy has become sufficiently firm.

V. Operations for Entropion and Trichiasis

925. Apart from the disfigurement it occasions, entropion is mainly of significance because it causes trichiasis. The relief of the latter is therefore the main object of entropion operations. Entropion producing trichiasis may be either spastic or cicatricial, and trichiasis or distichiasis may occur without entropion.

926. Removal of Cilia.—The simplest way of curing trichiasis and distichiasis—applicable specially when there are but a few misplaced cilia and more particularly when there is no condition causing a progressive entropion—is to remove the inturned lashes. This is best effected by *electrolysis.*

The two poles of a constant-current battery are so arranged that the positive pole is formed by a plate electrode, the negative pole by a slender needle. The former is applied to the temple, and the latter is introduced into the hair follicles of the cilium, and then the circuit closed. At once a light foam is seen to exude from the root of the cilium. This is formed by the bubbles of hydrogen gas which are developed at the negative pole

and give evidence of the chemical decomposition of the tissue fluids produced by the electric current. By virtue of this decomposition an adequate destruction of the hair follicle results without any eschar being produced. The cilium can now be very readily drawn out, or it falls out afterward of itself, and it never grows again. The operation is pretty painful, and hence it is a good plan to inject procaine beneath the skin of the lid near its free border, or use infiltration anæsthesia. Electrolysis is used especially when there are only a few misplaced cilia that require removal.

927. Operations for Spastic Entropion with Trichiasis.—Spastic entropion develops only when the skin of the lids is abundant and relaxed (see § 438). If then the skin is put upon the stretch by pinching up a horizontal fold of it in the fingers, the entropion disappears. Upon this observation depend those methods of operating for entropion which produce contraction of the skin of the lid in a vertical direction. The methods of operating that are most in use are:

1. *Gaillard's Suture.*—This operation as modified by Arlt is performed as follows: One needle of a double-armed thread is entered at the junction

FIG. 414.—GAILLARD-ARLT'S OPERATION FOR ENTROPION.

A, sutures seen from in front. B, seen in vertical section; situation of the loop. C, the same after the loop has been drawn tight. B and C magnified 2 × 1.

of the middle and inner thirds of the lower lid. The point of entry lies close to the border of the lid (*c*, Fig. 414 B), the point of exit at a distance below it upon the cheek equal to about the breadth of the thumb (*d*). The second needle is passed in a similar fashion and near the first, so that the bight of the thread lies upon the skin near the border of the lid (Fig. 414 A,) and the two threads run downward beneath the skin of the lids in a parallel direction. A similar loop of thread is applied at the junction of the middle and outer thirds of the lid. If the two ends of each thread are tied over a small roll of adhesive plaster or of iodoform gauze and drawn tight, a horizontal fold of skin is pinched up upon the lower lid (*a*, Fig. 414 C), and thus the entropion is made to disappear.

In order to produce a sufficient effect, the threads are drawn tight enough to produce a slight degree of ectropion, which subsequently disappears. The threads should be left in until cicatricial bands, which shall produce the same effect that the threads did, form along the channels made by the threads. The entropion is apt to set in again subsequently in spite of the operation. The latter, accordingly, is adapted to those

cases only in which we are dealing with an entropion of presumably short duration, as, for example, that form which develops beneath a bandage.

Instead of Gaillard's suture we may also employ one of Snellen's (not to be confounded with Snellen's suture for ectropion). This was modified by Stellwag in the following way: "One or two stout threads armed with curved needles at both ends are passed through the entire thickness of the lid, starting from the deepest portion of the retrotarsal fold of the conjunctiva (*a*, Fig. 415), so that they form at the fundus of the conjunctival sac one or two loops four to five mm. in length and placed parallel to the free border of the lid. Each needle is next carried back through its point of exit in the external covering of the lid (*b*), then is passed vertically between the latter and the fibro-cartilage to a point just at the external lip of the lid, there (*c*) is brought out again, and the two ends of each thread are drawn as tight as necessary over a roll of adhesive plaster and tied."

FIG. 415.

2. *Excision of a Horizontal Strip of Skin.*—A horizontal fold of skin is pinched up between two fingers, its size being so gauged that the entropion shall be relieved by it without, however, an ectropion or a lagophthalmus being produced; then this fold is snipped off with one stroke of the scissors, and the two edges of the wound are next united by means of some sutures. If the operation is to be effectual, the upper edge of the wound must lie pretty close to the free border of the lid. The result of this operation is ordinarily permanent, although here, too, recurrences of the entropion sometimes take place through subsequent stretching of the skin. Hence the much more complicated Hotz's operation (§ 928), which fastens the skin of the lids to a fixed point, the attached border of the tarsus, is also employed in spastic entropion.

3. *Excision of a Vertical Strip of Skin.*—If we push backward upon the entropionized lid in the region of the orbital margin, the lid goes back into

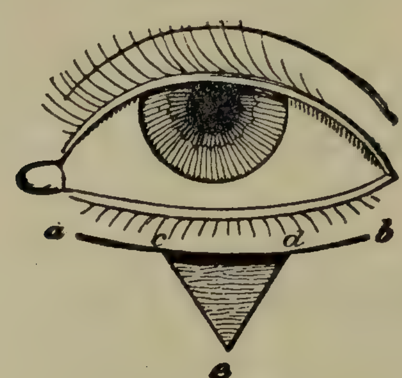

FIG. 416.
Operation for entropion by Von Graefe's method. Way of making the incision.

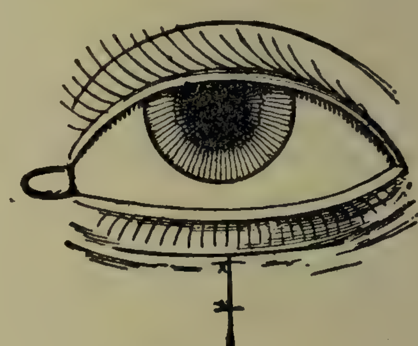
FIG. 417.
The same after the edges of the wound have been united.

place. We make use of this fact when we try to keep the lid in the proper position by means of an appropriately placed bandage (§ 438); but we can also secure a permanent effect of this kind by shortening the skin of the lid at the margin of the orbit in a horizontal direction, so that it becomes tense and presses the lid backward. We first make an incision *ab* (Fig. 416) parallel to the lid and then from the extremities of the middle third of this incision make the converging incisions *ce* and *de* downward as far as the margin of the orbit. The piece so delimited is cut out, and then the two lateral borders of the incision, after the connections have been loosened up

sufficiently, are united in a vertical direction (Fig. 417; Von Graefe). The scars resulting from this operation are rather more visible than those resulting from the excision of a horizontal strip of skin, because they run perpendicular to the direction of the cutaneous folds; but, on the other hand, this operation affords more security against relapses.

4. *Galvano-puncture.*—In this method a series of punctures parallel with the lid margin and two or three mm. from it are made through the skin and well down into the tarsus with a galvano-cautery point (Ziegler). This gives very good results.

5. *Canthotomy,* since it relieves blepharospasm, may also be employed to relieve spastic entropion. In those cases in which blepharophimosis is also present, the canthotomy must be made with a conjunctival suture; in other cases a temporary canthotomy often suffices.

928. Operations for Cicatricial Entropion with Trichiasis.—In these the aim is either to give the inverted cilia a proper direction by transplanting the zone of hair follicles or to get at the cause of the trouble—the distortion of the tarsus—by straightening the latter.

The main methods are as follows:

1. *Hotz Operation.*—This operation was first proposed by Anagnostakis. When it is made upon the upper lid an incision is first carried through the skin of the latter, passing along the upper border of the tarsus from one end of it to the other. Then the lips of the wound are separated, and the bundles of fibres of the orbicularis which are visible at its bottom are exsected in a breadth of four mm., so that the tarsus is laid bare. If the tarsus is very thick it is thinned by shaving off thin slices from its front surface with a scalpel. Then three or more sutures are applied. Each of these passes first through the upper lip of the cutaneous wound, then through the upper border of the tarsus, and lastly from within outward through the lower lip of the cutaneous wound (Fig. 418). When the sutures are tied down, the wound is closed and the lower end of the lid is rotated outward (Fig. 419). The idea underlying this operation is to raise the bed of hair follicles, by attaching the skin to a fixed point—namely, the upper border of

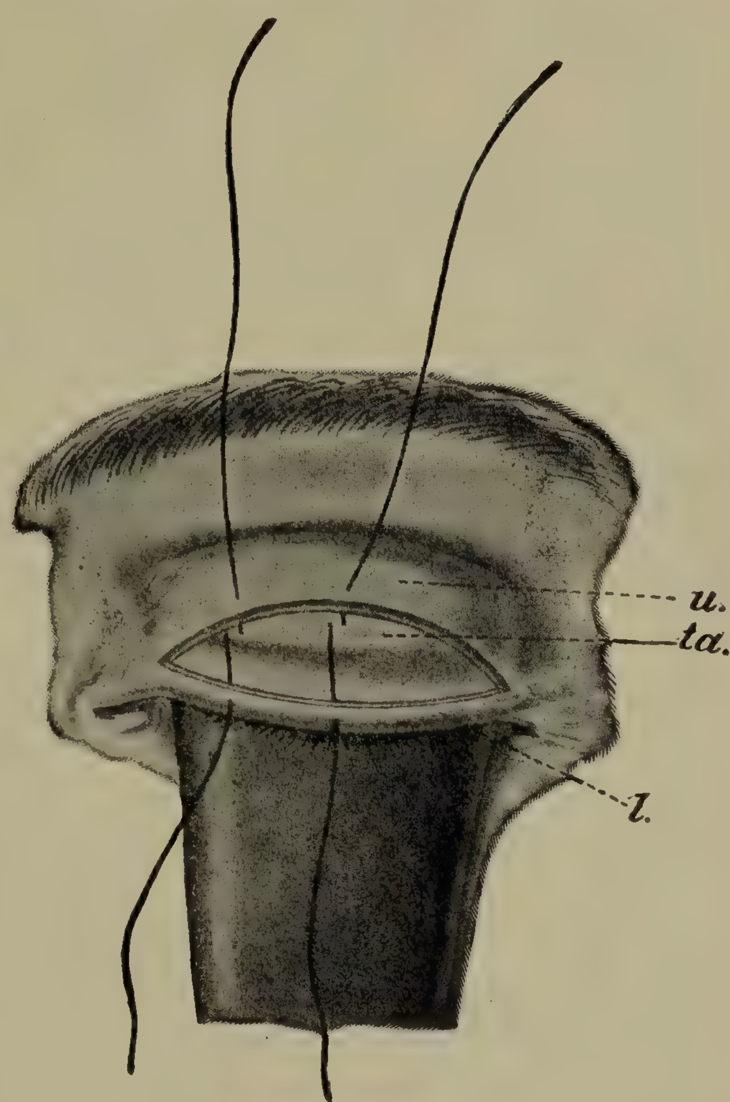

Fig. 418.—Hotz Operation; Application of Sutures (after Meller.)
The figure shows two of the sutures in position. They are entered from above, passing successively through the skin, *u*, upper border of tarsus, *ta*, and the rim of skin, *l*, above the cilia. Cf. Fig. 419.

the tarsus. The exsection of the fibres of the orbicularis is done with the intention of diminishing the power of this muscle which tends to force the lid backward.

In the lower lid the method of operating is the same, but the cutaneous incision, in accordance with the smaller height of the tarsus, runs closer to the free border of the lid.

In *Oettingen's* operation the incision in the intermarginal space (§ 920) is carried to a point above the upper border of the tarsus, so that all the skin which covers the tarsus can be displaced upward. This portion of skin is then, by means of sutures, attached near its free border which carries the cilia, to the upper border of the tarsus (Fig. 420). Below the free border of the lid, which has been thus elevated, there remains quite a large wound, formed by the anterior surface of tarsus. This defect may be covered with a flap taken from the mucous membrane of the lip. This method is applicable when there is a very irregular distribution of the cilia. Upon the same principle depend the methods of Kostomyris, De Wecker, and Warlomont.

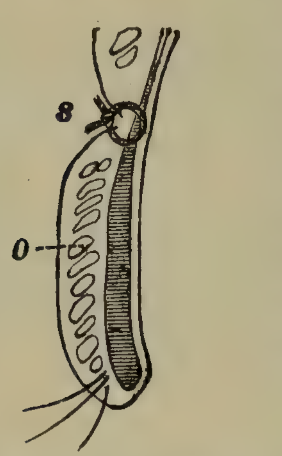

Fig. 419.— Hotz's Operation After Tying Sutures.—*o*, fibres of orbicularis; *s*, suture tied.

2. **Panas's Modification of Snellen's Operation.**—Snellen in order to secure proper rotation of the lid excised a wedge-shaped piece from the tarsus. In Panas's modification the operator divides the skin two or three mm. above the free border of the lid and parallel to the latter (*i*, Fig. 421 A), the incision running the whole length of the lid. Then, beginning at this incision, he dissects up the skin as far down as the free border of the lid and as far up as the upper border of the tarsus. Next, cutting down upon the horn plate, he makes an incision which runs in the same way as the incision in the skin, and divides the tarsus through its entire thickness, including the conjunctiva tarsi. By this incision the lower half of the tarsus, together with the free border of the lid, is made freely movable, and by means of sutures can readily be rotated forward far enough for the cilia to be properly directed. The sutures are formed into loops and passed above through the edge of the tarsus and the tarso-orbital fascia, and the free ends of the loop are brought out behind the skin of the lid along the intermarginal line, and here are tied over a glass bead (Fig. 421 B).

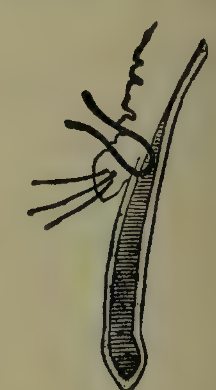

Fig. 420.—Oettingen's Operation. Magnified 2 × 1.

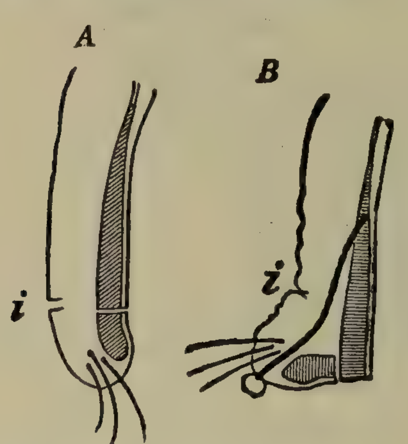

Fig. 421.—Panas's Operation.
A, after completing the incisions in the skin and tarsus. B, after applying the suture.

The operation of Panas ensures against relapses, which may occur after Hotz's operation, although they do so rarely if in performing the latter the tarsus is properly thinned so as to be made pliable enough. On the other hand, in the operation of Panas there is some danger of necrosis, and if the rotated segment is too large there will be lagophthalmus. Epdawi in doing the operation, after cutting down to the tarsus, removes the lower part of the orbicularis muscle—thus rendering the end of the lid thinner and more shapely.

Both operations exert their greatest effect in the middle of the lid and are of special service when the trichiasis is greatest here. When the trichiasis is present simply at the temporal or nasal end of the lid, *Spencer Watson's operation* is the best. In this an incision is made in the intermarginal space, and a second one is made parallel to the border of the lids and above the row of cilia (Fig. 422, in the outer half of the upper lid). Then, instead of detaching the strip of skin, thus marked out, at both ends,

it is separated at one extremity only. The bed of hair follicles is thus transformed into a long, slender flap which is free at one extremity, but at the other is connected with the skin of the lids (*a*, Fig. 422). Then a second flap of skin similar in shape is fashioned by making a second incision about three mm. above the first and parallel to it, and thus marking out a narrow strip of skin, which is likewise left in connection with the skin of the lid by one extremity only (Fig. 422, *b*). The base of this flap must lie at the temporal end of the lid, provided the base of the lower flap, which bears the cilia, is situated at the nasal end, and vice versa. Then the two flaps are interchanged, so that the one which carries the cilia gets to lie above, and the one that was above gets to lie below, along the free border of the lid (Fig. 422, a_1 and b_1). The flaps are kept in their place by sutures. This operation should not be performed along the whole length of the lid, since the flaps then would be too long in proportion to their narrow base, and hence would be likely to die.

Gayet, Jacobson, Dianoux, and Landolt likewise employ flaps with a pedicle.

Each of the methods named has certain *advantages* and disadvantages. The experienced operator will not employ any one of them exclusively, but will select the method to suit the case in hand, and when necessary will even combine two different methods. Thus Hotz's method may be combined with the excision of a strip of skin, with the detachment of the bed of hair follicles by a supplementary intermarginal incision, or, if the distortion of the tarsus is a very conspicuous feature, with the excision of a wedge from the latter. If *normally placed lashes* or both *lid border and lashes* are missing these may be replaced by a dermic graft (§ 934) taken from the eyebrow containing hair (Esser). The defect in the eyebrow may be replaced by a pedicled flap taken from the scalp or may be simply brought together by sutures (Wheeler).

Fig. 422.—Spencer Watson's Operation for Trichiasis.

This is represented as being performed in such a way that the flaps do not extend over the entire length of the lid, but only over one-half of it. The outer half of the upper lid shows the flaps in their natural position; the inner half shows the position of the flaps after they have been interchanged.

929. Excision of Tarsus (Tarsectomy).—This is done not only to relieve trichiasis, but also to effect a radical cure of its underlying cause, trachoma. Two operations are done:

(*a*) *Partial Tarsectomy with Excision of the Retrotarsal Folds.*—As performed by Blaskovicz, this operation is advised in trachoma with marked infiltration of both the tarsus and retrotarsal folds. The upper lid is everted and an incision parallel to the lid border is made from outer to inner canthus through the healthy scleral conjunctiva demarcating it from the diseased fold. Three sutures are introduced through the lower lip of the conjunctival wound in such a way that each forms a loop two mm. broad, with its bight on the epithelial surface of the conjunctiva. Traction is made with these, and the scleral conjunctiva is dissected far down (nearly or quite to the cornea—Orcutt). An incision is then made through tarsal conjunctiva and tarsus, parallel with the free border of the lid and 2.5 or 3 mm. from it, so as to delimit all the diseased portions of tarsus and conjunctiva. These diseased portions are dissected out, care being taken not to injure the orbicularis or Müller's muscle. The piece thus removed measures about 2.5 cm. long and one cm. broad. The conjunctiva is then drawn up by the looped sutures, which are passed through the lid from within outward entering the stump of the tarsus one mm. in front of its cut edge, and emerging on the skin surface where they are loosely tied (Römer).

For Gillet de Grandmont's tarsectomy see § 936.

(*b*) *Total Tarsectomy.*—This operation, devised by Heisrath, has been modified by Kuhnt. It is used in the cicatricial stage of trachoma, when the tarsus is degenerated and incurved and the conjunctiva is wholly or in large part cicatricial. The lid is everted, and an incision made through conjunctiva and tarsus close to the free border of the lid and parallel with it. The cut segment of tarsus is then dissected from its attachments and particularly from the conjunctiva, which is no longer tightly adherent to it (see § 468). The dissection is carried as far as the upper border of the tarsus. The insertion of the levator is then divided, and the tarsus, being now free from attachments, is removed. The cut edge of conjunctiva is sutured to the rim of tarsus left at the free border of the lid (Römer).

VI. Operations for Ectropion

930. Operations for Spastic Ectropion.—In spastic ectropion, which does not yield to reposition and the application of a bandage, *Snellen's suture* gives the best service. Like Gaillard's suture in entropion (§ 927), it consists of two loops, one of which is situated at the junction of the external and middle thirds, the other at the junction of the middle and inner thirds, and which are passed downward (or in the upper lid upward) beneath the skin of the lid. The point of entry, however, is different. In Snellen's suture this lies at the summit of the ectropionized conjunctiva—that is, under ordinary circumstances close to the posterior margin of the tarsus (*a*, Fig. 423 A). From this point the needle is passed down beneath the skin of the lid to a point about at the summit of the lower margin of the orbit, and there is brought out again (*b*). The second needle with the other end of the thread is carried down close to the first and parallel with it. The two extremities of the thread which thus come to view upon the cheek are tied over a roll of adhesive plaster or iodoform gauze and drawn tight until a slight degree of entropion (Fig. 423 B) is produced. The same thing is done with the second loop. The mode of action of the operation depends upon the fact that the protruding portion of the conjunctiva, which is caught in the loop, is drawn downward and forward in the direction of the skin. This suture can also be employed in senile ectropion, although in this case it usually has no permanent effect.

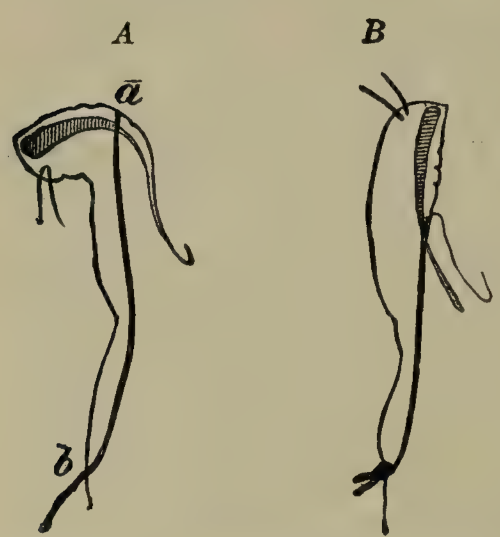

Fig. 423.—Snellen's Suture for Ectropion.
A, before tightening the loop. B, after tightening the loop.

931. Operations for Paralytic and Senile Ectropion.—*Paralytic ectropion* indicates the performance of *tarsorrhaphy.* The latter is also frequently performed in *senile ectropion,* although in the latter, *Kuhnt's operation* gives much better results. This is nothing but a modification of the old method of Antyllus. It depends upon the fact that the lid when ectropionized is elongated, and that, when it has been made stiffer by being

shortened, it becomes applied to the eyeball. The shortening is accomplished by cutting out a triangular piece, the base of which must lie at the border of the lid, since here the elongation is most considerable. In order that a coloboma of the lid may not be produced, the excision should not affect the entire thickness of the lid, but only its inner layers—namely, the conjunctiva and tarsus, (see Fig. 424).

The operation as performed by Meller is a combination of Kuhnt's and Dieffenbach's (see § 933). In this we first determine the amount of tarsus to be excised by grasping the lower lid with two anatomical forceps and bringing these together so as to pinch up the tarsus in the middle of the lid and make it protrude. The amount of tarsus that has to be thus pinched up in order to make the lid fit snugly to the eye indicates the area that should be excised. Suppose this included in the line *de* (Fig. 424), we then split the lid by an incision in the intermarginal line extending from *e* to *b*.

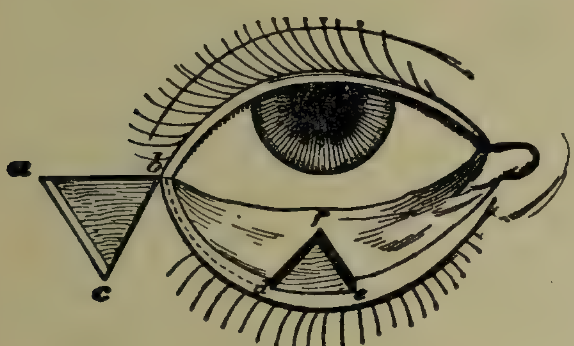

FIG. 424.
Operation by the method of Kuhnt and Dieffenbach. Way of making the incision.

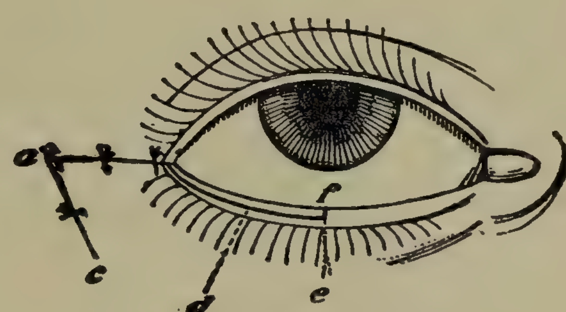

FIG. 425.
The same after the edges of the wound have been united.

From the two points *e* and *d* we carry two converging incisions backward through conjunctiva and tarsus to *f* and excise the triangular piece so delimited. We further cut out the triangle of skin *abc*. Next we displace the cutaneous lamina of the lid to the temporal side, sliding it on the lamina containing the mucous membrane until *ef* gets to lie against *df* and *bc* against *ac*. Then these cut edges are united by stitches (Fig. 428, *ef* and *ac*).

This operation, which gives excellent results, is also called the Kuhnt-Meller and Kuhnt-Szymanowski operation.

Slight cases of senile ectropion may be relieved by the moderate cicatricial contraction produced by a row of *galvano-cautery* punctures made in the tarsus four mm. from the lid border and parallel with it, the punctures being four mm. apart (Ziegler).

932. Operations for Cicatricial Ectropion. Blepharoplasty.—With respect to cicatricial ectropion, the least serious cases are those in which not much skin has been lost and the contraction of the lid is caused simply by a few cicatricial bands, especially if these draw the lid in against the bone and attach it to the latter—a condition which so frequently remains after caries of the margin of the orbit. In such cases the cicatricial bands may either be *divided* subcutaneously or be cut out altogether, according to their situation, and the cutaneous wound be closed again by sutures. The effect of such an operation is made more thorough and also more lasting by a tarsorrhaphy performed at the same time. If, however, much of the skin

of the lid has been lost, we will have no success with this simple operation, but must proceed to the performance of *blepharoplasty,* the object of which is to replace skin that has been lost. Blepharoplasty is used not only in cases of cicatricial ectropion, but also to cover large defects produced by operations, e. g., those for removal of tumors of the lids. It may be done by the implantation of a pedicled flap or a skin graft.

Before implanting a flap into the lid, the latter must be properly prepared. The cicatricial portion of the lid is first divided by an incision parallel to the free border of the lid, and then the more deeply situated cicatricial bands are also cut through until the lid is freely movable and can be brought into its normal position without any strain being put upon it. Those portions of the cicatricial skin which appear to have but little vitality are excised, in doing which, however, the free border of the lid, wherever it has been preserved, is spared as far as possible; then the lid is brought into the proper position and retained there by being united to the other lid. A good way of doing this is to denude three areas, each three mm. long, on the border of the upper lid just inside the eyelashes and just opposite these, three like areas on the lower lid, and then bring these opposite areas into contact by double-armed sutures. Each of these latter entering through the skin of the lower lid is passed first through the lower, then through the opposite upper denuded area and emerges through the skin of the upper lid border where the two ends are tied down on a rubber plate (Weeks). (See Fig. 429). After the lid has been thus put in place, the incision which was made for the purpose of releasing it gapes widely so as to form quite a large raw surface, which must be covered with skin.

933. Blepharoplasty for Defects not Involving the Entire Thickness of the Lids.—In many defects, especially those due to ulceration or gangrene, the conjunctiva and lid border and in most cases at least part of the thickness of the tarsus are left. Such defects, in which there is a smooth posterior epithelial surface and a framework to build on, can be covered in with skin either in the form of pedicled (sliding) flaps or of non-pedicled skin grafts. The transplanted portion of skin must be of the shape of the defect to be filled, but a third larger in all its dimensions since it always shrinks, (except in skin grafts taken from the upper lid—see § 934).

Sliding skin flaps.—The methods of covering the raw surface with sliding flaps are very numerous, varying according to the size and shape of the defect. In all cases, in order that the nourishment of the flap shall be satisfactory, its base must be made sufficiently broad and must not be twisted too much in the act of transplantation. For the same reason the flap should not be dissected off clean, but the subcutaneous fat together with the vessels running in it, should be included.

Following are some of the principal methods:

1. *Fricke's* method is especially adapted for elongated losses of substance, whether upon the upper or the lower lid. To cover these defects a tongue-shaped flap (*L*, Fig. 426) is fashioned, the base of which adjoins one end of the loss of substance (*S*). The flap is most commonly taken from the skin of the temple and the cheek and is rotated so as to fit into place,

being then attached by sutures to the edges of the defect which have previously been rendered movable by undermining. The raw surface which remains at the spot where the flap has been dissected off can usually be diminished considerably in size by means of sutures; the remaining surface is allowed to heal by granulation or it is covered with Thiersch grafts. At the base of the flap a swelling is formed, due to twisting and larger in proportion to the amount of the twisting. This swelling flattens afterward so as to become less perceptible; should it cause disfigurement, it may be excised later.

2. *Dieffenbach's* method becomes applicable whenever the defect either has the form of a triangle (the base of which looks toward the border of the lid), or can be readily brought into the triangular form (*s*, Fig. 426). It is better adapted to the lower than to the upper lid. The flap (*l*) is ordinarily taken from the temporal side of the defect—i. e., from the cheek. An incision is made toward the temporal side along the prolongation of the base of the triangle. It should be somewhat longer than the latter, in consideration of the shrinking which the flap will undergo. From the outer extremity of this section (*a*) a second incision is made downward. In this way a quadrilateral flap is circumscribed, the base of which is situated below.

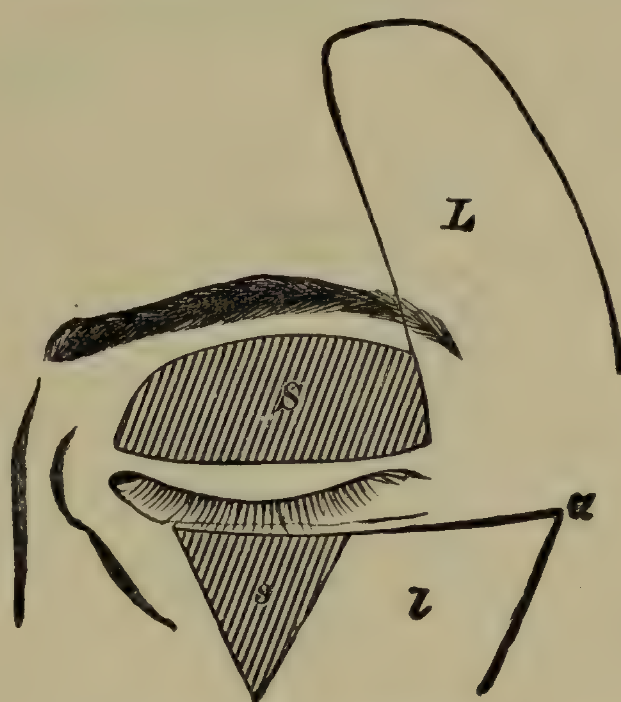

FIG. 426.—BLEPHAROPLASTY.
The method of Fricke is presented on the upper lid; that of Dieffenbach on the lower. (The flap *b* should be rhomboidal instead of rectangular).

Figure 426 illustrates Dieffenbach's original method. In the Arlt-Blascovicz modification, which gives the best results, the upper edge of the sliding flap, *l*, is not carried straight out but down and out, and the outer side of the flap curves toward the inner so that the flap is wedge-shaped instead of rectangular, its base being narrower than its top (Török and Grout).

This flap is now detached by dissection, and is turned slightly toward the nasal side so as finally to lie upon the loss of substance, to which it is then attached by sutures. The raw area which remains at the spot from which the flap was taken is made as small as possible by means of sutures, and the remainder of it is allowed to heal by granulation, or later may be covered with a Thiersch graft (§ 934).

3. The *Wharton-Jones* operation is applicable to scars near the lower lid margin. A V-shaped incision with its apex downward is made so as to include the scar. The triangular piece of skin, thus delimited, which includes the scar tissue, is dissected up to near the lid margin, and the eyelid, being thus released is shoved up into its proper position, carrying the triangle of skin with it (Fig. 427). Below this there is now a raw spot shaped like an arrowhead pointing downward. To cover this in, the skin

on either side of the V is undermined, and then the lower portions of the V are stitched to each other, and the upper portions are stitched to the displaced triangle of skin. The arrow-shaped wound is thus converted into a Y-shaped scar.

4. A *quadrangular defect in the middle of the lower lid* may be covered by two somewhat curved oblong sliding flaps, which are brought in from each side till they meet and are then sewed together (Knapp-Celsus, Blascovicz). When the flaps are brought together a crescentic defect may be left below, which is filled in by sliding a tongue-shaped flap from the side.

5. For a *defect at the inner canthus* Blascovicz after removing the scar and freeing the lower lid, carries the latter in so as to cover the defect. In order to hold it more securely in place he makes at the inner end of the flap an incision four mm. long parallel with the lid margin and distant four mm. from it. This incision gapes. Into the gap he dovetails a flap five mm. long drawn from the skin above the canthus (Grout).

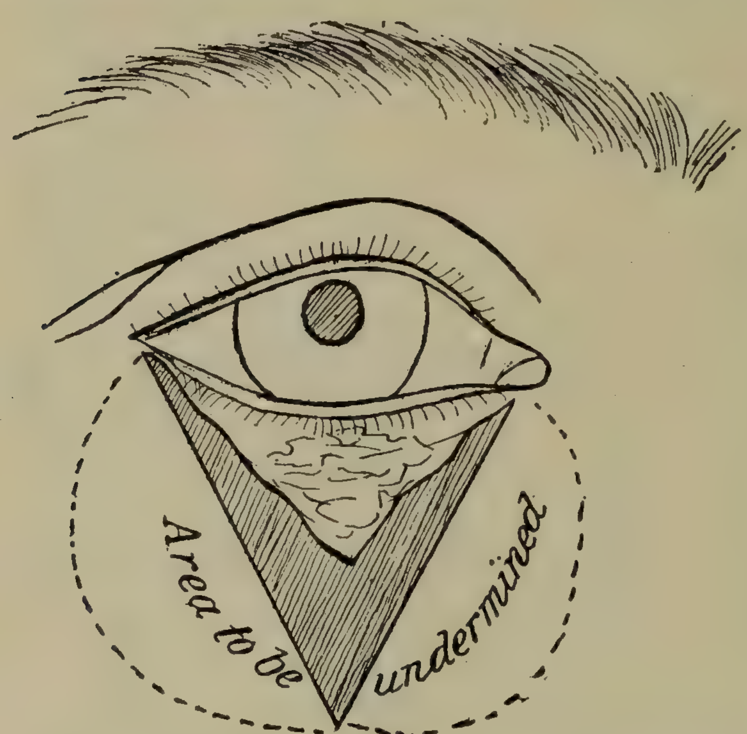

FIG. 427.—WHARTON JONES OPERATION (after Beard).—The dissection is shown with the triangle of skin containing the scar shoved up into place.

6. For a defect *above the inner canthus*, Landolt after splitting the upper lid and forming it into a quadrilateral flap, slides this flap along to cover the defect. This leaves a raw spot above the outer canthus, which heals by granulation.

934. Non-Pedicled Flaps; Skin Grafts.—The cosmetic results obtained with pedicled flaps usually leave much to be desired. Much more satisfactory are the results obtained with skin grafts when properly applied.

In one method of skin-grafting, very thin pieces are taken which contain only the most superficial layers of the skin—namely, the epidermis, the rete Malpighii, and the apices of the papillæ (*epidermic graft*). In another method portions of skin are used which include the entire thickness of the cutis, and which are transferred to the loss of substance either cut up into small pieces, or under the form of flaps of some size (*dermic graft*). The skin may also be taken from other persons or from amputated limbs with sound skin. (Dermic grafts are also called *Le Fort* and *Wolfe* grafts from Le Fort who first employed them and Wolfe who popularized their use in blepharoplasty). A position midway between the epidermic and the dermic grafts is occupied by *Thiersch grafts*, which are of comparatively large size, but which besides epithelium contain only the most superficial layers of the cutis.

In operations on the lids, dermic grafts, especially those taken from the upper lid or from the temple give excellent cosmetic results (Wheeler).

These soon get to look like the skin surrounding them. When these spots are not available, grafts may be taken from some other part, such as the inner surface of the arm, the skin of which is thin and as nearly as possible free from hair.

The lids are stitched together in the way described in § 932. All cicatricial tissue is carefully removed leaving a clean raw surface which is to receive the graft. A graft is then cut corresponding to this raw area and somewhat larger (one-third larger if cut from

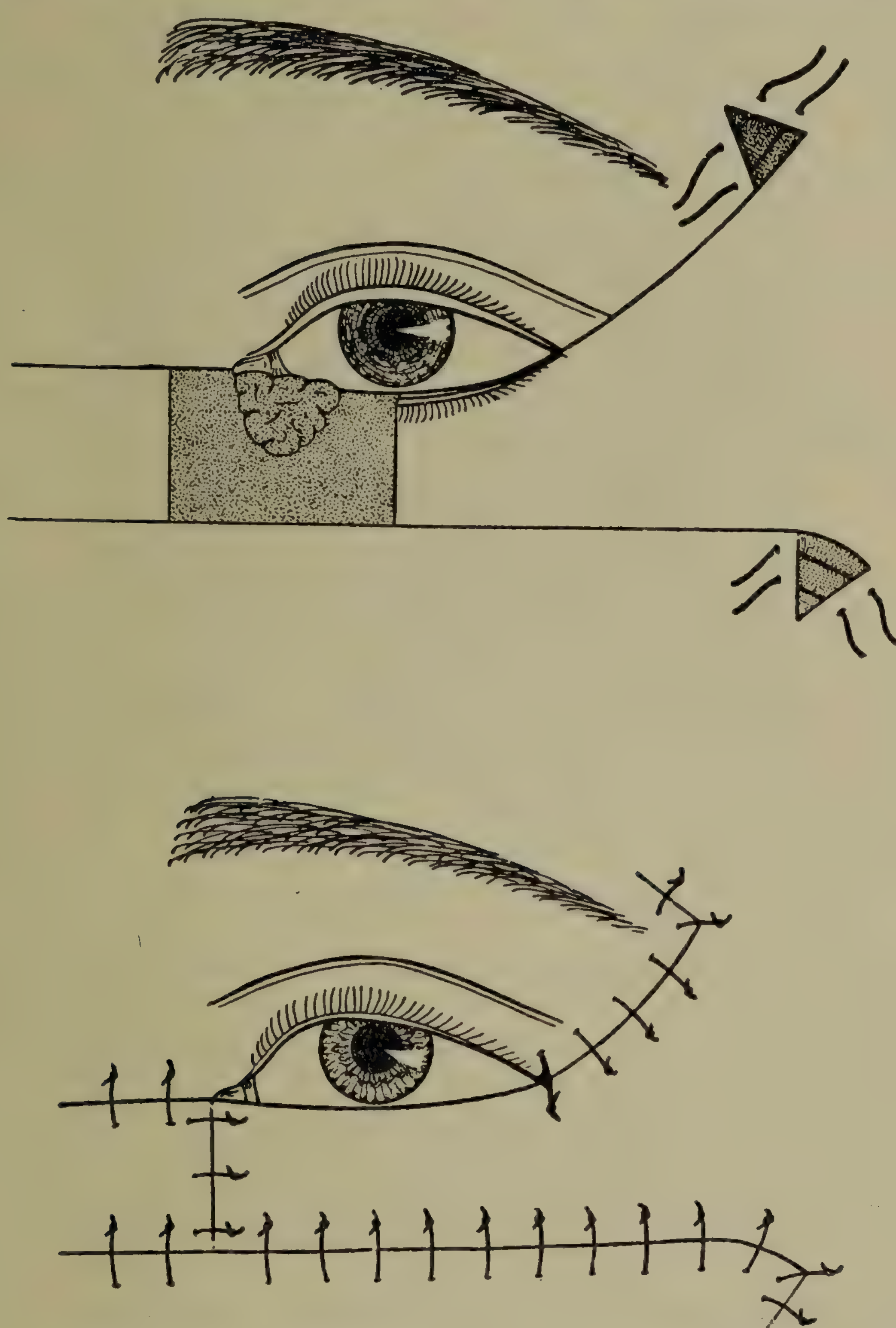

Fig. 428.—Celsus-Knapp's Blepharoplasty with Burrow's Triangles (after Török and Grout). By the excision of the little triangles the outer flap is permitted to slide in without undue tension. Cf. Fig. 406 B.

the arm, only slightly larger if cut from the temple or lid). Great care is taken not to pinch or otherwise injure the graft, which after being outlined with a scalpel is sliced off with a Graefe knife. When the graft is detached it is laid on a piece of gauze wet with warm saline solution, and all subcutaneous tissue trimmed away. It is then sewed in place with interrupted sutures, and covered with rubber tissue slightly smeared with vaseline. Over this, gauze is packed and fastened in place with adhesive plaster and a very firm pressure bandage. The dressing is renewed in four days and kept up for two

weeks. The adhesions connecting the lids are left uncut for three months. The raw surface from which the graft is taken is covered in by undermining its edges, applying relaxation sutures to bring the edges closer together, and then sewing the latter together. Defects of the lower lid repaired by a graft from the upper lid of the same eye or defects of the upper lid repaired by graft from the upper lid of the other eye give remarkably good cosmetic results.

In blepharoplasty, skin grafts may be applied not only on the free surface as in the method above described, but may be buried in the tissues. Thus, in bad cases of ectropion Gillies after dissecting out all the scar tissue and undermining the surrounding skin, inserts into the subcutaneous cavity thus formed a dental-compound mold which is

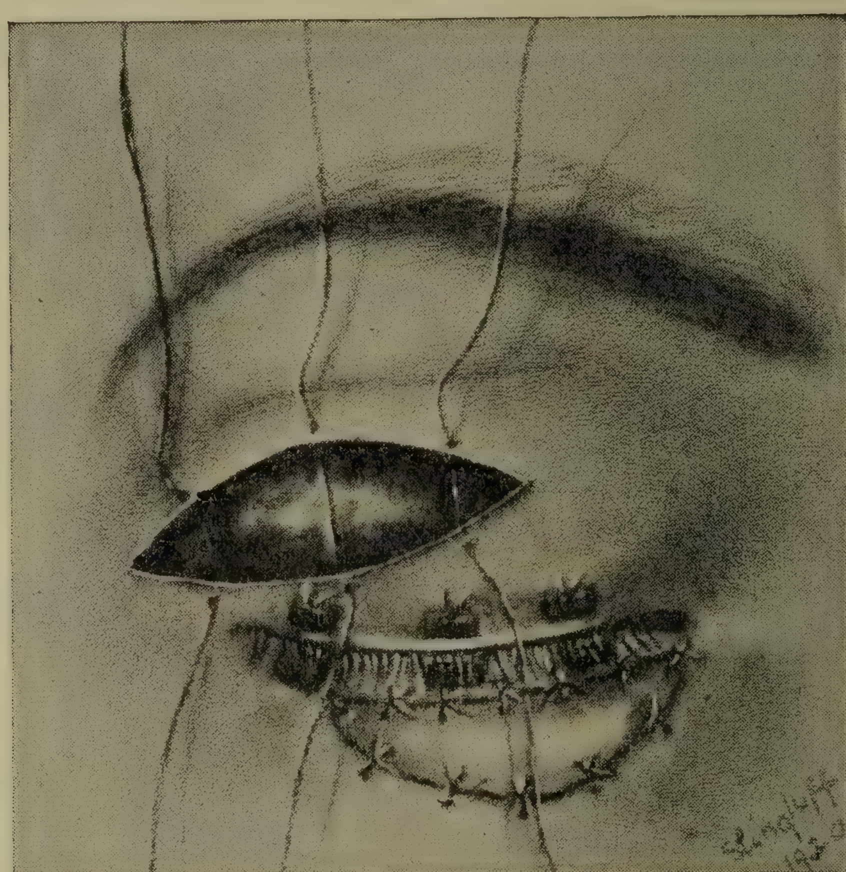

FIG. 429.—DERMIC GRAFTS IN BLEPHAROPLASTY (after Wheeler).—Skin graft from upper lid sutured in place to cover raw area in lower lid; sutures ready to tie to close wound in upper lid from which graft has been taken. Lids stitched together by double-armed sutures tied over rubber tubing.

made to fit it and which is wrapped round with a Thiersch graft, raw side out. Over this the skin is brought. The mold is left in for two weeks, then removed through a cutaneous incision. This method, which is called *epithelial outlay* is analogous to the epithelial inlay of Esser, used for restoring the cul-de-sac (§ 916). When there is very extensive loss of tissue, so that there is not enough skin left to cover the mold, Gillies' *epithelial overlay* may be applied. In this method the remains of the eyelid are sewed into the proper position, the scar tissue is removed, and the resultant large raw area is covered with a Thiersch graft which is held in place by a mold corresponding in size and shape to the defect (McKee).

935. Blepharoplasty in Defects Involving the Border and Entire Thickness of the Lids.—In many cases, especially after operations or extensive injuries, part or the whole of the lid including the lid border is entirely absent. Then neither skin grafts nor sliding skin flaps are applicable since they cannot replace the lacking epithelial lining and tarsus. For defects of this sort the following methods have been used:

1. A defect involving the entire thickness and the palpebral border of a *portion of the upper lid* can be filled in by a similarly shaped sliding flap taken from the entire thickness of the lower lid, but not including the palpebral border (Wicherkiewicz).

This flap which by its base retains its connection with the lower lid is slid up without being rotated until it fits into the gap in the upper lid where it is stitched in. The resultant through-and-through defect in the lower lid is brought together by sutures so as to form a vertical line. The base of the transplanted flap is cut loose from its attachment to the lower lid after eight days.

2. A similar defect in the *lower lid* may be covered in by a sliding flap fashioned out of the conjunctiva and tarsus of the upper lid (Köllner). The latter is inverted and the flap is fashioned and detached except at its palpebral border where it is left, attached to the upper lid. The flap is then swung forward, turning on its attached edge as on a hinge, until it comes to lie in the defect in the lower lid. Its conjunctival surface thus faces toward the eye and its raw surface faces outward. It is stitched in place and the raw surface covered with a skin graft or a sliding skin flap. The lids are kept stitched together for eight days, then the transplanted graft is cut loose from its attachment to the upper lid.

3. In more or less *complete absence of the lid* Büdinger makes use of the cartilage of the ear. First a pedicled skin flap is formed to replace the cutaneous structures of the lid. Then there is cut from the proximal portion of the auricle a flap the size and form of which correspond to the free posterior surface of this skin flap. The flap from the auricle consists of the skin on the posterior surface of the latter and of the portion of cartilage belonging to it. It is placed on the raw posterior surface of the new lid and is attached there by stitches. The cartilage of the ear replaces the tarsus, and its cutaneous lining replaces the conjunctiva. Since the skin on the anterior surface of the auricle has no defect in it, the auricle is not button-holed; the wound on the ear heals without being sewed up, leaving but a small scar which on account of its situation on the posterior surface of the ear is not conspicuous.

VII. Operations for Ptosis

936. Shortening the Lid.—An operation is indicated both in congenital ptosis and in old cases of acquired ptosis which cannot be cured in any other way. A whole series of methods of operating for ptosis has been proposed, a circumstance which proves that no one of them gives perfectly satisfactory results. This can be readily understood, since, in fact, the physiological action of a muscle cannot be perfectly replaced by any surgical operation whatever.

The oldest method consisted in shortening the lid by excising a *fold of skin*, a procedure which Von Graefe combined with excision of the subjacent bundles of fibres of the orbicularis (the antagonist of the levator palpebræ). This method has been abandoned, because a sufficiently great shortening of the lid always results in a considerable degree of lagophthalmus; and if only a little skin is excised the operation is unsuccessful. Good results, however, especially in congenital ptosis, may be obtained without undue lagophthalmus by *tarsectomy*. This is done in the same way as in Kuhnt's total tarsectomy for trachoma (§ 929) except that the conjunctiva being in this case intimately adherent to the tarsus is not dissected from the latter, but is removed with it. If the ptosis is incomplete, a slice of the tarsus may be left at the upper end (Wheeler).

In Gillet de Grandmont's operation the tarsus is exposed by an incision made tnrough the skin of the lids, parallel to the free border of the latter and three or four mm. from it. Then two incisions are carried through tarsus and conjunctiva, one parallel with the free border of the lid and two or three mm. from it, the other arching up from the two ends of the first incision, so as to include a semilunar segment of tarsus and conjunctiva some three mm. high in its middle. This segment is removed and the gap in tarsus and skin closed by sutures.

937. Advancement of the Levator.—The other methods of operating for ptosis are based on attempts to make good in some way for the deficient muscular power of the levator. If there is a levator which, though weakened, can still contract, we attempt to increase its effect upon the lid; if the levator is entirely absent, we endeavor to invoke the aid of other muscles, like the frontalis or the superior rectus, for lifting the lid. An increase in the action of the levator is effected by shortening the latter. This idea lies at the foundation of the operation for *advancement of the levator* proposed by Eversbusch.

A cutaneous incision is made about midway between the border of the lid and the eyebrow, and passing in a horizontal direction from one end of the lid to the other. Starting from this incision, the skin and the fibres of the orbicularis are loosened from their attachments, so that they can be pushed to one side, and the upper border of the tarsus together with the levator can be exposed to view. A loop of thread is then passed through the tendon as high up as possible, the two ends of the loop being carried down between the tarsus and the skin of the lids and brought out in the intermarginal space. If these ends are tied there and drawn taut, the tendon of the levator is drawn down by means of the loop thus passed through it, so that the tendon is folded and thus shortened. Three such loops should be applied—one in the middle and one on either side. This method gives good results, which, however, are not always permanent, as the tendon which has been drawn down by the loops of thread subsequently retracts.

Better and more permanent results are obtained if we shorten the tendon by resecting a portion of it. The skin of the lid is incised along the convex border of the tarsus, and from this point dissected up as far as the eyebrow. The fascia tarso-orbitalis and, after this has been divided, the tendon of the levator now lie exposed to view. Of the tendon a portion is exsected whose length must be regulated by the degree of ptosis. Then the anterior extremity of the shortened muscle is sewed again to the convex border of the tarsus.

938. Replacement of Levator by Frontalis.—If the levator is completely inactive, shortening of it would be without effect. We then endeavor to replace its action by that of the frontalis, starting from the fact that persons with ptosis strive to lift the lid by wrinkling the forehead. This is regularly done in bilateral ptosis, since otherwise the patient would not be able to see. But in unilateral ptosis where there is no such compulsion, wrinkling of the forehead commonly is absent and in that case, then the following methods are inapplicable. By the wrinkling of the forehead the eyebrows are lifted and thus indirectly the lid is lifted too. But the skin of the lid must be entirely smoothed out before the edge of the lid can be elevated at all, and hence the greatest part of the effect that the contraction of the frontalis is able to exert is lost. Hence, the idea arose of making the elevation of the lid greater by connecting it directly with the fibres of the frontalis. This may be effected by means of a *subcutaneous suture* (Dransart, Pagenstecher).

One needle of a double-armed thread is entered above the free border of the lid and is carried up beneath the skin of the lid to the arch of the eyebrows, above which it is brought out again. The second needle is passed alongside the first. There is thus a loop of thread lying within the lid, the middle of the loop being situated above the free border of the lid and its two ends emerging above the eyebrows. These ends are tied over a roll of adhesive plaster or iodoform gauze and drawn tight. Ordinarily three such

loops are applied alongside of one another and they are left in situ until firm cicatricial bands are formed along the suture tracks—bands which extend from the lid to the surface of the frontalis muscle and connect these two structures together (give the muscle what may be called a tendon for the upper lid).

Hess's method depends upon the same principle. In order that the scar which is left shall not be visible the incision is made in the eyebrow which has previously been shaved; it occupies the middle of the latter and traverses its entire length. Starting from this incision, the skin of the lid is detached with the knife downward as far as the free border of the lid. Then, as with the sutures described above, three loops of thread are passed from the lid up to the forehead. The entry is made in the outer skin about in the middle of the topmost part of the lid in order that when the threads are drawn up a fold of skin may be formed which will imitate the covering fold. The point of exit is about two cm. above the eyebrow. The loops of thread are drawn up until the lid is elevated a good deal, since the effect of the operation afterward diminishes somewhat. Since in the period immediately succeeding the operation, the lid cannot cover the eye, the latter during this time must be protected from desiccation by a dressing with a moist chamber (§ 442). Hess's method is much more successful than simple sutures because the skin of the lid which has been made raw on its under side is displaced superficially on its bed which is also raw; and by the adhesion of these raw surfaces it is kept permanently in its new place.

The operation of Panas tries to secure the connection between the lid and the frontalis muscle by the formation of a pedicle from the skin of the former, which pedicle is attached to the skin of the forehead and to the surface of the muscle.

Fig. 430 shows how the pedicle is cut out of the skin of the lid. The pedicle, *s*, after being defined by incision, is dissected from its bed until it is freely movable; then a horizontal incision, *a*, is made through the skin directly above the eyebrow. Starting both from this incision and from the wound already made below, the skin of the eyebrow is undermined so that a bridge of tissue is formed, beneath which the pedicle, *s*, is slipped so that its upper margin is in contact with the upper lip of the incision, *a*. Its attachment to the latter is effected by means of a loop of thread, the centre of which lies on the cutaneous side of the pedicle, while its extremities, *b b*, are passed through the upper lip of the wound. By drawing the loop tight the pedicle is lifted up and is attached to the upper border of the wound. If necessary, a second loop may be applied, and also some interrupted sutures, to secure exact adaptation of the edges.

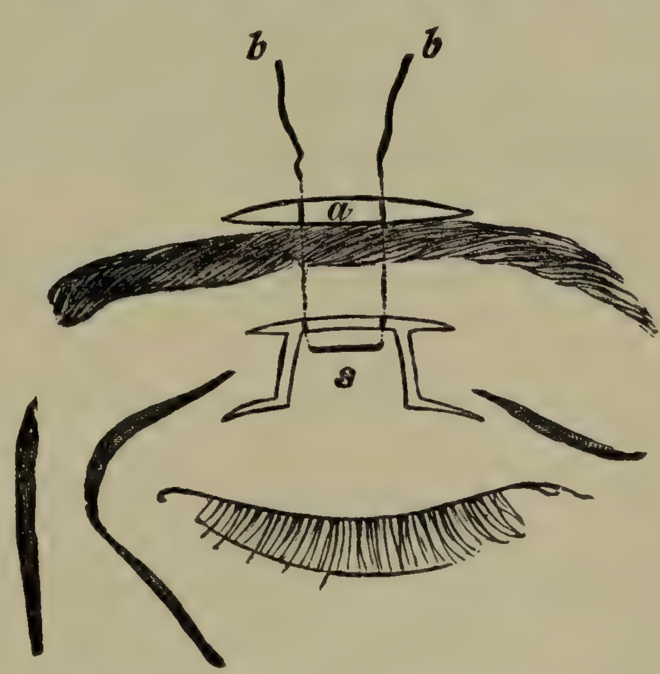

Fig. 430.—Panas's Operation for Ptosis.

In the *Tansley-Hunt* modification of this operation the incision is so made that the base of the pedicle is close to the free border of the lid. On either side of the lower half of the pedicle, an elongated triangle of skin is exsected, each triangle having its base directed toward the pedicle. In order to secure better union, the epithelial surface of the pedicle is scraped before the latter is drawn up into the hole under the eyebrow (Wheeler).

The operation produces a satisfactory effect but has the disadvantage of leaving scars which run perpendicular to the direction of the fibres of the orbicularis and hence are pretty conspicuous.

939. Replacement of the Levator by Superior Rectus.—The superior rectus is also available for replacing the levator, provided the former itself is not defective nor paralyzed, as it may be in congenital ptosis.

Following *Motais's method*, we first expose this muscle by making a transverse incision two cm. long through the conjunctiva over the insertion of the tendon, and dissect up the conjunctiva back to the fornix. We retract the upper flap by two sutures passed through its middle and five mm. apart. Between these sutures we divide the conjunctiva by a vertical incision, which is carried back to the fornix. We now evert the upper lid, grasp the upper border of the tarsus with fixation forceps, and carry the vertical incision in the conjunctiva on to the upper border of the tarsus. Next we retract the two sides of the split conjunctival flap by means of sutures passed through each close to the tarsus, and free the superior rectus from its attachments on either side. Then, starting from the end of the vertical incision in the conjunctiva, we make a transverse incision through the levator along the upper border of the tarsus, and working through this incision, detach the tissues from the anterior surface of the tarsus, so as to form a pocket extending down to the lower border of the lid. Lifting the superior rectus with a squint hook, we pass a double-armed thread through the tendon three mm. from its insertion, so as to form a loop on the scleral surface of the latter. On either side of this loop we split the muscle longitudinally by incisions, three to four mm. apart, running forward to the insertion and backward ten to twelve mm. The tongue thus fashioned we detach at its insertion, leaving the nasal and temporal portions of the tendon connected with the sclera, and then by means of the two threads projecting on the upper surface of the tongue, carry it forward into the pocket on the anterior surface of the tarsus. The threads are brought out just above the free border of the lid, and tied over a roll of gauze tight enough to produce a marked primary over-effect. The conjunctival wound, especially in the cul-de-sac, must be carefully closed (Bruns, Shine). Good operators regard this as one of the best procedures for ptosis.

CHAPTER XXXV

OPERATIONS ON THE CORNEA AND REGION OF ANTERIOR CHAMBER

I. Removal of Foreign Bodies

940. Foreign bodies penetrating into the cornea should be removed as soon as possible. If they are superficially situated, it is an easy matter to pry them up with a suitable instrument. For this purpose we use a special needle, which is made broad at its upper end (Fig. 431). In default of such a foreign-body needle we also may make use of a sharp sewing needle which has been previously sterilized by heating in a flame. When the foreign body is not deeply imbedded, it is better to use a blunt spud to pry it up with. It is advantageous first to render the cornea insensitive by the repeated instillation of cocaine or holocaine. In the case of fragments of iron, besides the foreign body the ring of brown-colored corneal tissue adjoining it should be scraped off with a tiny curette or gouge.

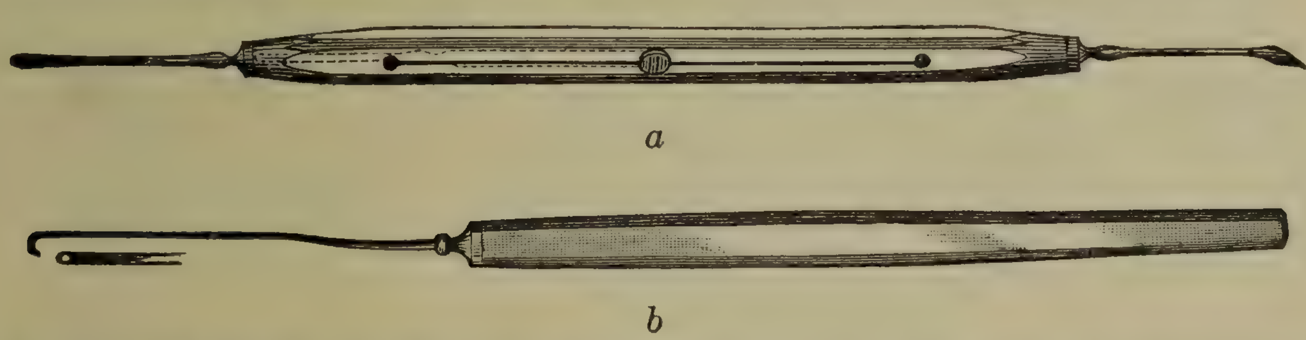

Fig. 431.— *a*, foreign-body needle and spud. *b*, Knapp's flexible and blunt foreign-body hook.

If the foreign body has penetrated into the deeper layers of the cornea, it is often necessary to incise the lamellæ of the cornea that lie above this foreign body so as to be able to draw it out with the forceps. If the point of the foreign body projects into the anterior chamber there is the danger that, in attempting to grasp the foreign body, the latter may be pushed in still farther, and may injure with its point the capsule of the lens. In such a case, therefore, the indication sometimes is to make an opening in the cornea near its margin and through this introduce an instrument into the anterior chamber. With this we press the foreign body from behind forward, so that we can grasp it by its anterior extremity and extract it.

II. Keratotomy and Keratectomy

941. Keratotomy.—Paracentesis, or puncture of the cornea (keratotomy), may be performed either with the keratome (Fig. 433, *a*) or with Von Graefe's linear knife (Fig. 444, *a*).

(*a*) In making paracentesis with the *keratome* the latter is plunged in close to the outer and lower margin of the cornea. Then the keratome is pushed a little farther forward, so that the wound gets to be two or three

mm. long, when the knife is withdrawn very slowly from the wound. Then, to make the aqueous flow off, we need only depress the peripheral edge of the wound (*c*, Fig. 395) gently with a Daviel's scoop (Fig. 444, *b*). The escape of the aqueous should be gradual, and preferably intermittent.

Paracentesis with the keratome is made—1. In progressive ulcers of the cornea whose advance either along the surface or into the depth of the tissues can not be arrested by medicinal treatment. In the case of corneal ulcers which threaten rupture we anticipate the occurrence of the latter by performing paracentesis. We thus avoid having the perforation take place too suddenly, and also avoid the occurrence of a prolapse of the iris. When the floor of the ulcer is greatly thinned and is bulging, we select this as the site of our puncture. 2. In ectasiæ of the cornea of various kinds, and also in markedly bulging prolapses of the iris, or in the staphylomata that develop from the latter. In these cases paracentesis must be followed by the use of a pressure bandage (see § 943). 3. In obstinate inflammations of the cornea or uvea, and also in opacities of the vitreous, in order to exert a favorable effect upon the nutrition of the eyeball by altering the conditions of tissue metamorphosis. 4. In suppuration of operative wounds. Here repeated paracentesis (H. Knapp), combined with irrigation and the introduction of antiseptic solutions (De Schweinitz) may sometimes help. 5. In elevation of tension when this is likely to be transient, as, for example, in irido-cyclitis or in swelling of the lens. 6. For removing hypopyon, when it extends high up. 7. For removing foreign bodies in the anterior chamber. In this case because the keratome passes through the cornea obliquely (§ 895) we must place our external incision not quite at the site of the foreign body but a little distance from it.

In all these cases except the last, it is not infrequently necessary to repeat the paracentesis one or more times. If only a few days have elapsed since the first paracentesis, a new incision is not required when the latter is repeated, but the wound, not being solidly united, may be pried open with a Daviel's spoon.

Paracentesis with the *Graefe linear knife* is done in ulcus serpens. The Graefe knife, whose cutting edge is directed straight forward, is entered to the outside of the temporal border of the ulcer, in the healthy portion of the cornea; then it is pushed in the anterior chamber toward the nasal side until its point is brought out again through the cornea to the inside of the nasal margin of the ulcer. We then may be said to have the ulcer lying upon the edge of the knife, which latter, therefore, has simply to be pushed farther to the front in order to split the ulcer from behind forward. The section should have both its terminal points lying in sound tissue, and, if possible, should be so made that the most intensely yellow, progressive portion of the ulcer is bisected by it. After the section has been completed the hypopyon is removed. The section must be reopened daily with a Weber's knife (Fig. 398, *f*) or with a Daviel's scoop, until the ulcer begins to grow clean.

This operation, devised by Heuerman in 1765 and revived by Guthrie in 1843, and later by Saemisch, is usually called the *Saemisch* (or by Englishmen the *Guthrie*) *incision*.

n making it we must take care not to injure the lens and not to let the excape of aqueous ake place too suddenly. The hypopyon is either evacuated spontaneously, especially f the patient makes pressure with his lids, or it can be grasped by means of forceps introluced into the wound, and drawn out. For, in ulcus serpens, it is not thin and liquid, ut of a tenacious, viscid consistence. In consequence of the diminution of pressure fter the escape of the contents of the anterior chamber, hæmorrhages often take place rom the iris, which, already hyperæmic before the operation, now becomes still more listended with blood. This distention of the iris is probably the cause of the violent pain vhich regularly follows upon the discharge of the contents of the anterior chamber, lthough the incision itself is but little felt. After incising the ulcus serpens we always et an attachment of the iris to the cornea during healing, which, however, would not ave failed to occur, even apart from the operation, in those cases in which incision is ndicated at all. The lens may be extruded at the time of operation and sometimes he latter is followed one or two days later by an expulsive hæmorrhage (Sichel).

942. Keratotomy and Keratectomy in Staphyloma.—(a) **Total Staphyloma.**—The simplest procedure in total staphyloma is *incision.* This is lone with the expectation that as a consequence of it the staphyloma will ollapse, and, because of the retraction of the cicatricial tissue of which it onsists, will remain permanently flat. Evidently this procedure is crowned with success only when the staphyloma is thin-walled enough to collapse after the incision has been made; it is, therefore, indicated only in those recent staphylomata which are still akin to prolapse of the iris. The incision which is done with a cataract knife, is made in various ways: either in a straight line and transversely across the middle of the staphyloma (Küchler) or in a curved direction and concentrically with the lower corneal margin, so that a flap is formed of the wall of the staphyloma. The latter way of making the section has the advantage of causing a marked gaping of the wound, since the flap contracts owing to the drawing up of the cicatricial tissue. Consequently, the lips of the wound are prevented from rapidly reuniting, in which case the ectasis would soon be reproduced and the incision would have to be repeated. If the wound in the flap should not gape sufficiently, the flap must be retrenched by the removal of a part of it. After the completion of the section, the lens, in case it is still in the eye, must be removed by lacerating the anterior capsule.

In old thick-walled staphylomata *ablation* (Beer) is indicated. To do this we first divide the conjunctiva all round the cornea and separate it from the sclera, so that it can be drawn forward to the proper extent. Then we pass stitches through the free edge of the conjunctiva in such a way that if drawn tight directly after the operation they will close the aperture made by the ablation. (If the stitches were not thus prepared beforehand ready to apply at once, we might readily have loss of vitreous). We may apply either interrupted or tobacco-bag sutures (for one good method see Fig. 432). When the stitches have been passed we turn the conjunctiva back and the stitches with it and scrape off the limbus and the epithelium at the margin of the staphyloma so that when the conjunctiva has been drawn up over these parts after the operation it will adhere to them. Then laying the stitches out of the way, we detach the lower half of the staphyloma from its base by a curved incision with the cataract knife, and grasp-

ing the flap thus formed with a forceps detach the upper half of the staphyloma with the scissors. The lens, which now presents, we remove by opening the capsule, and then at once close the wound with the stitches.

Those cases of staphyloma of the cornea in which a considerable ectasis of the sclera has developed as the result of an increase of tension are in general not adapted for ablation. We should then run the risk of getting a violent hæmorrhage in consequence of the sudden diminution of the previously increased tension. For such cases, in which the eyeball is increased sometimes to quite an enormous size, the only thing feasible is *enucleation,* which relieves the troublesome symptoms and at the same time also the disfigurement, inasmuch as an artificial eye can then be worn in place of the hideous, enlarged eyeball.

943. (b) **Partial Staphyloma.**—In this, treatment seeks a threefold object: to improve the sight, to cause flattening of the ectasis, and to prevent the development of an increase of tension, or to do away with it in case it has already set in.

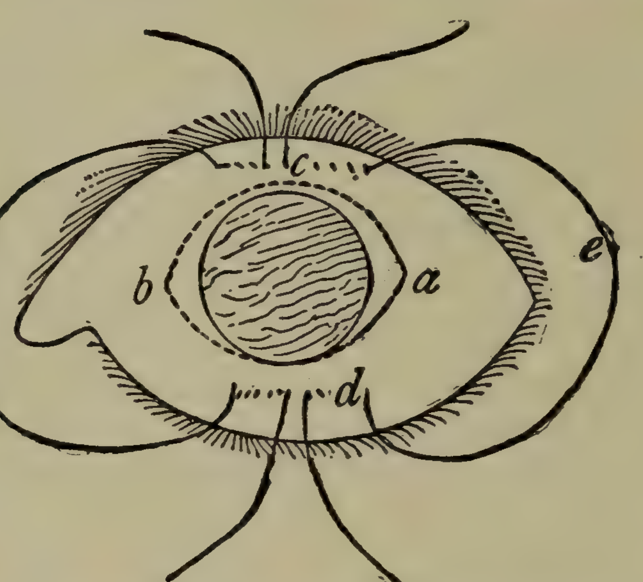

FIG. 432.—ABSCISSION OF STAPHYLOMA. KNAPP'S METHOD. (After Norris and Oliver.)

Before abscising the staphyloma a stitch is entered 4 mm. above the vertical meridian of the cornea (at *c*), passed temporally through the conjunctiva and episcleral tissue, brought out so as to form the loop *e*, re-entered below at *d*, and brought out again beneath the vertical meridian of the cornea. A similar stitch is passed on the nasal side. The staphyloma is then abscised by Beer's method and the two ends *c* and *d* of the first stitch drawn taut until the loop *e* is converted into a straight line. The same thing is done with the inner stitch. The gap made by the abscission is thus converted into a vertical slit. The sutures are then tied.

Simple incision, which must be followed up by the application of a pressure bandage for a pretty long time, accomplishes its end only in recent staphylomata, the walls of which are still soft. In older and thicker scars, *excision,* with or without the operation of uniting the edges of the wound by sutures or of covering them with a conjunctival flap (conjunctivo-keratoplasty), is to be preferred.

Sutures passed through the edges of a staphyloma readily tear out. To prevent this, perforated gold strips may be applied at the opposite lips of the wound and the sutures after being passed through the holes in the strips may be tied down on the latter (Tenner).

But the most approved remedy that we possess against ectatic scars is *iridectomy* or *trephining* followed by the application of a pressure bandage (see § 953). Iridectomy should be done before the process is far advanced, otherwise the result is apt to be unsatisfactory (A. Knapp). In thick-walled and unyielding staphylomata, it is advisable to combine excision and conjunctival transplantation with iridectomy. We begin by doing the former, and put off the iridectomy to some weeks later, when a flat cicatrix forms—doing this to prevent renewed bulging of the recent cicatrix. A very small staphyloma we may remove completely by excising with a corneal trephine and implanting a piece of normal cornea in the gap (*transplantation of cornea,* see § 945).

If we have to do with a partial staphyloma in which, owing to the increase in tension, the sight has been already either in great part or

altogether annihilated, no gain in this regard can be expected; yet in most cases it will be best to perform iridectomy or trephining in order to prevent the further consequences of the increased tension, such as partial ectasis of the sclera, enlargement of the entire eyeball, etc. Of course, iridectomy can be performed in such cases only so long as the anterior chamber is still existent. When by increased tension the iris has been squeezed against the posterior surface of the cornea and cemented to it, iridectomy has become technically impossible.

944. Operations for Keratoconus.—Operation is performed in keratoconus in order to substitute a resistant cicatrix for the attenuated apex of the cone. This is effected either by the *galvano-cautery* or by *excision*. In applying the former it is best to use a dull red heat and make one or more furrows from the cauterized spot to the limbus by a superficial application of the cautery point (Elschnig). In order to prevent renewed bulging of the scar, many combine cauterization or excision with a corneo-scleral trephining (§ 959) or an iridectomy so as to reduce the tension which, though not too high for a normal eye, is too high for the attenuated and purple yielding cornea of a keratoconus.

Fox, with a cataract knife, makes a horizontal incision through the cornea three mm. above its horizontal meridian, does an iridectomy, cuts out a semilunar strip from the lower lip of the corneal incision, and then brings the edges of the corneal wound together with sutures. Alonso makes a horizontal incision through the apex of the cone, trims off the upper and lower lips of the incision, and then brings them together with fine sutures previously passed through the cornea after the method used by Kalt in cataract extraction. The whole is covered for a week with two conjunctival flaps which are fashioned before the corneal incision is made and which are drawn together and sutured over the cornea.

The corneal cicatrix produced by cauterization or excision may be directly in front of the pupil, in which case an optical iridectomy will have to be performed later; and tattooing of the central scar may be done to diminish the dazzling.

The operation for keratoconus does effect a flattening of the cornea, but the curvature of the latter is never quite regular, and, moreover, there is usually produced a central scar of the cornea with its optical disadvantages. We must therefore be contented if a moderate improvement of sight is secured by the operation. The operative treatment does not always give a lasting result, the cornea in many cases bulging again subsequently, although not as much as it did before. For these reasons we usually decide upon operation only in the more advanced stages of the disease. All operations involve some risk, as glaucoma, irido-cyclitis, cataract, or possibly sympathetic inflammation may result from them.

945. Keratoplasty.—*Transplantation of the cornea* (keratoplasty) may be used to partly replace diseased by healthy cornea. Hippel's trephine, the small crown of which is set in rotation by clock work, serves for performing this operation. The operation was first devised with the view of making a transparent spot in the case of complete opacity of the cornea which left no portion open for iridectomy. A round piece is excised, with the trephine from the opaque cornea usually in its pupillary area. In the gap is placed a piece which is taken from a healthy cornea with the same trephine crown and which, therefore, exactly fits in the hole. It is most advantageous to take the piece designed for transplantation from a human cornea. Opportunity for getting this is afforded when an eye with a sound cornea has to be enucleated. The human cornea

has the advantage over that of animals in that it has the same thickness as the diseased cornea and hence fits into it, and also because it becomes readily united. Union in fact almost always takes place, but in very few of the cases as yet observed has the piece thus incorporated remained transparent. In other cases it gets opaque in the weeks immediately following, and so much so generally that the purpose of the operation—the restoration of sight—is not attained. On the other hand, transplantation satisfactorily accomplishes its object when the case is one of thin, protruding, or fistulous scars which it is desirable to remove entirely in order to prevent subsequent harmful consequences. The operation in this case is done in the way described above, except that after cutting out the diseased portion we must start from the opening and free the iris as far as possible from its attachments to the cornea, in order to effect a thorough removal of the anterior synechia. The operation is possible only when the scar that is to be excised has not too great an extent, and it requires care and skill to avoid injuring the capsule of the lens. The transplanted portion becomes opaque in these cases also, but the object of the operation, which is to replace the thinned scar by firm tissue and to remove the anterior synechia, is not on this account frustrated.

III. Tattooing of Cornea

946. This is done to diminish the disfigurement produced by white scars on the cornea. It depends upon the observation that many bodies, as, for example, grains of powder, may become imbedded in the cornea and remain there permanently. Tattooing consists in giving the white scar a black tint by means of india ink, which is introduced into the cicatricial tissue by being repeatedly pricked in with a needle.

The tattooing needles used for this purpose consist either of a bundle of ordinary sharp-pointed needles (Taylor), or of a single broad needle which is channeled for the reception of the ink (grooved needle of De Wecker). The ink is poured on the cornea and pricked in by repeated thrusts of the needle. Then the ink is washed off, the effect observed, and the process repeated until the effect is satisfactory.

Tattooing should be applied only in the older scars, which are solid and flat. For in thin or ectatic scars, the latter may be weakened by the inflammatory reaction, which always follows tattooing, and thus an increase of the ectasis or even an elevation of tension may be set up. Nor is tattooing a proper procedure for eyes that have passed through a severe attack of irido-cyclitis, since this disease might be lighted up again by the operation.

In dense white scars *Fröhlich's method* is advisable. By cutting a shallow furrow with Hippel's corneal trephine we demarcate a circular area of the size of the pupil in the scar. Within this area either the epithelium alone or this and the most superficial lamellæ of the cornea with it are removed, the parts thus exposed are coverd with minute scarifications, and then the India ink is rubbed into them. In the course of years the black color fades somewhat and requires to be renewed by a repetition of the operation.

In cases in which only a part of the pupillary area of the cornea is opaque, and the rest is transparent, tattooing the opaque portion may actually improve the sight, since the cicatrix, being made less transparent, does not produce as much diffusion of light as before.

When it is desired to simulate a blue or brown iris, tattooing may be done with a properly sterilized pigment of the appropriate color (Ziegler).

IV. Iridectomy

947. Technique.—Iridectomy by Beer's method is performed as follows: Entry is made with a keratome in the vicinity of the margin of the cornea, and sometimes a little to the outside, sometimes a little to the inside of the latter, according as the point up to which it is desired to excise the iris is more or less close to its ciliary margin. The blade of the keratome should

make the proper angle (about 45°) with the curve of the eye at the point of entry. If the blade is entered too perpendicularly it will be likely to engage in the iris and cannot be turned as readily after the cornea has been penetrated; if it is too slanting its point will emerge too far forward in the anterior chamber. The direction of the push that carries the keratome forward must be strictly parallel to the blade of the instrument. As soon as the point of the keratome enters the anterior chamber the handle must be depressed so that the blade lies parallel to the iris. The blade is pushed forward in this plane until the wound is of the desired length (four to eight mm. according to the breadth of the portion of the iris that it is proposed to cut out); in doing this the keratome must be so held that the section is concentric with the margin of the cornea (*aa*, Fig. 436). Withdrawal is performed slowly, and with the instrument pressed against the posterior surface of the cornea, so as not to injure the iris or lens, which push forward as the aqueous flows off. After completing the section, the iris forceps (Fig. 433, *c*, *d*, *e*) with its branches closed is introduced into the anterior chamber and pushed on up to the border of the pupil. At this point the branches are allowed to separate and a fold of the iris is grasped, gentle pressure being at the same time made upon the latter. The iris is now drawn from the wound, and at the moment when it is most upon the stretch it is cut off close to the wound with the curved scissors or with the scissors forceps of De Wecker (Fig. 433, *h*). (In an iridectomy done for glaucoma, instead of cutting off the iris, a number of the English operators snip it at the edge and then tear it from its root). After the sector of iris has been excised it now only remains by introducing a spatula (Fig. 433, *i*) into the wound to put back into the anterior chamber any iris that may have been wedged into the wound, so that at the completion of the operation the pupil and the coloboma have their proper shape.

If we can not push the lance far enough along, because the anterior chamber is shallow, we *enlarge* the section by cutting laterally with the lance as we withdraw it. The section in the tunics of the eye for an iridectomy may be made with the *lance* or with the *Graefe knife*. With the latter the tunics of the eye are divided from behind forward as is done in the cataract operation. The section made with the lance is smoother and passes much more obliquely through the tunics of the eye than does the section made with a narrow knife. It, therefore, closes better after the knife is withdrawn. Hence, the lance is to be preferred in every case in which no special obstacles oppose its application. This latter is the case: 1. When the anterior chamber is shallow. Here we can not push the lance far without running the risk of pricking the iris and lens. In optical iridectomies, in which a short section is ordinarily sufficient, this is a matter of less importance, but in glaucoma, in which the anterior chamber is often so shallow, we would be able to make the section with the lance neither peripheral enough nor long enough. 2. When the anterior chamber is absent the section with a lance is absolutely impossible, while we can always push a Graefe knife for a sufficiently great distance between the cornea and iris. 3. When the patients are unruly or when the operator's experience is small, the danger of wounding the lens with a Graefe knife is always less than with a lance and many operators employ Graefe's knife to the absolute exclusion of anything else for iridectomy. Unfortunately, the use of the Graefe knife meets with a limitation in that we can cut with it only at the upper and lower borders of the cornea (because elsewhere the orbital margin gets in the way). Sections at the nasal or temporal borders of the cornea can be made only with the lance.

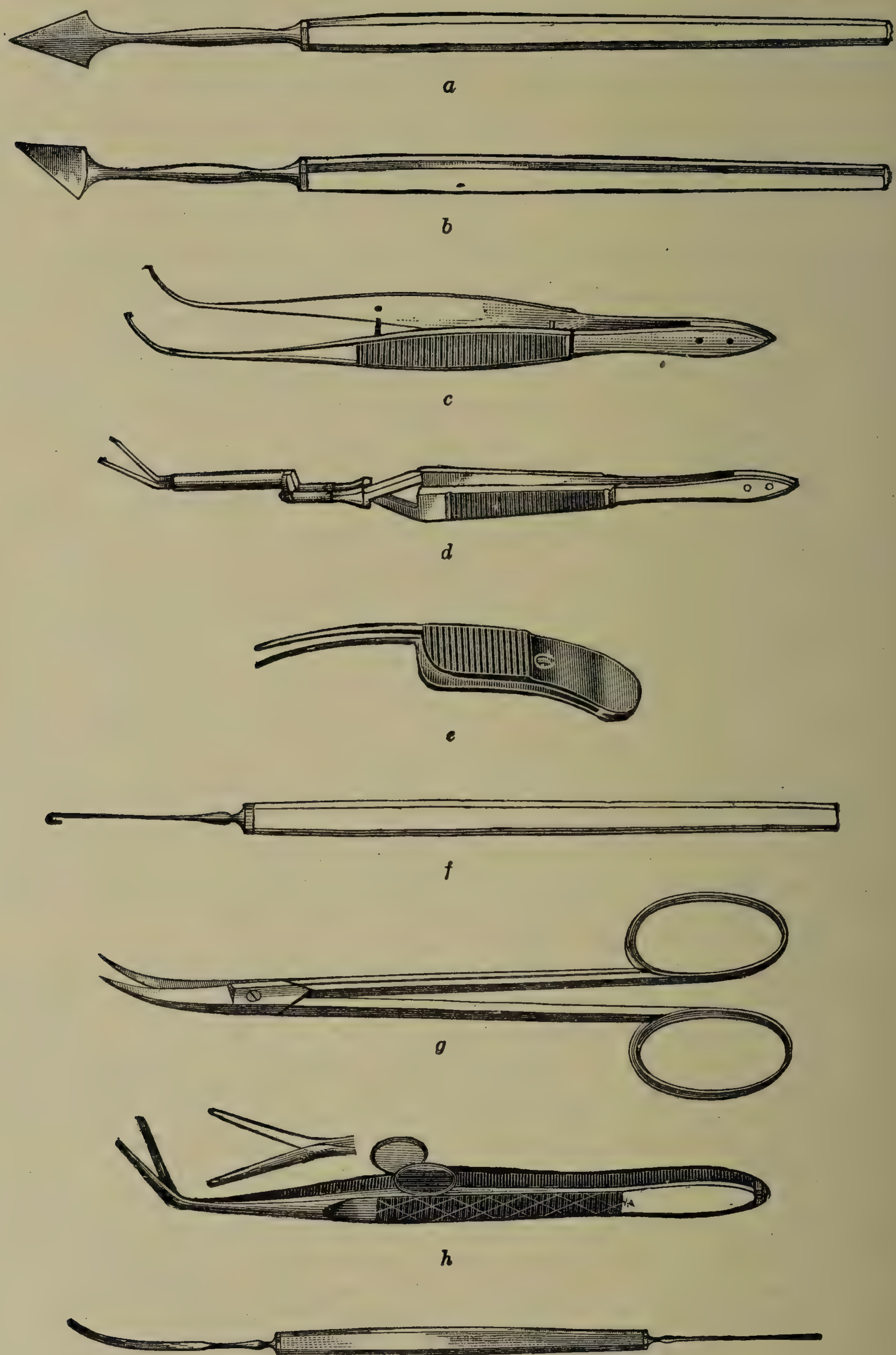

FIG. 433.—INSTRUMENTS FOR IRIDECTOMY AND IRIDOTOMY.

a, straight keratome (lance knife). *b*, angular keratome. Used in iridectomy (§ 947), paracentesis (§ 941), simple linear extraction of cataract (§ 966). *c*, *d*, *e*, iris forceps for passing into the wound made by the lance knife and grasping the iris. *f*, Tyrrel's blunt iris-hook used when the lens is absent or dislocated. *g*, curved scissors for excising the iris. *h*, De Wecker's scissors (pince ciseaux). *i*, combined spatula and blunt-pointed probe. Other instruments required are a spring speculum or Desmarre's retractor, to keep the lids apart, and a fixation forceps to hold the eyeball (see Fig 388).

For grasping the iris a minute *blunt hook* (Fig. 433, *f*) may be used instead of an iris forceps. With this the margin of the iris is caught up and drawn out. The hook is suitable for cases when (1) we wish to excise only a small bit of iris at the pupillary margin; (2) when the iris has no fixed support behind it in the lens (in dislocations or absence of the lens). For in the latter case when the arms of the forceps are pressed against the iris, in order to grasp it, the iris gives way, whereas it can be picked up with a hook. See also remarks on extra-ocular iridotomy, § 954.

948. Accidents.—The mishaps which may occur in the course of an iridectomy are: 1. *Injury of the iris or lens* with the lance or forceps either through the clumsiness of the operator or through the restlessness of the patient. Injury done to the lens capsule entails a traumatic cataract, which not only produces a new obstacle to vision but also endangers the eye by giving rise to inflammation or increase of tension. Such a traumatic cataract may be produced even in cases in which no instrument touches the lens. When the intra-ocular pressure is high or when there is strong pressure exerted by the patient, the edge of the lens is pushed against the incision, and in consequence the capsule of the lens may be ruptured at this portion of the lens margin. The result is a subsequent opacification of the lens. Because of the wound in the capsule the lens may make a spontaneous exit through the incision, either immediately after the operation is completed or some days later. 2. *Iridodialysis.* If the eye makes a violent movement at the moment when the operator grasps the iris with the forceps, the iris may by this means be separated from its insertion to a varying extent, or even in part be torn out of the eye. In iridectomy done on account of occlusion of the pupil, iridodialysis may also be produced in the following way: The operator grasps the iris and tries to draw it out of the wound. To effect this it ought first to be set free from the pupillary membrane. But if the connection between the latter and the iris is very firm, the two do not separate, but the membrane and also the iris of the opposite side follow the course of the traction, so that an iridodialysis is produced upon the side opposite the iridectomy. Hence, in such cases, the iris ought always to be first released from the pupillary membrane by lateral movements of the forceps before it is drawn out of the wound. By iridodialysis the excision of the iris is rendered difficult, great bleeding is set up, and often, too, a double pupil is produced (see § 723). 3. The last-named result may also occur from the fact that at the site of the iridectomy the *sphincter* is *left behind,* so that it separates the pupil from the coloboma like a bridge. This accident may occur because the iris is excised before it has been drawn far enough out of the wound. We shall not have to complain of this disagreeable occurrence if we observe the two following rules: The first is, not to grasp the iris with the forceps until we have pushed the instrument forward as far as the pupillary margin so as to get this latter between its branches. The second rule is, to cut off the iris only when it has been drawn out far enough for its black posterior surface to be visible (Fig. 393). If, nevertheless, the sphincter should remain behind, we enter the anterior chamber again with a blunt hook and draw up the bridge of sphincter in order to cut it off. There may, however, be another reason why the sphincter remains in situ, namely that it is so firmly adherent to the lens capsule that it tears away from the iris rather than follows the latter as it is withdrawn. In this case we refrain from any further attempt to remove the sphincter, as otherwise we might easily injure the capsule of the lens. 4. When we operate in a case of total posterior synechia it often happens that within the area of the coloboma the retinal pigment of the *iris remains upon the lens capsule,* with which it is intimately united by exudation. In that case, immediately after the iridectomy is completed, we may suppose that we have made a fine black coloboma, and it is only upon lateral illumination that we become convinced that the coloboma is not black, but dark brown—i. e., is filled with pigment. The optical result of the operation is then nil. It happens not less frequently in total posterior synechia that it is absolutely impossible to bring the iris out of the wound for the purpose of cutting it off. The iris, on the one hand, is so rotten, and, on the other hand, is so firmly attached to the lens, that the forceps, instead of drawing the iris out, only tears small fragments out of it. Both in this case and in the one in which the pigment layer remains behind, there is nothing left to do but to remove the lens also, by an extraction, even when the lens is still transparent. 5. *Prolapse of vitreous* is particularly apt to occur

in iridectomy when the zonula is diseased, as, for example, in subluxation of the lens or in hydrophthalmus, and also when we operate on very small children, in whom the zonula is as yet very frail. 6. *Hæmorrhage into the anterior chamber* or the *retina* often occurs after iridectomy especially in congestive glaucoma. The sudden diminution of the pressure, the fact that we are operating in a very hyperæmic eye, and the degeneration of the vessel walls are all accountable for this. The blood in the anterior chamber is sometimes unusually slow in being absorbed, because the normal channels of outflow are stopped up. The retinal hæmorrhages cause no special harm, except when one of them happens to involve the region of the yellow spot. 7. *Detachment of the chorioid* (§ 760). 8. Immediate *reduction of sight* and *field of vision* (in iridectomy done in non-congestive glaucoma—see § 844).

949. Indications for Iridectomy; Optical Iridectomy.—Iridectomy is indicated in a number of conditions. One of these is:

1. *The presence of optical obstructions.* These consist in opacities of the refractive media, occupying the area of the pupil. Among these belong: (*a*) Opacities of the cornea; (*b*) a membrane in the pupil (occlusio pupillæ); (*c*) opacities of the lens, such as lamellar cataract, nuclear cataract, or an anterior polar cataract of particularly large diameter. Furthermore in cases in which the pupil is considerably displaced toward the periphery as the result of an incarceration of the iris, iridectomy is done to bring the pupil behind the centre of the cornea; and it is also done in subluxation of the lens when the attempt is made to shift the pupil to a place in front of the part that contains no lens.

For any good to be gained from the performance of an iridectomy for optical purposes, the following *conditions* must be present:

(*a*) The opacity must be so *dense* that it prevents the formation of distinct images upon the retina, and does not simply interfere with vision by giving rise to dazzling. In the latter event the dazzling would actually be increased by the iridectomy. A mistake that we frequently meet with is that of making an iridectomy when there are comparatively slight opacities of the cornea; by such an operation the sight is made worse instead of better. To avoid this mistake we first make an accurate determination of the visual acuity, then dilate the pupil, and once more test the sight. If the latter then proves to be considerably better than before the pupil was dilated, iridectomy is indicated; otherwise not (see also § 951).

(*b*) The opacity must be *stationary.* In the case of opacities of the cornea the inflammatory process must have completely run its course; in opacities of the lens it must be stationary forms of cataract that are in question. Otherwise we run the risk of having the very spot become opaque which we have selected for making the artificial pupil in.

(*c*) *The parts concerned with the perception of light*—the retina and optic nerve—must be capable of performing their functions. This fact is determined by testing the vision and the field of vision, especially with the candle flame (see §§ 185 and 265).

The following conditions must be regarded as *contraindications* to iridectomy for optical purposes:

1. Deficiency or total absence of light perception. 2. Strabismus of long standing in the eye which is affected with the opacity. In this case, even if the operation was

technically a perfect success, not much gain in sight would be got, owing to the amblyopia ex anopsia which exists in such eyes. The result of the operation is doubtful and it is often technically impossible to perform in—3. Flattening of the cornea. For, where applanatio corneæ has developed, it is always a sign that there has been in conjunction with the keratitis an irido-cyclitis, which has left thick membranous exudates behind the iris. Hence, even if we actually succeed in excising the iris, a free aperture is not produced, and we are confronted by a hull of exudation. 4. Incarceration of the entire pupillary margin in a corneal scar with consecutive forcing of the iris against the posterior surface of the cornea. When this condition has lasted for quite a long time it is impossible to excise the iris (§ 948).

950. Results of Optical Iridectomy.—The *success* of an optical iridectomy as far as vision is concerned, very often falls short of the expectations which both physician and patient have entertained in regard to it.

This is especially the case with iridectomy in scars of the cornea. In this case there are various reasons for the vision's being often so defective, even when the operation

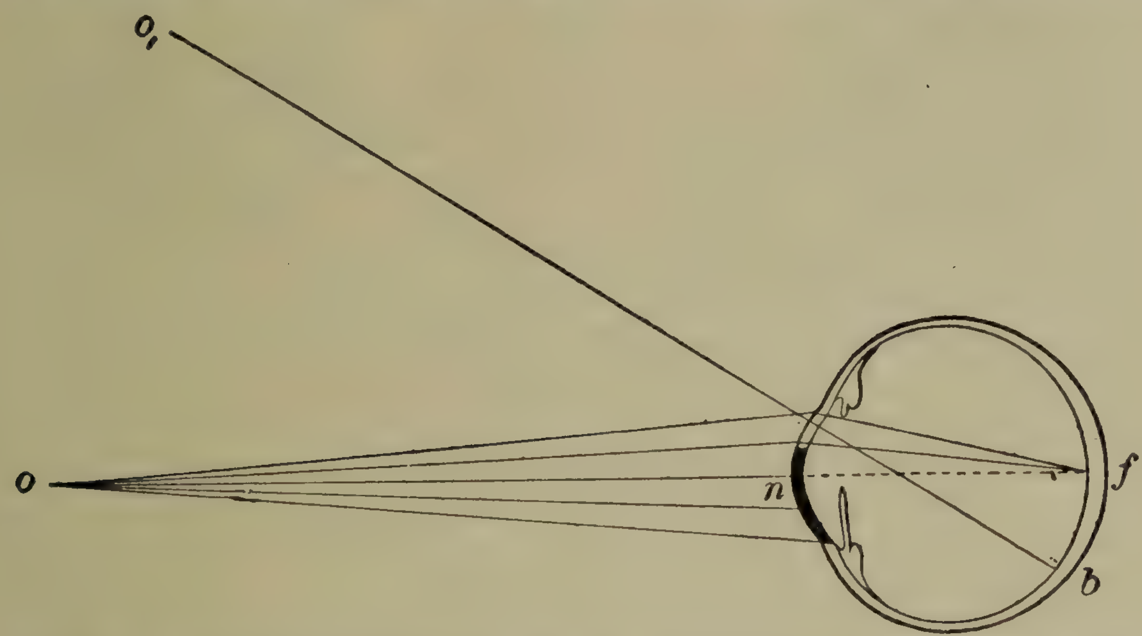

Fig. 434.—Path of Rays in the Case of an Eccentrically Situated Pupil.

itself has been a complete success. The chief one is that normally a considerable degree of astigmatism is present in the peripheral part of the cornea which has been used for the iridectomy. This astigmatism is increased partly by the effect of the adjoining scar, partly by the operation itself. To this is added the astigmatic refraction of those rays which in the peripheral portion of the coloboma pass through the margin of the lens. This astigmatism, which is for the most part irregular, has a greater effect than usual, inasmuch as the new pupil is large and is almost or quite immovable, and hence can not lessen the size of the diffusion circles (see § 168). Moreover, the cornea over the coloboma is often less transparent than was supposed before the iridectomy, for slight opacities are scarcely visible when a light-colored iris is behind them, while they at once become obvious when after iridectomy a black coloboma forms the background. Still greater is the disappointment in store for the operator when after a successful iridectomy he finds the coloboma white instead of black, because the lens has become opaque.

The optical result of an iridectomy made on account of a corneal scar may often be improved if we make the scar opaque by tattooing it (see § 946).

How must a man adjust his eye in order to *see with an eccentrically situated pupil*? Let us assume that the eye is affected with a central scar of the cornea (*n*, Fig. 434), so that it can see only with the assistance of a coloboma which has been made upward. Must this eye, in order to fix an object, *o*, be turned downward so that the coloboma may lie opposite the object? By no means. The refraction of the rays in such an eye takes place in precisely the same way as in a sound one. The only difference is, that the portions of the beam emanating from *o* which enter the eye are not those situated

in the centre, but those situated above and corresponding to the coloboma. These latter throw the image upon the fovea, *f*, provided the object lies in the line of vision. An object, o_1, which is situated opposite the coloboma would form its image at *b*, below the fovea, and would therefore not be seen by central vision. Hence an eye with an eccentrically placed pupil performs fixation in the same way as does a normal eye.

951. Incision and Coloboma in Optical Iridectomy.—A coloboma which is made for optical purposes must be so fashioned as to cause as little disturbance from dazzling as possible. This result is secured when the coloboma is narrow and does not reach to the margin of the cornea (*O*, Fig. 435). An excision extending up to the root of the iris would expose the margin of the lens and also the interspace between it and the ciliary processes, and thus admit a great quantity of irregularly refracted rays into the

Fig. 435.

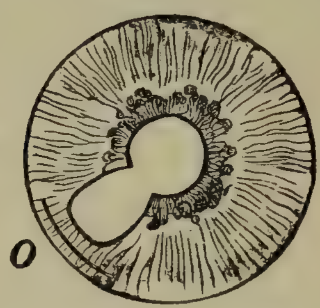

Fig. 436.

Fig. 435.—Optical Iridectomy. Magnified 2 × 1.—*o*, coloboma. The black line shows site of corneal incision.
Fig. 436.—Iridectomy in a Case of Increase of Tension. Magnified 2 × 1. *a a*, external orifice of the wound situated in the sclera; *i i*, internal orifice situated at the sclero-corneal junction.

eye. In order to make the coloboma narrow and not too peripheral, the incision must be short, and lie at, or even inside of, the limbus (see Fig. 435). Those cases constitute an exception to this rule, in which only the most exterior marginal portion of the cornea has remained transparent, so that the iridectomy must, for obvious reasons, be quite peripheral.

That spot is chosen as the *site* of the coloboma at which the media are the most transparent. Wherever possible, we avoid making the coloboma above, as in that case it would be partly covered by the lid. If the media are everywhere equally transparent (as when there is a cicatrix situated exactly in the centre of the cornea, a pupillary membrane, or a perinuclear cataract), the iridectomy is performed downward and inward (Fig. 435), because in most eyes the visual axis cuts the cornea a little to the inner side of the apex (§ 163).

Where to place the iridectomy can be determined by the *pear-shaped stop*. This is a disc resembling in appearance Fig. 435, in that it has in its centre a circular aperture four mm. in diameter, with an off-set notch four mm. long and 2.5 mm. wide. The pupil is dilated with homatropine, and the disc is set as close to the eye as possible and with its central hole concentric with the pupil. The disc is then turned until the patient sees best, when the position of the off-set notch will indicate the proper site for the iridectomy.

952. Iridectomy for Glaucoma.—2. Iridectomy is indicated in primary glaucoma, and also in secondary glaucoma resulting from ectasiæ of the cornea or sclera, from seclusio pupillæ, from irido-chorioiditis, etc. In hæmorrhagic glaucoma iridectomy is often a failure. In general, the success of the operation is better the earlier it is performed. Nevertheless,

an operation is sometimes done in cases of increased tension even when the perception of light has been already abolished, in which case there can be no idea of restoring sight. Then it is simply a case of relieving pain or of avoiding further degeneration (and especially further ectasis) of the eyeball.

When iridectomy is made for increase of tension, the section must be situated pretty far back in the sclera (1.5 mm. from the limbus—see Fig. 436) and must be long. The coloboma need not be very broad, but ought to reach to the ciliary margin of the iris (Fig. 436). If there are not at the same time any optical conditions, to which regard must be paid in making the iridectomy, the latter must be directed upward so that the coloboma may be partially covered by the upper lid, and thus the confusion due to dazzling may be lessened.

As a preliminary to iridectomy in glaucoma it is important to reduce the tension as much as possible by miotics, cathartics, and sedatives. In this way it may be possible to avert too sudden a reduction of tension with consequent post-operative hæmorrhage. In cases where there is considerable danger of the latter, it is advisable to precede the iridectomy with a posterior sclerotomy (see § 976 and cf. § 845).

Iridectomy is often *difficult to perform* in congestive glaucoma, on account of the cloudiness of the cornea, the shallowness of the anterior chamber, the rottenness of the iris, and the great painfulness of the parts. Hence, the excision of the iris does not always turn out to have been done correctly. Luckily it is precisely in congestive glaucoma that even a poorly performed iridectomy is usually followed by the effect desired.

On account of the great tension of the glaucomatous eye, the *edges of the wound* after iridectomy do not close up as well as in the case of other iridectomies—e. g., those made for optical purposes. Hence, more frequently than is otherwise the case, we get, instead of direct union of the edges of the wound, healing with the interposition of an interstitial tissue, as a consequence of which ectasis of the cicatrix or cystoid cicatrization is readily produced.

In *hydrophthalmus*, iridectomy is associated with greater danger than in the glaucoma of adults, and that mainly because of the defective condition of the zonula. By reason of this, when, after the escape of the extremely abundant aqueous, the lens is driven forward, rupture of the zonula, and hence escape of vitreous may take place. Another source of danger consists in the fact that we are dealing with children, of whom quiet behavior is not to be expected. Hence trephining is preferred (§ 959).

The *effectiveness of iridectomy in glaucoma* is regarded as consisting in the fact that the scleral scar, acting as a substitute for the impermeable ligamentum pectinatum, enables the aqueous to filter out (filtration scar, § 843). Increasing condensation of the iridectomy scar is, therefore, thought to be the cause of the subsequent recurrences of increase of tension, and hence, cystoid scars, in which the œdema of the conjunctiva over the scar leaves no doubt as to the oozing of the liquid through it, have been regarded as the most advantageous.

953. Other Indications for Iridectomy.—Other indications for iridectomy are:

3. *Ectatic cicatrices of the cornea* (partial staphylomata), in order to cause their flattening. This is the more likely to succeed, the more recent and the thinner-walled the staphyloma is—that is, the closer akin it is to a prolapse of the iris. In thick-walled and unyielding staphylomata it is best to combine iridectomy with excision and conjunctival transplantation (see § 943). The iridectomy should be performed in such a way that the incision

lies in the sclera and a broad coloboma is produced, reaching to the margin of the iris. We try to find for our iridectomy that spot which represents the greatest improvement that can be made in the vision, the pupil being shifted to a point behind the most transparent part of the cornea. Furthermore, by means of the iridectomy, an increase of tension is prevented from developing, or, if it has already developed, it is done away with. Moreover, in cases of staphylomata with thin walls, a flattening of the ectasis is obtained by the iridectomy, if the eye is kept beneath a pressure bandage for a long time after the operation.

4. *Recurrent iritis*, in which case the iridectomy is designed to prevent the recurrences—an object, however, which is not always attained. The operation is to be done during an interval in which there is no inflammation.

5. *Fistula of the cornea.* Iridectomy here serves the purpose of securing the formation of a firm cicatrix. We must wait to perform the operation until at least some trace of the anterior chamber has been restored, as otherwise the operation is impracticable on technical grounds.

6. *Foreign bodies* imbedded in the iris, which sometimes can be removed only by excising the portions of the iris in which they occur. The like is true of cysts and small tumors of the iris.

7. *As a preliminary to the operation for cataract*, iridectomy is done mainly when we are dealing with complicated cataracts (e. g., those complicated with posterior synechiæ, increase of tension, etc.). In this case the iridectomy must be made upward, so that the coloboma may also be utilized for the extraction of the cataract, which as a general thing is made upward.

V. Iridotomy

954. Iridotomy consists in simply dividing the iris without excising a piece of it, and in this respect differs from iridectomy. It serves the purpose of making a new aperture in the iris when the pupil is closed and of thus producing a new pupil. As the incision in the iris would also affect the lens which lies behind it and would thus produce traumatic cataract, this operation is adapted only to those cases in which no lens is present. In most cases the operation has to do with eyes which have been operated upon for cataract but which have lost their sight again through a subsequent irido-cyclitis. In these cases the iris is united with the exudation membrane and with the secondary cataract to form a firm diaphragm, which separates the cavity of the chambers from the cavity of the vitreous. To restore sight the diaphragm must be perforated. This can be accomplished by a simple incision, if this is so directed as to divide the diaphragm along a line perpendicular to that of greatest tension; then the incision gapes from retraction of the edges of the wound and leaves a slit-like pupil (cat's-eye pupil).

The operation may be performed with—

(*a*) The *Graefe knife.*—This is plunged through cornea and diaphragm, and the latter is divided in a direction perpendicular to that of greatest tension. This method is applicable only when the diaphragm is not too thick. Were this the case, the diaphragm would offer great resistance to

the knife, and in the endeavor to divide it the ciliary body would be pulled upon, and this might start a new attack of irido-cyclitis.

(b) The *scissors forceps* (pinces ciseaux) by De Wecker's method. With a keratome an incision is made along the corneal margin, and through this the scissors forceps is introduced closed into the chamber. Here the instrument is opened, and its posterior sharp blade is plunged through the diaphragm while the anterior blade remains in the anterior chamber; then, by closing the scissors forceps, the diaphragm is divided perpendicularly to the direction of greatest tension. This is a severer operation than the former, and is also usually associated with loss of vitreous. On the other hand, it does not cause any dragging, as in it the diaphragm is divided just as a sheet of paper is cut in two by scissors.

(c) *Ziegler's knife-needle* (Fig. 442 *f*).—This is entered at the upper sclero-corneal junction, with the blade turned on the flat. The point is carried down toward the bottom of the anterior chamber and then swung 3 mm. to the left. Next the edge of the knife is turned downward, the point is plunged into the iris, and the blade gradually drawn back so as to make an oblique cut in the iris from below upward. The blade is then swung over to the other side of the chamber and a similar cut made from below upward meeting the first cut at its apex. A triangular tongue of iris is thus marked out, which falls down, leaving a triangular pupil.

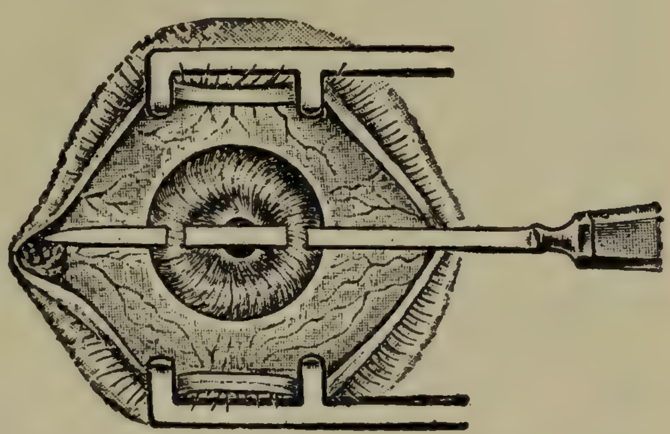

Fig. 437.—Transfixion of the Iris in Seclusio Pupillæ.
After Czermak-Elschnig.—The figure also shows the bulging of the iris (iris bombée) due to the seclusion.

Iridotomy sometimes fails owing to the too great firmness of the diaphragm, which may actually be ossified; but even an excellent immediate result may be nullified because the old irido-cyclitis is lighted up again by the operation and the pupil made by it is closed again by renewed exudation. Hence, we put off the performance of iridotomy as long as possible until all inflammatory symptoms have disappeared, unless we are compelled to operate speedily by special circumstances, such as protrusion of the iris, increase of tension, or beginning atrophy of the eyeball.

In order to be able to perform an iridotomy without danger to the lens, in case this is present, we may do an *extra-ocular* (or *precorneal*) *iridotomy*. We make a puncture in the limbus with the keratome, as for an iridectomy, draw out the iris, preferably with a blunt hook, incise it in a radial direction (from the pupillary to the ciliary border), and then return it to the anterior chamber. In this way a V-shaped gap is made in the iris, and accordingly we use this method of operating in place of an optical iridectomy, when we wish to get a very narrow coloboma.

Another case in which it is possible to perform iridotomy when the lens is present, without injuring the latter, is found in hump-like protrusion of the iris such as results from seclusio pupillæ; for here a considerable interspace—namely, the enlarged posterior chamber—separates the iris from the lens. Iridotomy in this case may be done by *transfixion* of the iris (Fuchs; see Fig. 437). A Graefe knife is entered about one mm. to

the inner side of the temporal margin of the cornea, passed through the anterior chamber, and made to emerge at a spot near the inner margin of the cornea and symmetrically situated with respect to the point of entry. The points of entry and exit lie in the horizontal meridian of the cornea, and the knife is held so that its blade is parallel to the base of the cornea. Since the iris is driven forward, the knife as it is being carried through the anterior chamber pierces the most protruding portion of the iris both temporally and nasally and makes holes in it. These holes remain permanently open and restore the communication between the anterior and posterior chambers; the iris returns to its former position, and the intra-ocular pressure becomes normal. In cases of hump-shaped protrusion of the iris, this operation may consequently be made instead of an iridectomy; and in cases of this sort it may be done as a preliminary operation to an iridectomy, so that the latter can be done later under more favorable conditions.

VI. Anterior Sclerotomy and Sclerectomy

955. Operations for Producing a Filtering Cicatrix.—In recent years a whole series of operations have been devised for reducing the intra-ocular tension by making a permanent filtering cicatrix in the region of the anterior chamber. This is effected either by simple division of the sclera at the angle of the chamber (anterior sclerotomy), by division of the sclera with inclusion of the iris (iridotasis, iridencleisis), or by excision of a portion of the sclera (sclerectomy). Sclerectomy again, may be accomplished either by excising a portion of the thickness of the sclera so as to make it permeable (Lagrange operation), or by making a permanent hole in the sclera (sclerostomy).

956. Anterior Sclerotomy.—This operation devised by DeWecker (Fig. 438) is one of the first of the operations suggested for effecting a filtering cicatrix.

A Graefe knife is entered one mm. outside of the temporal margin of the cornea, and brought out at an equal distance to the inside of the nasal margin. The points of entrance and emergence are therefore symmetrically situated, and are selected as though the intention was to form a flap two mm. high out of the upper part of the cornea. And, in fact, after the counter-puncture has been made, the incision is carried upward by sawing cuts just as if this flap was to be separated, but the knife is withdrawn before the section is completed. Thus, at the upper margin of the cornea there remains a bridge of sclera, which connects the flap with the parts below and prevents the gaping of the wound. Hence, by this operation two sections at once are made in the scleral margin separated by a narrow bridge (*s*, *s*). Sclerotomy may be made downward as well as upward.

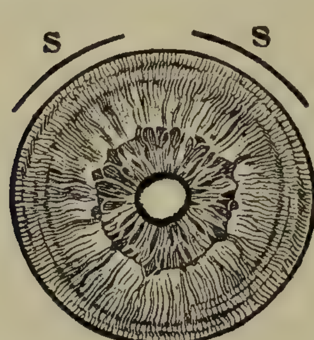

Fig. 438.—Sclerotomy by De Wecker's Method.

In Herbert's small-flap sclerotomy an opening is made into the anterior chamber through a ⊓-shaped incision in the sclera close to the cornea.

Sclerotomy, on account of the peripheral position of the wound, is very likely to cause prolapse of the iris. We should therefore try to produce a marked miosis by eserine before the operation; the spasmodically contracted sphincter then keeps the iris in the anterior chamber. If in spite of this the iris becomes wedged in the wound, and cannot be satisfactorily replaced it must be drawn out and cut off. The danger of prolapse is not present if there is already a coloboma of the iris, and the sclerotomy incision is placed near the site of the coloboma—the sclerotomy being made because of the recurrence of a glaucoma for which an iridectomy has already been performed.

957. Iridotasis.—This old operation, performed by Critchett in 1857, has been revived by Borthen and used with apparent success in glaucoma.

Atropine is instilled half an hour before the operation. A transverse cut, 6--8 mm. long is made through the conjunctiva at a point ten mm. above the cornea. The conjunctiva is undermined down to the corneal margin and the flap reflected. A keratome is entered in the sclera just above the cornea making a wound not over four to five mm. broad. The iris forceps is introduced and the iris is grasped by its free edge and pulled into the wound with its under surface up. The conjunctival flap is then replaced so as to cover in the scleral wound containing the iris (Harrower).

The operation of **Iridencleisis** (Holth) is performed in the same way except that the conjunctiva is not undermined clear to the corneal margin but to within three mm. of it and the iris is pulled out of the wound, split with a DeWecker's scissors and then allowed to retract into the wound. The idea is that the fistula formed by the incision, being lined with iris tissue will less readily close. At the same time it is covered with a particularly thick flap of conjunctiva. The danger in these operations would seem to be the likelihood of late infection transmitted through the imbedded iris tissue. Such infection has in fact occurred (Roy).

One application of iridotasis is to *relieve an iridodalysis* (Bulson). A small incision is made in the limbus and the detached portion of iris is brought up into the wound where it is held either by a suture or by the compression of the lips of the wound.

Seton-Drainage.—This operation, similar in principle to iridotasis, maintains drainage by means of a silk thread passing from the anterior chamber into the subconjunctival space. The operation, first proposed by Rollet, and adopted by Mayou and Zorab, is thus performed by the last-named surgeon, who calls it *aqueoplasty*: The conjunctival flap is formed and the keratome entered in much the same way as in iridotasis. The iris, however, is not withdrawn, but a loop of No. 1 white silk thread is introduced through the wound, a little way into the anterior chamber, with the free ends of the loop projecting out into the subconjunctival space. The flap is replaced so as to cover these ends and the wound and is sewed in position by stitches. Zorab has done the operation in a large number of cases of glaucoma in all stages with good results.

958. Anterior Sclerectomy.—The chief varieties of anterior sclerectomy are Lagrange's operation and Elliot's sclerostomy or trephining.

Lagrange Operation.—In this the incision 5 mm. long (Weeks) is made with a Graefe knife in such a way as to divide the sclera in the irido-corneal angle (Fig. 439). Puncture and counter-puncture are placed 1 mm. from the limbus. The operator, as he cuts, holds the knife flat, so as to pass very obliquely through the sclera, and come out beneath the conjunctiva several millimetres back of the limbus. He then divides the conjunctiva so as to form a flap. After the conjunctival flap has been laid back upon the cornea, the tongue which the knife has fashioned from the sclera becomes visible. This tongue is grasped with the forceps and divided with the scissors, so that the sclera is deeply beveled. After this sclerectomy has been done, iridectomy is performed in the usual way, and the conjunctival flap is brought up and laid over the defect in the sclera. Forty-eight hours after the operation, massage should be performed daily to promote the outflow through the cicatrix, and kept up till a satisfactory filtering scar is produced.

Occasionally the same bad results may follow as in trephining (§ 959), but blocking of the cicatrix and late infection are not apparently as frequent (Weeks).

959. Elliot's Sclerostomy or Trephining.—This operation, suggested by Argyll Robertson in 1876, is performed by Elliot in the following way. The conjunctiva is divided with the scissors 6 to 7 mm above the cornea by a cut made concentric with the latter and of such a

length that its ends on either side are in a line 1-2 mm. below the top of the cornea (Fig. 440, *aaa*). This demarcates a conjunctival flap which, in its central area only (*effe*), is undermined as far as the limbus. As the cornea is approached, the dissection of the subconjunctival tissue is carried down to the sclera (in the area below *ff*) and when the limbus is reached the dissection is carried into the cornea by splitting up the superficial layers of the latter for a distance of 1 mm. with the blunt point of the scissors or some like instrument. The superficial layer of the cornea thus peeled off is reflected with the conjunctival flap (*abba*) thus exposing a semilunar dark-looking area of split cornea (*c*). At the exact mid point of the arc bounding this area the trephine is applied (in such a way as just to be bisected by the sclero-corneal margin). Several sizes of trephine are used but Elliot prefers the 2 mm. size. The trephine is tilted slightly forward so as to cut through on the corneal side before it does on the scleral and is revolved until by the cessation of resistance the operator can appreciate that it has entered the anterior chamber. The button of sclero-corneal tissue, still attached by a scleral hinge, is grasped with a forceps and cut off. The iris which prolapses into the opening is snipped off, so as to produce a narrow peripheral, often button-hole iridectomy. The flap is then put back in place without being sutured.

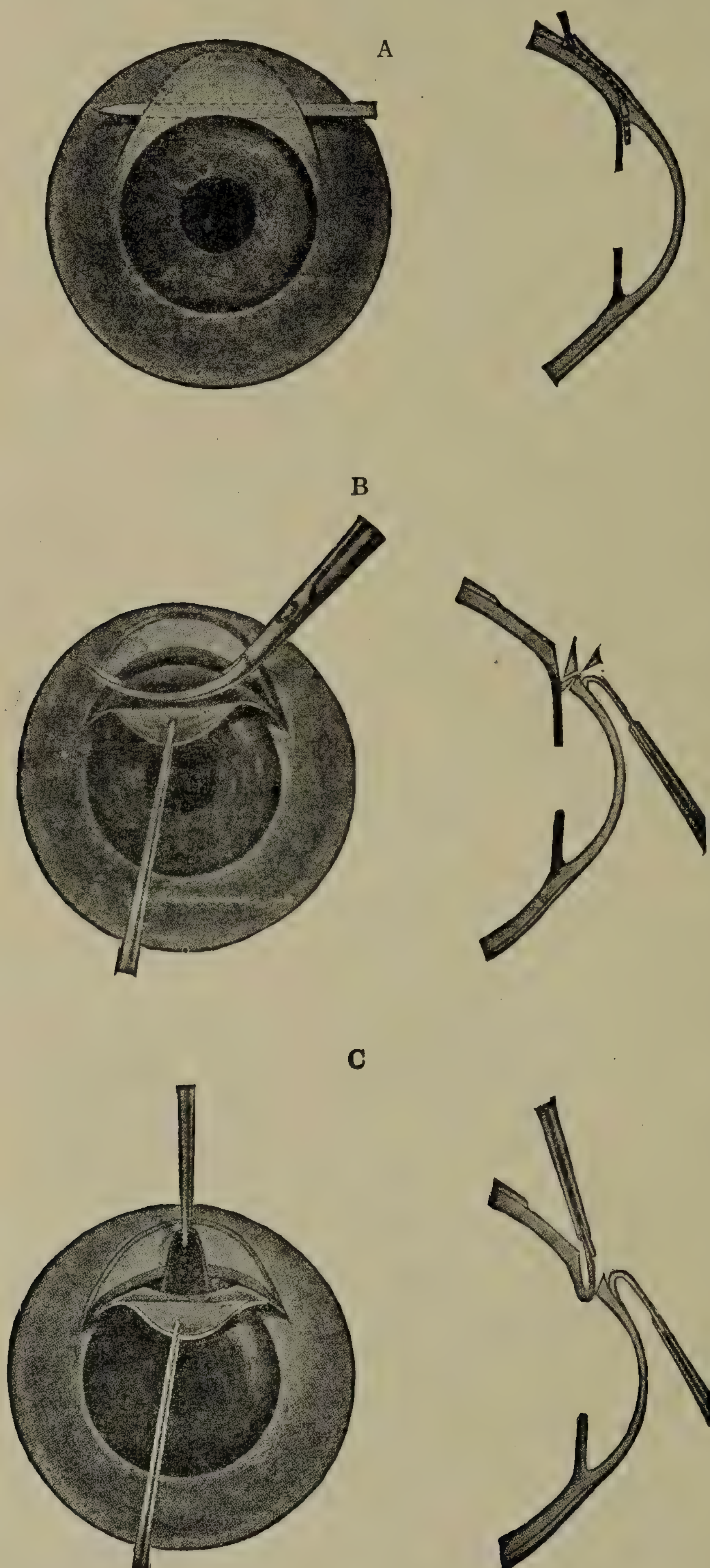

Fig. 439.—Lagrange's Operation—after De Schweinitz.
A.—Division of Sclera and Conjunctiva.
B.—Resection of the Sclera.
C.—Making the Iridectomy.

Untoward results which may sometimes follow trephining are (a) *Loss of vitreous.* (b) *Detachment of the chorioid* (§ 760). (c) *Intra-ocular hæmorrhage.* (d) *Closure of the fistulous orifice by scar tissue.* This is less likely to happen if the dissection is made according to Elliot's rules, leaving a free filtering space of undissected subconjunctival tissue (*ade*, Fig. 440) on either side. If it occurs the scar may be dissected out under the strictest antiseptic precautions (Dodd) or, better still, the adhesions may be broken up with a spatula, like that used in cyclodialysis, introduced through a conjunctival incision placed at some distance from the trephine opening (Wilder). (e) *Deterioration of vision* and *contraction of field* in spite of reduction in tension. In some cases this may be due to focal infection or to metabolic (endocrine) disturbance. (f) *Permanent hypotony.* This not very frequent sequela is sometimes followed by irido-cyclitis and cataract. For this reason A. Knapp prefers to use a smaller trephine than two mm., although

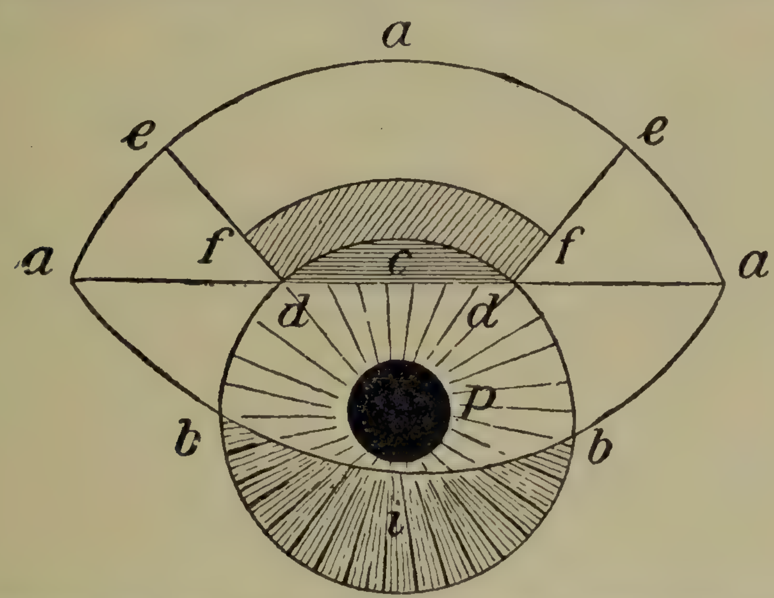

FIG. 440.—TREPHINING FOR GLAUCOMA; DIAGRAMMATIC REPRESENTATION OF THE AREA LAID BARE BY THE CONJUNCTIVO-CORNEAL FLAP (after Elliot).

aaa, line of conjunctival incision; *abba*, flap thrown down on the cornea; *c*, dark arc-like area of split cornea; *dd*, straight line of reflection of the flap; *edde*, area dissected up in order to enable the cornea to be exposed; *effe*, area of the upper part of the wound from which the conjunctiva alone is dissected up, when commencing to make the flap; *fddf*, area immediately above the limbus cleared right down to the sclera, in the course of the dissection of the flap; *ade*, the area on each side, in which the subconjunctival tissue is spared as much as possible in order to preserve the check-ligament-like action, which helps the flap to lie in good position after the operation is finished; *i*, iris; *p*, pupil. The trephine is applied exactly at the limbus, so that the hole made is bisected by the latter. This hole would be represented by a circle about half the size of the pupil, straddling the limbus at *c*.

as Elliot points out the small opening made with this gives trouble when the iris fails to prolapse as we cannot readily enter it with our instruments. (g) *Late infection.* The result of this is sometimes panophthalmitis, sometimes irido-cyclitis with destruction of sight, and sometimes a mild iritis with inflammation about the aperture, going on to recovery (Butler). Statements with regard to the frequency of secondary infection vary greatly. On the whole, it does not seem to occur often if the operation is properly performed.

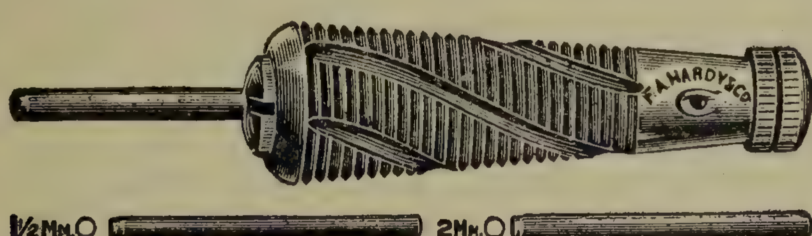

FIG. 441.—ELLIOT'S TREPHINE.

Trephining finds its most extensive *application* in glaucoma, especially in the chronic congestive and the non-congestive forms, and particularly in cases in which the visual field is contracted nearly to the fixation point, since in these iridectomy sometimes causes further contraction with consequent loss of central vision (§ 844), while with trephining this is less likely to occur. It is also the operation of election in buphthalmus. It has also been used with success either alone or combined with other measures (cauterization and excision of cornea) in keratoconus and staphyloma of the cornea.

Holth's Punch Operation.—In this a conjunctival flap is cut and reflected as in the Elliot operation, and the cornea is split as in the latter but only so far as to enable the opening in the sclera to be made up to the corneal edge. A special bent keratome or broad needle is introduced at a point 1.5 mm. above the cornea and carried into the anterior chamber. Into this cut a delicate punch is introduced which cuts out a D-shaped segment of the sclera. Over the hole thus left the conjunctival flap is replaced and sutured. By some (Butler, Koller) this operation is preferred to trephining, because with it no pressure is exerted on the glaucomatous eye.

Fergus' operation consists of a scleral (not sclero-corneal) trephining combined with a cyclodialysis.

960. Operations for Effecting Intra-Ocular Drainage.—Artificial drainage of the anterior chamber, undertaken to relieve glaucoma, may be effected not only exteriorly, as in sclerectomy and sclerotomy, but also directly into Schlemm's canal or into the perichorioidal space. Drainage into the latter is produced by—

Cyclodialysis.—In this operation, devised by Heine, the operator first makes a curved incision in the conjunctiva 8 mm. long and situated 8 to 10 mm. down and out from the limbus, undermines the conjunctiva freely toward the limbus, and by a transverse incision 6 mm. long and situated 6 to 8 mm. from the limbus divides the sclera, proceeding carefully from without inward down to but without injuring the chorioid. He then introduces a spatula into the wound and carries it forward with a rocking motion between chorioid and sclera, closely hugging the inner surface of the latter. When the tip of the spatula appears in the anterior chamber, the spatula is swept from side to side so as to detach the ciliary body for a third of its circumference and put the angle of the chamber widely in communication with the perichorioidal space. Then the spatula is withdrawn, and the flap put back and secured in position with stitches.

This operation often fails to produce a permanent effect, hence is usually reserved for cases in which iridectomy or trephining fails or is inapplicable (cf. §§ 843, 845).

Another operation is *puncture of the angle of the anterior chamber* from the anterior chamber itself (by De Vincentiis' method). By this means the path for the escape of the aqueous into Schlemm's canal is opened again. The operation is made with a special, small, sharp-pointed knife, plunged in at the corneal margin and carried through the anterior chamber so as to scarify the opposite portion of the sinus of the chamber. This operation is technically feasible only when the anterior chamber is sufficiently deep. Hence, it does not come in for consideration in most cases of inflammatory glaucoma. In appropriate cases, with deep enough anterior chamber, it has produced good results where an iridectomy has failed (Fuchs).

CHAPTER XXXVI

OPERATIONS ON LENS AND VITREOUS CHAMBER

I. Discissio Cataractæ

(a) Discission of Soft Cataracts

961. Technique.—Discission[1] of soft cataracts is done in order to effect absorption of the lens by opening its anterior capsule. Discission is performed with a sickle-shaped needle or with the knife-needle (see Fig. 442), which is passed in through the cornea (keratonyxis)[2]. The site of the puncture is the centre of the lower and outer quadrant of the cornea, at which spot the needle is passed through the cornea and perpendicular to the latter, and is then pushed forward in the anterior chamber as far as the anterior capsule of the lens. (In order to minimize the risk of post-operative infection, Byers and others prefer to enter the conjunctiva beyond the sclero-corneal margin and then through the periphery of the anterior chamber.) The lens-capsule is then laid open by one or more incisions in the area of the pupil (which has previously been dilated with atropine). The needle must be handled very lightly, no pressure being made with it, but simply sweeping movements; moreover, the incisions should not penetrate deep into the lens. The needle is then withdrawn from the eyeball, this being done quickly so that the aqueous may not escape.

After the operation the aqueous enters the lens through the wound in the capsule, and the lens swells up and is gradually absorbed in the manner described at length under the head of traumatic cataract (§ 657). In fact, discission is nothing but an imitation of the kind of injury of the capsule that accident so frequently produces.

Discission is adapted for all soft cataracts—i. e., for those which are capable of complete resorption because they have as yet no hard nucleus. This is the case in children and in adolescents. Discission may also be made in those cataracts that still contain transparent portions of lens substance, since these become opaque under the influence of the aqueous. The form of cataract most frequently requiring this treatment is zonular cataract. Lastly, discission may also be employed for removing perfectly transparent lenses, when it is a question of doing away with a high degree of myopia by operation.

In the cases cited, discission may be the only operation done; i. e., after doing it we simply wait for a gradual resorption of the lens to take place. This usually requires some months, and a repetition of the discission is often necessary. But, to shorten the duration of treatment, we may also employ discission simply as a preliminary operation to make the lens fit for extrac-

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1 From *discindere*, to split (i. e., the lens capsule). 2 From κέρας horn, and νύττειν, to prick

tion. For this purpose we make the discission quite free, so as to get a rapid opacification and swelling of the lens. As soon as this has advanced to a certain point—which is the case some days or weeks after the discission—the softened and disintegrated lens is removed by a linear extraction.

The main advantage of discission consists in the freedom from danger of the operation itself and in the simplicity of the after-treatment. Since the small puncture in the cornea closes again directly, the patient is not compelled to keep to his bed after the operation and the bandage may be dispensed with after one day. If the course is favorable, no further treatment is required than to keep the pupil dilated with atropine until the

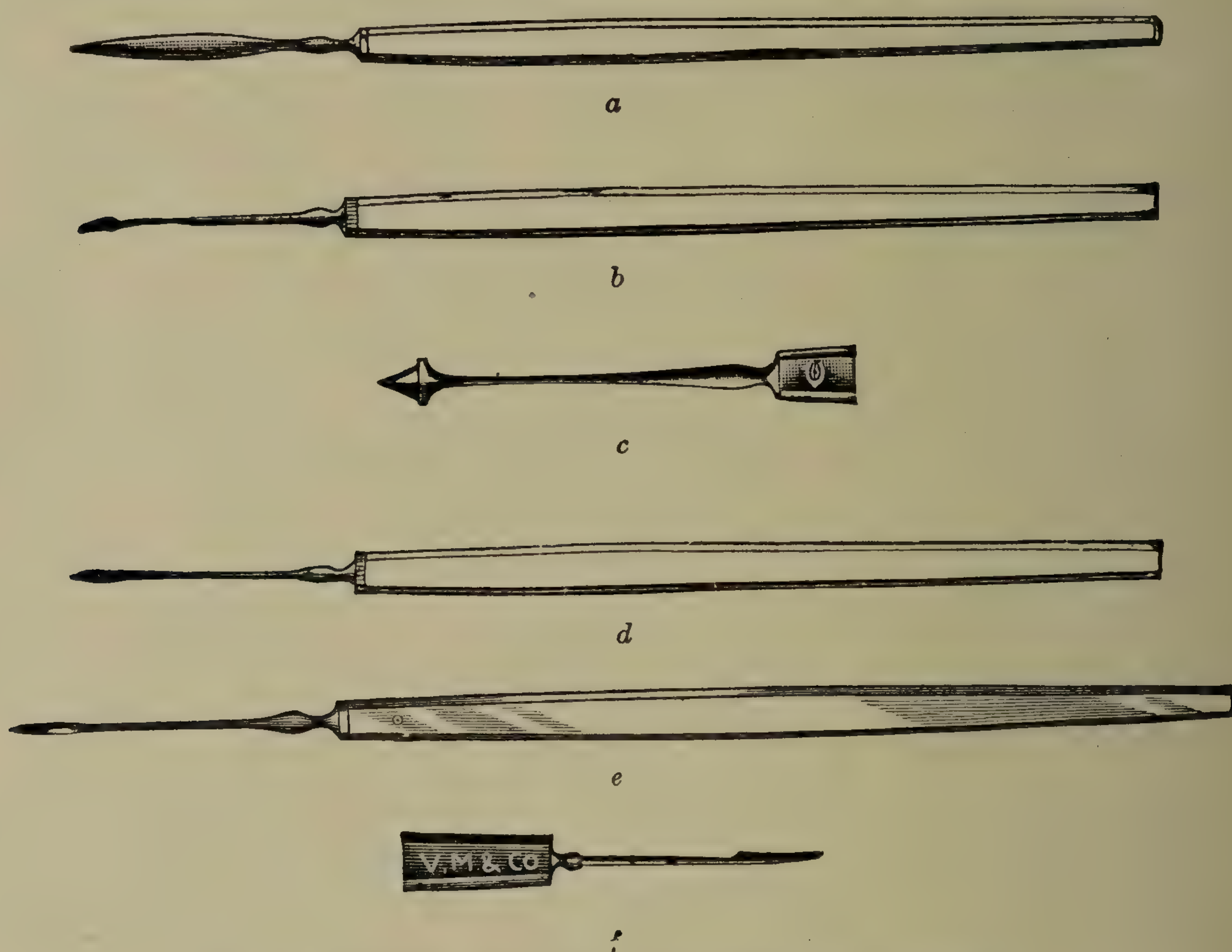

FIG. 442.—INSTRUMENTS FOR PARCENTESIS AND DISCISSION.

a, broad needle for paracentesis. *b*, Bowman's stop needle. *c*, Desmarre's broad paracentesis needle. *d*, Beer's cataract needle. *e*, Knapp's knife-needle. *f*, Ziegler's knife-needle. Other instruments required are a speculum and fixing forceps (see Fig. 388) and for some membranous cataracts De Wecker's scissors (Fig. 433, *h*).

resorption of the lens is complete. Discission, therefore, is the only cataract operation which can be employed with very small children who do not keep quiet after the operation.

962. Accidents.—During the after-treatment various accidents may occur, necessitating interference on the part of the physician. These are sometimes produced by the fact that the process of swelling takes place with too great violence; sometimes, on the contrary, by the fact that the swelling and absorption of the lens are brought to a standstill.

The *violent swelling* of the lens may be caused by too extensive a splitting of the capsule, owing to which the lens is exposed to the action of the aqueous over an excessively large area. In other cases, again, there exists in the lens a peculiar tendency to swell, which makes itself apparent even

with small incisions of the capsule. The results of a rapid swelling of the lens may be either increase of tension or irido-cyclitis. The former manifests itself by the dull appearance of the cornea, by an increase of tension perceptible to palpation, and often by the occurrence of pain; and these symptoms indicate a condition which if allowed to persist, would lead to blindness due to excavation of the optic nerve. The iritis is caused either by the mechanical injury (pressure) or the irritation which the swelling masses of lens substance produce in the iris[3]. Both increase of tension and iritis are particularly to be apprehended in elderly persons, because these stand swelling of the lens worse than others do. To avoid these accidents the pupil must be kept well dilated with atropine, so that the swelling masses of lens substance may come into contact with the iris as little as possible. Excessive swelling is most effectively combated by iced compresses, which, moreover, have an antiphlogistic action. The best remedy against the consequences of excessive swelling of the lens is the removal of the swelling masses by extraction. If we had intended at the start to have an extraction succeed the discission, a considerable swelling of the lens is rather what we would desire. But if we wish to get along with a discission alone, we must make the discission itself quite limited, so as not to be compelled by excessive swelling of the lens to perform an extraction afterward.

In contradistinction to the cases just mentioned, there are others in which from the outset the processes of swelling and resorption of the lens *take place to an insufficient degree.* In this event we are often dealing with the kind of lenses which are most likely to be found in aged persons, and which can swell but little. In other cases everything goes well at first, but after a part of the lens has been absorbed the swelling and resorption come to a standstill. The cause of this commonly lies in a union of the capsular wound, which takes place to such an extent that the aqueous no longer comes into contact with the lens fibres. In either case the indication is to repeat the discission, in doing which one may proceed more boldly than in the first operation and make an extensive opening in the capsule.

963. Contraindications.—Discission is contraindicated—1. In elderly persons whose lenses already have a nucleus, and whose eyes, moreover, do not bear well the swelling of the lens. 2. In subluxation of the lens, a condition which is recognized by the tremulousness of the latter. In this case discission is impracticable on technical grounds, since the lens being insufficiently fixed in its place would recede before the discission needle. 3. When there is considerable thickening of the capsule of the lens, as in this case the discission needle would cause luxation of the lens before it could tear through the capsule. 4. In the presence of posterior synechiæ, which render the dilatation of the pupil by atropine impossible. In such a case an iridectomy would have to precede the discission.

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[3] According to Verhoeff the iritis may be an anaphylactic phenomenon occuring in eyes sensitized to lens protein (§ 714). Hence, before contemplating an operation in which the lens capsule is to be opened, it is well to try to determine by the skin reaction to lens protein whether the patient is thus sensitized.

(*b*) *Discission of Membranous Cataracts* (*Dilaceration*)

964. The discission of membranous cataracts is not made with the view of effecting their resorption, since shrunken cataracts no longer contain much or any matter capable of being absorbed. On the contrary, the object is to make in the cataractous membrane a free opening through which the patient can see. It is frequently done as a secondary operation after the extraction of cataract to remove a secondary cataract. The operation may be performed either through the cornea or through the sclera.

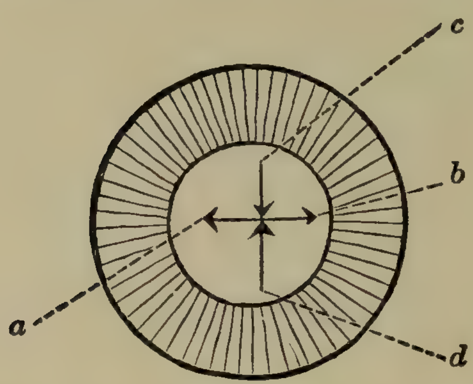

FIG. 443.—INCISION WITH THE KNIFE-NEEDLE IN SOFT PRIMARY AND IN SECONDARY CATARACT. (After Knapp in Norris and Oliver.)

The knife-needle is entered 3 mm. from the margin of the cornea in the horizontal meridian. The needle is advanced to about 2 mm. beyond the anterior pole of the lens, thrust through the lens capsule and (if the lens is present) through the superficial layers of the lens, and drawn back so as to make a horizontal incision, *ab*, 4 to 5 mm. long. Then the point of the needle is raised toward the cornea and pushed upward in front of the capsule to the point *c* from 2 to 2.5 mm. above the horizontal incision. Here the capsule is transfixed and is divided by a downward sweep as far as the horizontal incision. The same manœuvre is done on the lower half of the capsule from *d*, below upward, so that the three cuts make a crucial opening with arms 4 or 5 mm. long. When the operation is done as a preliminary to extraction, the incision may be longer and deeper, and in membranous cataracts will be varied in size and situation to suit the exigencies of the case. One of the best forms of incision is the V-shaped one of Ziegler (see § 954).

In the operation *through the cornea* (keratonyxis), the membrane is divided by a Graefe knife, a knife-needle, or a De Wecker's scissors in the same way that the iris is divided in iridotomy (§ 954). The corneal incision is preferably made in or near the limbus.

The operation was formerly and is still sometimes done with a needle (Fig. 442) which tears instead of cutting the membrane. As this produces undue traction Bowman used two needles introduced from opposite sides. These met near the centre of the membrane and were then drawn apart by sweeping movements. In the operations done at present the membrane is cut, not torn.

In cases with dense membrane, discission may be difficult, especially if there are adhesions binding the iris to the membrane. In these cases Colonel Smith after preliminary atropinization, does an iridectomy, or, if an iridectomy has already been done, excises one arm of the coloboma, so as to get into the posterior chamber. Into this he inserts a spatula, detaching the adhesions all round and working toward the pupil. The membrane thus detached is drawn out with an iris forceps and excised. Quite similar is the procedure of Beard who carries a very narrow keratome down to the lower edge of the pupil and incises the membrane there. The incision is enlarged by lateral movements of the keratome, adhesions are detached with a spatula, and then the upper part of the membrane is drawn out with a Tyrrel's hook and cut off. Wilder in these cases sometimes uses two knife-needles crossed like a pair of scissors.

In the *operation through the sclera* (scleronyxis[4]) a needle or better a Graefe knife or the knife-needle is plunged in perpendicularly through the sclera, six mm. behind the external margin of the cornea, and somewhat below the horizontal meridian, and is then pushed forward so that its point passes through the cataractous membrane into the anterior chamber close to the external margin of the pupil. Then the attempt is made to tear the cataract to the greatest possible extent by means of sweeping movements in which the point of the needle travels from before backward. The difference between discission through the cornea and that through the sclera lies in the fact that we are able, by the latter method, to bring a much greater force to bear upon the cataract with the needle and even depress it into the vitreous.

[4] From *sclera* and *νύττειν*, to prick.

Scleronyxis is adapted only to those cases in which there are but few, if any, portions of the lens left which are able to swell up. In soft, non-shrunken cataracts, which it is designed to subject to the processes of swelling and resorption, one should not make discission through the sclera, for in that case, in order to split the anterior capsule, we would have to pass the needle through the entire lens and break the latter all to pieces—a procedure which, apart from the fact that we might easily luxate the whole lens in doing it, would give rise to excessive and violent swelling.

II. Extraction of Cataract

965. The object of cataract extraction is to remove the lens from the eye immediately, and as completely as possible. In its fullest form it consists

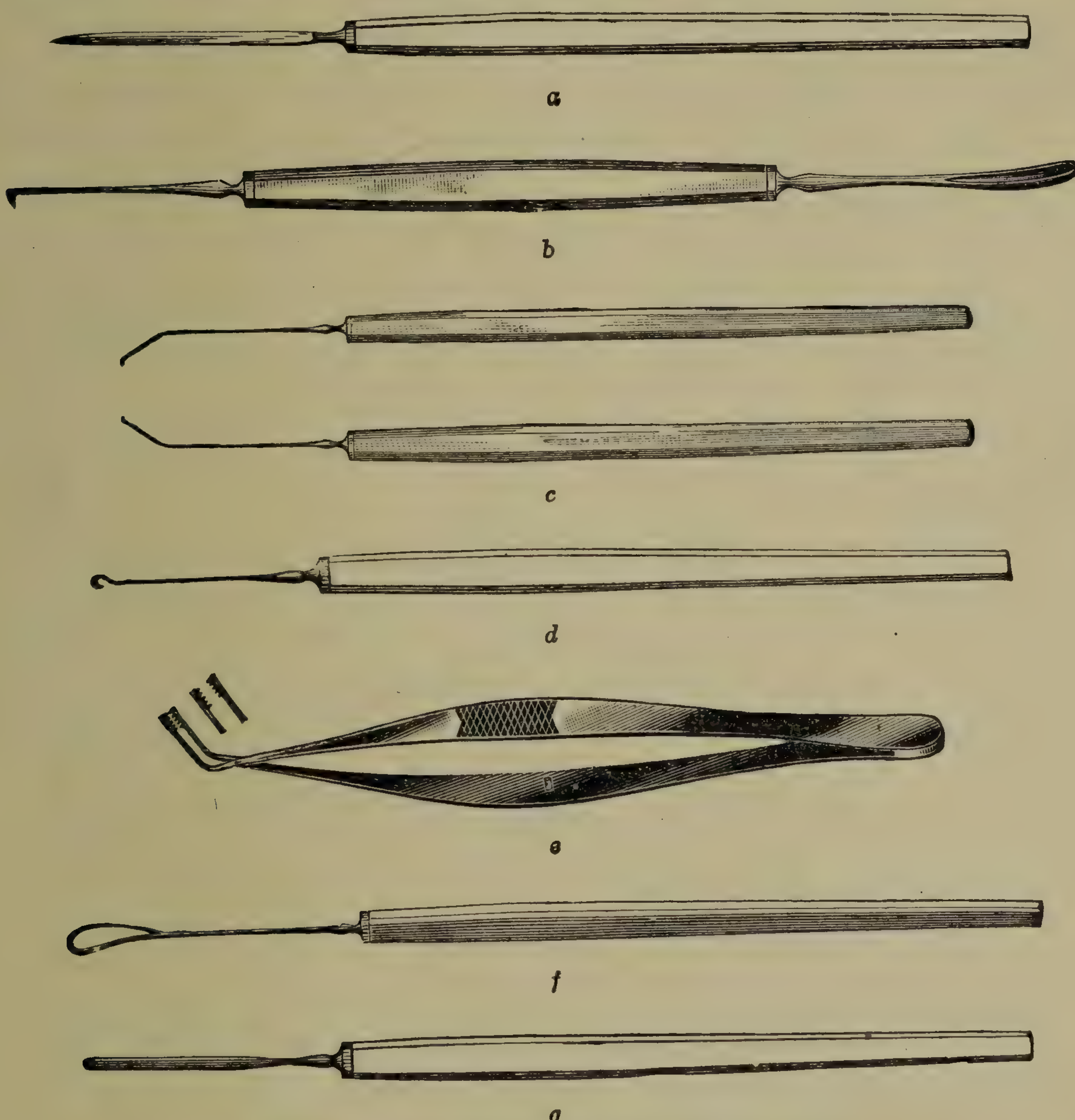

Fig. 444.—Instruments for Cataract Extraction.

a, Graefe's cataract knife (linear knife). Used also in paracentesis, iridectomy, iridotomy, sclerotomy and discission (§§ 941-944, 947, 954, 956, 958, 964). *b*, cystitome and Daviel's spoon. *c*, Knapp's cystitome (right and left), used in cataract extraction without iridectomy. *d*, Tyrrel's sharp hook for tearing the capsule. The blunt hook (Fig. 433, *f*) is also occasionally used. *e*, capsule forceps. The Kalt capsule forceps used in extraction in the capsule (§ 969) has cup-shaped and flattened extremities without teeth. *f*, wire loop. *g*, spatula to reduce the iris and stroke it out smooth. Other instruments are a speculum and fixing forceps (see Fig. 388) and if the cataract is done with iridectomy an iridectomy forceps and scissors (see Fig. 433).

essentially of four steps: 1. The making of a section whose dimensions vary in accordance with the size and consistence of the cataract. This section

may lie in the cornea or in the sclera. 2. Iridectomy. 3. Opening of the anterior capsule in order to allow the lens to escape from it. 4. Expulsion (delivery) of the lens. This is usually effected by pressure on the eye. Hence the operation should be called *cataract expression*, the term extraction being reserved for the comparatively few cases in which the lens is lifted or pulled out of the eye by instruments (see §§ 966, 971). In linear extraction and many cases of flap extraction the iridectomy is omitted; and in one method of flap extraction the anterior capsule is not opened since the lens is extracted in the capsule. The most usual methods of extraction are:

(*a*) *Linear Extraction*

966. Linear extraction is so called because the section is at least approximately linear (see § 895). Linear extraction, like discission, is performed both in soft and in membranous cataracts, and consequently is done in two different ways, which are modifications of each other:

1. In operating upon a *soft cataract* a very broad keratome is introduced at the limbus in the lower margin of the cornea, during which manœuvre the blade must be held parallel to the corneal margin. (H. Knapp, followed by others, made the incision in clear cornea 2 mm. from the periphery of the latter). The keratome is then pushed forward until the wound in the lower margin of the cornea has a length of 4 to 7 mm. (*S S*¹, Fig. 445.) Then the lens capsule must be very thoroughly torn up in the area of the pupil, which has been previously dilated by means of atropine. For opening the capsule we may use either a discission needle, a sharp hook (Fig. 444, *d*) or a capsule forceps (Fig. 444, *e*). After this the lens masses are discharged by making pressure on the eyeball with the finger through the upper lid and at the same time depressing the peripheral lip of the wound with a Daviel's spoon. By this means the contents of the eyeball are subjected to quite a great pressure, and the wound is made to gape open. This manœuvre is repeated until all parts of the lens have been removed from the eye. The lens masses may also be removed by irrigation (Wilder).

Fig. 445.—Linear Extraction. Magnified 2 × 1. Pupil dilated with atropine.

If linear extraction is done to remove a lens that has been swollen up by a preliminary discission, the opening of the capsule is omitted, since the capsule has already been torn open by the discission.

2. When a *membranous cataract* is to be operated upon, the section is made in the same way. Then a sharp or blunt hook or a forceps is introduced through the section, and with these instruments the cataractous membrane is grasped and drawn out through the wound. (Cf. the methods of partial extraction described in § 964.)

The advantages of linear extraction consist in the fact that the section is short and passes through the cornea obliquely, for which reason it closes readily, does not necessitate an iridectomy, and does not require any very strict after-treatment. But owing to this very fact that the section is so

short, this method is adapted only to membranous or to soft cataracts—i. e., to those that have no hard nucleus of any size, since the latter could be removed through such a wound only with difficulty, or could not be removed at all.

As pointed out by Beard, Wilder, and others, linear extraction may be used in cataracts—especially lamellar, traumatic, shrunken and membranous cataracts—occurring in people up to forty, and may sometimes be performed up to the age of forty-five.

(*b*) *Flap Extraction*

967. Technique.—This operation produces in the margin of the cornea a curved section of an extent requisite for the removal of large, hard cataracts. The indication for its performance is furnished by all cataracts which have a hard nucleus of any size and are hence not adapted either for discission or for linear extraction. The operation consists of four steps:

First step: Performance of the section. This is done with the Graefe linear knife, which is introduced in the limbus at S (Fig. 446), in such a way that the cutting edge looks upward. Then the knife is passed through the anterior chamber to the point of counter-puncture, S^1, The counter-puncture should lie exactly opposite the site of the puncture, the two being, at such a level that the upper third or fourth of the cornea is separated by the section so as to form a flap. H. Knapp advised that the puncture be made about 1 mm. above the temporal end, and the counter-puncture 1 mm. above the nasal end of the horizontal meridian. After the transfixion has been made, the section is completed by sawing cuts, so that through its whole extent it divides the sclera just behind the limbus. As soon as the knife has cut through the sclera, it lies behind the conjunctiva, by cutting which a conjunctival flap is formed about 2 mm. broad. In doing this it is advisable to turn the knife rapidly upward so as to divide the conjunctiva quickly. Otherwise, since the conjunctiva is extensible and hence not easy to divide, the knife would strip it up from the sclera a long way back, and the conjunctival flap would become too broad.

The section is made upward, so that in case iridectomy is performed, the coloboma, too, may be situated above and be covered by the upper lid. The section as above described is long enough for the largest-sized cataracts. If we have a cataract to operate upon whose nucleus is probably small, we may make a correspondingly smaller section.

Some operators make the section in the transparent cornea in which case no conjunctival flap is formed. The advantage of the conjunctival flap is that it becomes very quickly agglutinated to the subjacent parts, and so closes the wound exteriorly, even when the edges of the incision in the cornea or sclera have not yet united. It thus protects the wound from subsequent infection.

Second step: Iridectomy. After the conjunctival flap has been turned down upon the cornea, so that the wound may be exposed to view, the latter is entered with the iris forceps, the iris is grasped close to the pupillary margin, drawn out, and cut off with a single sweep of the scissors.

It is best to make the excision of the iris as slender as possible (Fig. 446). With this object in view, we draw the iris from the wound only far enough for its pupillary margin to become visible, and then, holding the scissors forceps perpendicular to the

direction of the wound, snip off simply the apex of the tag of iris. A small coloboma averts prolapse of the iris as certainly as does a large one (see pages 855 and 925), and causes less confusion from dazzling.

Third step: *Opening of the capsule.* This is performed with the capsule forceps, the delicate teeth of which are directed backward (Fig. 444, *e*). The forceps, closed, is introduced and passed on in the anterior chamber until the centre of the pupil has been reached. Here the forceps is allowed to open, and by the exertion of light pressure the capsule is grasped over as large an extent as possible, and is drawn out of the wound.

The opening of the capsule was made by Von Graefe with a cystitome—i. e., with a triangular cutting lancet (Fig 444, *b* and *c*). The capsule forceps is better because with this the anterior capsule is not only split, but also has a piece taken out of it. Thus, the capsular wound is prevented from closing quickly and by its closure interfering with the resorption of the fragments of the lens that remain. Since the employment of the capsule forceps, secondary cataract has become much less frequent, although at present unripe cataracts are operated upon much more often then formerly.

Fourth step: Expulsion of the lens. The Daviel's spoon is placed parallel to the wound against the lowermost part of the cornea, and light pressure is exerted with it upon the latter upward and backward. Instead of the spoon, we may use the finger, pressing with this through the lower lid upon the region of the lower margin of the cornea. The pressure must be stopped the instant the greatest diameter of the lens has passed through the wound.

Enlarging the Section.—If the lens comes out with difficulty, it is proof that the incision needs to be enlarged. This is best done with Stevens's scissors (Fig. 452), one blade of which is inserted into the anterior chamber between cornea and iris in the angle of the chamber. The enlargement is made along the line of the limbus.

After the operation is finished, the "*toilet*" of the eye is next in order. The portions of the cataract which are still left in the eye, and also any extravasated blood, are removed by stroking with the lower lid or a spatula, the iris is replaced from the wound into the anterior chamber by the introduction of the spatula, until the pillars of the coloboma have the proper position (see page 852), then the conjunctival flap is stroked out smooth, and the eye is bandaged.

Many operators follow the extraction with *irrigation of the anterior chamber* with weak antiseptic solutions, partly in order to wash out the fragments of lens which remain behind, and partly to disinfect the interior of the eye. Irrigation sometimes causes permanent opacity of the cornea or iritis, but rarely does so if made with non-irritating solutions, such as normal (or, better, half-strength normal) salt solution or boric acid. Special instruments have been devised for it (syringe of Mac Keown, Lippincott's irrigator), but a simple rubber bulb with slender, flattened glass tip answers all requirements.

Some operators, after the operation is concluded, cover the wound with preformed conjunctival flaps, which are sewed on. The objections to such flaps, as summarized by Colonel Smith, are increased time and traumatism in making the operation, obscuration of the field by blood and loose conjunctiva, difficulty in making the incision of the proper size, and increased difficulty in extracting the lens and replacing the iris.

In *subconjunctival extraction* (Husain) a conjunctival flap is made above the cornea much as for a trephining (§ 959). This is reflected and an incision made with the kera-

tome in the limbus. The incision is enlarged laterally with scissors until it is large enough for the passage of the lens. The succeeding steps are like those of the ordinary operation. After the removal of the lens the flap is put back in place and sutured. Quite similar is the operation done by Czermak.

Corneal Suture.—H. W. Williams applied a corneal suture after the extraction. As the main object of the suture is to prevent loss of vitreous, it should be applied before the latter has a chance to escape, i. e., before the extraction. Hence, Mendoza, Kalt, Stevenson, and others apply corneal sutures before making the section. These sutures are not passed through the entire thickness of the cornea, but only deep enough to take a firm hold, and after passing are drawn out into wide loops which are laid to one side so as not to interfere with the section. After the operation is completed they are drawn up and tied tight. (Cf. the pre-operative placing of sutures in operations for staphyloma and keratoconus—§§ 942, 944).

968. Simple Extraction.—Of the four steps of the operation the second may be omitted, and the operation done *without iridectomy*. The operation is then called simple extraction, as distinguished from the *combined extraction*, or operation with iridectomy. In the latter case the iris must be carefully replaced after the operation is finished, and then eserine must be

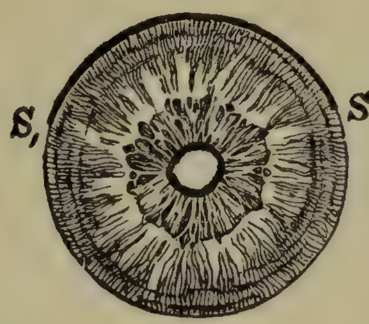

Fig. 446.—Flap Extraction with Iridectomy. Magnified 2 × 1. S S_1, scleral section, concentric with the limbus. a_1 a, edges of the sphincter.

Fig. 447.—Flap Extraction without Iridectomy. Magnified 2 × 1. S S_1, corneal section, which everywhere lies in the limbus. The pupil is greatly contracted by means of eserine. As a consequence of this marked miosis, the pupil is not perfectly circular, but somewhat irregular, and the fringe of pigment lining it has become broader.

instilled in order to prevent by the contraction of the pupil any subsequent prolapse of the iris (Fig. 447). (Some omit the eserine.)

A position midway between the operation with and without iridectomy is the *modified simple extraction* of Chandler, or operation with buttonhole iridectomy. This consists in exsecting, after the delivery of the lens has been effected, a very small bit of that part of the periphery of the iris which lies directly beneath the wound. The small hole thus made is concealed by the limbus and by the upper lid, so that the cosmetic and optical effect of the operations is like that of an operation without iridectomy, while yet the danger of a subsequent prolapse of the iris is rendered very much less (see § 896). Chandler points out that the retained cortical lens matter can be expelled through the hole thus made.

The omission of iridectomy has the advantage of keeping the patient's pupil round and mobile, but it also entails many disadvantages which limit the number of cases in which extraction without iridectomy is indicated. Thus: 1. The delivery of the lens is more difficult without iridectomy, because the lens has to be expelled through the narrow pupil, and to accomplish this pretty strong pressure is required. Hence, this method is not adapted to those cases in which a very easy delivery of the lens is desirable, as, for instance, in cases of tremulousness of the lens in which any kind of strong pressure would produce rupture of the zonula and hyaloid membrane and consequently

prolapse of the vitreous. 2. When the pupil is narrow the removal of cataractous remnants is difficult, for which reason the operation with iridectomy is preferable for unripe cataracts. 3. Extraction without iridectomy is not adapted to cases in which there is a complicated cataract connected with the iris by synechiæ. 4. In spite of the use of eserine, prolapse of the iris may take place in the days following the operation. In this case we are obliged to make a secondary excision of the prolapsed iris. Accordingly, extraction without iridectomy is not adapted to cases which show a great tendency toward prolapse of the iris, nor to those in which we can not count upon the patient's remaining quiet after the operation. It may also happen that an operator may, after taking all these facts into consideration, have decided upon an extraction without iridectomy, and yet in the course of the operation may find himself compelled to excise the iris. This is the case, for instance, when the pupillary portion of the iris is so unyielding (as it often is in old people), that it does not allow the cataract to pass through the pupil, and the latter has to be widened by an iridectomy before the passage can take place. In other cases the delivery of the lens goes on well, but the iris shows a tendency, in spite of careful reposition, to fall again into the wound. In this event it is better to cut it off at once than to run the risk of a subsequent prolapse of the iris. We may therefore say: Extraction without iridectomy gives under favorable circumstances the most perfect result, but is not adapted to all cases, and in many cases it can not be done at all; moreover, owing to the danger of a subsequent prolapse of the iris, it does not attain to the almost absolute certainty of success that belongs to combined extraction.

969. **Extraction in the Capsule.**—The ideal cataract operation would be to remove the lens in the closed capsule, so that no residue of the cataract would remain, no tags of capsule could be incarcerated in the wound and no after-cataract could form by thickening and folding of the capsule that had been left behind. In delivering the lens in its capsule it is desirable to combine the section with an iridectomy, in order that the exit of the lens may be easy. The exit is effected by strong pressure on the lower part of the cornea. But this, again, has the disadvantage that it often produces escape of vitreous and thus renders the operation more serious. The ideal results obtaining in successful cases of this method of operating, which was proposed by Smith, are therefore purchased at the expense of quite a large number of losses.

Extraction of the lens in its capsule was proposed by Pagenstecher for cataracts with thickened capsule. More recently it has been advocated as a routine method of dealing with cataracts of all kinds by *Colonel Smith* of India, who has operated on an enormous number of cases with great success. In the operation practised by him, the eye is first thoroughly douched with 1:2000 bichloride solution. Puncture and counter-puncture are made in the limbus and about in the horizontal meridian of the cornea, the blade of the knife being turned forward so as to cut the cornea more at a right angle and make a perfectly smooth incision which lies wholly in the cornea, but still is large. Iridectomy is then done, special care being taken not to injure the lens capsule. In the expression the assistant holds the lids far from the eye with fingers and with a blunt hook, the patient looks up, and the operator with another blunt hook presses on the cornea till the lens breaks loose and tilts forward, and with the same hook, engages the lens as it comes out. A spoon is placed behind the lens only if vitreous presents, and then not to lift the lens out but to keep the vitreous back. In cases in which the lens refuses to come out, the anterior capsule may be stripped off with the capsule forceps and the lens expressed in the usual way (Vail). Many doubt whether the final results of the operation are as good as those of extraction with capsulotomy. Outside of India it has been adopted by comparatively few operators.

Extraction after Subluxation with Capsule Forceps.—To avoid the dangers of undue pressure and consequent loss of vitreous, that obtain in the Smith operation,

A. Knapp subluxates the lens by traction instead of by pressure. He makes an incision just short of half the corneal circumference and with a conjunctival flap. After making an iridectomy he grasps the lens capsule below its centre with a Kalt capsule forceps, which takes firm hold of the capsule without tearing it (see description Fig. 444 *e*). With this he rocks the lens until the lower portion of the suspensory ligament ruptures and the lens subluxates. Then withdrawing the forceps, he makes pressure straight backward on the lower part of the cornea until the lens turns over and presents. The upper part of the suspensory ligament that still remains adherent to the lens is separated by a lateral or upward stroking motion. A similar operation has been proposed by Stanculeanu. Török grasps the capsule with the Kalt forceps and by making lateral and circular movements with this loosens the zonular attachments; then expels the lens by traction with the forceps combined with intermittent pressure on the sclera with the Daviel spoon.

Removal of Lens by Suction (**Phacoerisis**).—Suction, formerly applied in order to remove soft cataract, has recently been used for hard cataracts also. This procedure was advocated by Hulen and in late years has been done on a large scale by Barraquer who makes the suction with a special apparatus called the erisphake. The section is made in the usual way and is followed by a buttonhole iridectomy (§ 968). The cup-shaped erisphake is introduced into the anterior chamber and is slid under the iris and applied to the lens. Suction is then made, and by gentle vibratory movements the zonular fibres are broken up, so that the lens can be withdrawn in capsule.

970. Other Modifications of the Operation.—(*a*) We give the name of *preliminary iridectomy* to one that is made several weeks before a cataract operation. Many operators perform such an iridectomy even in uncomplicated cataracts because they believe that thus they make the cataract operation itself less severe and hence less dangerous. But preliminary iridectomy is done particularly in unripe cataracts in order to ripen them (*maturation*). This operation which was devised by Förster consists in rubbing the cornea in a circular direction with a blunt instrument (Daviel's spoon or a squint hook) after the excision of the iris. Since the cornea is so thin that it is dimpled by the rubbing, the latter in the pupillary area acts also upon the lens whose capsular epithelium in this area is in part lacerated and detached. Hence, the capsule is made permeable for the aqueous, by means of which the lens fibres are opacified. The massage of the lens results in the latter's becoming completely opaque within a few weeks or even a few days. The extraction of the lens should follow this preparatory iridectomy not sooner than four weeks.

Preliminary iridectomy is usually done only in complicated cataracts. It must above all be done when the cataract is associated with increase of tension because otherwise we should run the risk of having a destructive intra-ocular hæmorrhage (page 786).

Ripening by Förster's method is in general unnecessary. We can without waiting extract even unripe cataracts with success if we open the capsule quite extensively with the capsule forceps.

(*b*) The *preliminary capsulotomy* of Homer Smith is done especially in immature cataract. The capsule is opened with a scalpel-shaped knife-needle four to six hours before the extraction. The aqueous thus gains access to the lens substance, causing the soft portions of the latter to swell and become loosened from the capsule, so that when the extraction is made they come out easily.

(*c*) *Wenzel's Extraction.*—When owing to severe irido-cyclitis there is an adhesion between the surface of the iris and the lens, excision of the iris in the regular way is impossible. We then cut through the iris and open the capsule at the time of making the section in the cornea, by carrying the knife not only through the cornea, but also through the iris and lens capsule, which can easily be done since the anterior chamber in such cases is generally shallow (Wenzel).

971. Accidents Occurring in the Operation for Cataract.—The extraction may be made difficult or fail altogether, owing to accidents of various nature. Many of these are the fault of the operator. If the section proves

to be too short or if the capsule is insufficiently opened, the *delivery of the lens is difficult* or impossible. In this case the section must be enlarged (§ 967), or the capsule must be again ruptured, and this time more thoroughly. If the operator exerts too strong a pressure with his intruments upon the eyeball as a whole, or upon the iris or lens, the *zonula ruptures* and the vitreous gushes out. The greater the operator's skill grows with practice, the less frequently do these unlucky accidents happen to him. But there are other accidents which are caused by the abnormal condition of the eye that is operated upon, and in that case it generally does not lie in the power of the operator to prevent them. The most frequent of these accidents is *prolapse of the vitreous.* This takes place when the zonula ruptures. Such rupture not infrequently happens because the patient screws his lids tightly together and thus presses upon the eyeball. It also occurs when the zonula was defective before the operation, and hence, especially in hypermature and in complicated cataract. The significance of prolapse of the vitreous for the subsequent course of the operation differs according as it takes place before or after the delivery of the lens. In the former case the lens cannot be evacuated in the usual manner by pressure exerted upon the eye; for then the larger part of the vitreous would escape before the lens itself came away. Hence, the lens must be drawn out of the eye with instruments—i. e., extracted in the true sense of the word. For this purpose the proper instruments are Weber's loop (Fig. 444, *f*) or Reisinger's, or Agnew's double hook, which are introduced behind the lens and lift it out by force of traction.

Prolapse of the vitreous is much less to be dreaded when it takes place after delivery of the lens. The most serious harm that prolapse then does is that it hinders the accurate replacement of the iris, and also that the prolapsed vitreous lies between the lips of the wound and prevents their accurate coaptation. The vitreous may also give rise to suppuration of the wound, since it is very apt to become infected.

If the vitreous escapes in any quantity, there will be more or less collapse of the eyeball. This may be remedied by injecting warm sterile salt solution into the anterior chamber (see § 897).

For *hæmorrhage* after cataract extractions, see § 898.

A rare but unpleasant accident is that in which the lens, before being delivered, becomes *luxated,* and disappears into the vitreous from which it must, if possible, be extracted (§ 975).

972. Result of the Cataract Extraction.—An eye whose lens has been removed is *aphakic.* It presents the following appearance when the operation and the healing of the wound have pursued a normal course: The cicatrix left by the operation, if situated in the cornea, appears as a narrow gray line; if the section has been made in the limbus or in the sclera, the resulting cicatrix becomes later on almost unrecognizable. The anterior chamber is abnormally deep, the iris is tremulous and, when the operation has been done with iridectomy, presents a coloboma above. The pupil is of a pure black, but on lateral illumination presents to view a membrane of silky lustre, which not infrequently is thrown into folds.

This is the lens capsule which was left in the eye when the lens was removed. (This, of course, is absent if the extraction has been made in the capsule.) It is, however, only the posterior capsule of the lens (h, Fig. 448) that is intact throughout. The anterior capsule ($v\ v_1$) where it occupies the area of the pupil is lacerated and in part deficient; remains of it are applied directly to the posterior capsule. Since both capsules are transparent, the pupil appears round and black. Behind the iris the anterior capsule, where it was protected from the instrument used for making the opening, is preserved intact, and in conjunction with the posterior capsule incloses remains of the lens, which correspond to what was once the equator of the latter (Fig. 448, k). As the anterior and posterior capsules become agglutinated together in the pupillary area, these remains of the lens are shut off from the aqueous, and can not therefore be absorbed; in fact, they usually increase in amount, owing to *proliferation* of the cells of the capsule. They then form an annular swelling lying behind the iris (Soemmering's crystalline swelling). The lumen of the ring, which corresponds to the pupil, is closed by a thin, transparent membrane consisting of the two opposed layers of the capsule. As the opaque and swollen mass behind the iris is completely concealed, it in no way interferes with vision. If the operation has been made with an iridectomy, the mass is wanting in the course of the coloboma, because the anterior capsule was opened there likewise.

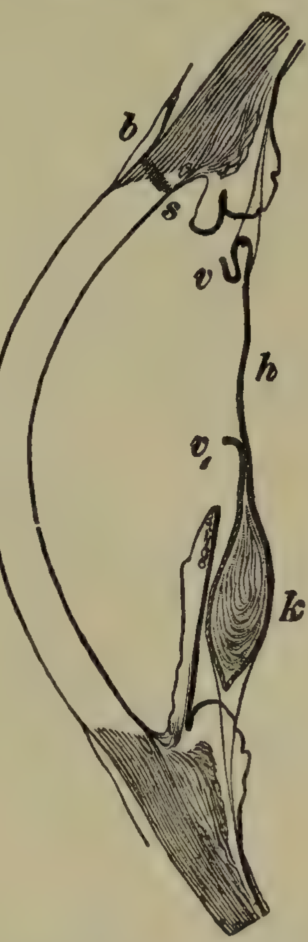

FIG. 448.—CROSS SECTION THROUGH THE ANTERIOR SEGMENT OF AN EYE UPON WHICH AN EXTRACTION HAS BEEN PERFORMED BY MEANS OF A FLAP SECTION. Magnified 4 × 1.

The section, s, which was made upward lies by its inner portion in the cornea, by its outer portion in the sclera; the latter is covered by the conjunctival flap, b. At a point corresponding to the section the iris is wanting except for a short stump. The anterior capsule presents a large aperture, the edges of which ($v\ v_1$) are curled over, while the posterior capsule, h, although slightly wrinkled, is unruptured. In the lower part of the eye behind the iris the remains of the lens which are inclosed in the folds of the capsule, form Soemmering's crystalline swelling, k, which is wanting in the parts above that correspond to the coloboma.

The removal of the lens abolishes the *accommodation* of the eye and produces a great alteration in its *refraction* (see § 193).

973. Secondary Cataract and Changes due to Inflammation.—It often happens, even in cases in which the operation has been well performed, that the result of the operation is impaired by the *retention of portions of the cataract.* This happens particularly when the operation is done on immature cataracts, but by no means fails to occur also in those that are mature and hypermature. If the anterior capsule is thoroughly opened, the portions of the lens left behind (if they were not already opaque previously) grow opaque, swell up, and become absorbed. In this case, therefore, a pure black pupil is ultimately obtained. But if the layers of the capsule become agglutinated early and shut off the remains of lens substance from the aqueous, these remains are not absorbed but persist as a white membranous opacity. This is called after-cataract (*cataracta secundaria*). If this is

present in only one part of the pupil while another part of it is quite clear, the sight may be perfect. But if the whole pupil is filled by the secondary cataract, the sight is diminished in proportion to the density of the opacity. It may also happen that the after-cataract does not develop until later on; the epithelium of the anterior capsule, which has been left behind, proliferating and inducing a secondary thickening and opacity in the latter. Similarly the capsule, even without becoming opaque, may induce diminution in sight, if in the course of time it becomes more and more wrinkled and thus causes irregular refraction of the rays of light.

After-cataract, when it interferes with sight, requires a secondary operation—namely, discission, or linear extraction. The secondary operation should not be performed until the eye ceases to show any trace of irritation, and in no case sooner than two weeks after the cataract extraction.

The result of a cataract operation may furthermore be impaired by *inflammation* (§ 902). If suppuration of the wound sets in, the eye is almost always lost. If irido-cyclitis develops, the secondary cataract is united by the exudate which is formed, to the iris and even to the ciliary processes (cataracta secundaria accreta). It depends upon the condition of the light perception whether the sight in such a case can be restored or not by a secondary operation (iridectomy or iridotomy).

974. **History of Cataract Operations.**—In the preceding pages it has been shown that there are various ways open to us for removing cataract. We may either subject it to resorption by means of discission, we may tear a hole in it by dilaceration, or we may remove it altogether from the eye. But not even by this list are all the methods of restoring the sight of an eye blind with cataract exhausted. We might also, instead of removing the opaque lens, push it away from its place behind the pupil so that the latter becomes free again. This artificial luxation is not only practicable, but as a matter of fact it has been practised for a thousand years; it is the oldest method of operating for cataract. This method, called *depression* (or *couching*) *of cataract* (depressio cataractæ), was made in the following way: A needle was passed into the sclera on the outer side of the margin of the cornea and about four mm. behind it, and it was pushed forward until at length it lay against the upper border of the lens. Then the point of the needle was lowered by a sweeping movement, and the lens was thus depressed into the vitreous. The moment this was done the pupil became black and the patient regained his sight. This was the only method of operating upon cataract practised in ancient times and throughout the Middle Ages. As time went on it was modified in various ways. The last and most important modification consisted in turning the lens over instead of depressing it. The needle in this case was passed by the margin of the pupil and into the anterior chamber, and with it pressure was made upon the upper part of the anterior surface of the lens. The latter was thus turned over in such a way that its anterior surface looked upward, its posterior surface downward. This procedure was called *reclinatio cataractæ.*

The operation above mentioned, or "cataract pricking," was, as a rule, practised by specialists. In the Middle Ages these went from one annual fair to another, and there operated upon those who were blind with cataract. When the operation had been successfully performed and the honorarium had been paid, the "cataract pricker" traveled to another place. He did not see his patient again after the operation, and it was a good thing for him that he did not, for the later consequences of the operation were as melancholy as the immediate result was brilliant. For the eyes very often were destroyed either by inflammation or by increase of tension. The inflammation probably was caused as a rule by infection with the cataract needle and not infrequently was transmitted to the other eye in the form of a sympathetic ophthalmia. At present, inflammation might generally be avoided by operating aseptically, but we

have no means of combating the other deleterious consequences of depression or reclination, and particularly the increase of tension that so frequently occurs when the lens is luxated. The operation is still much practised by native physicians in India.

It sometimes happens after depression or reclination that the lens fails to remain in its place in the vitreous, particularly when the latter is liquefied. In such cases either immediately after operation or later, in some cases not till years afterward, it rises and places itself in its old position behind the pupil; it may even pass through the pupil into the anterior chamber. Such cases of luxation of the lens into the anterior chamber gave the first occasion for the performance of *extraction* of cataract. This method of operating, if we are to believe some authors, was perhaps practised now and then even in ancient times, but at any rate it had in the Middle Ages fallen completely into oblivion. The first information that we have in regard to it we get from the seventeenth century, when there are several instances in which the lens was removed from the anterior chamber into which it had got after the operation of depression. The Frenchman *Daviel* had already done this in several cases, when in the year 1745 he first dared to undertake this operation upon a cataract which was situated in its normal position. In so doing Daviel inaugurated a new era in the history of cataract operations, since from that time the extraction of cataract began more and more to take the place of depression.

FIG. 449
Beer's operation for cataract by inferior flap with a Beer's knife. Beer's knife is also used for the ablation of staphylomata of the cornea (§ 942).

The original method of Daviel was naturally very much in need of improvement. Of the many modifications which it underwent in the course of time the last and best was that of *Beer*. The latter made the section with a knife invented by himself (Fig. 449) which broadens from point to handle so as to have a wedge shape. With Beer's cataract knife it is possible to complete the section by simply pushing the knife forward after it has been entered, and owing to this fact the section acquires a high degree of regularity. The section ran somewhat inside of the limbus, and separated exactly the lower half of the cornea from the sclera. Then, after opening the capsule, the lens was delivered, but no part of the iris was excised.

Beer's procedure was soon generally adopted, and was for a long time the prevailing method. In successful cases it gave ideal results. The pupil was black, round, and perfectly movable, and it was only upon close examination of the eye that it could be discovered that an operation for cataract had been performed at all. Unfortunately, it always happened that a considerable number of eyes were lost after this operation, especially by suppuration of the cornea. As at that time it was not known that this was caused by infection of the wound, the method of operating, and particularly the way in which the section was made, were regarded as accountable for it. Hence, other better procedures were sought after, and this time Von Graefe was the one to take the most important step forward and create a revolution in the method of performing extraction, by the invention of his method.

Von Graefe considered that the cause of the suppuration of the cornea in Beer's method lay in the shape of the section which was made with a flap. This gives rise to great gaping of the incision, in consequence of which the lips of the wound are not properly applied to one another, and this fact was supposed to furnish the cause of the suppuration. Von Graefe accordingly believed that the linear incisions were preferable, as he had become convinced of the promptness with which they healed in the case of simple linear extraction, an operation which had already been practised by him. Accordingly, he, as well as others, attempted to apply the *linear section* which was made with the lance knife, and which originally was employed only for soft or shrunken cataracts, to large cataracts with a hard nucleus. With this object these experimenters tried to make the linear incision as large as possible by placing it in the

upper part of the cornea, and by combining it with iridectomy. Others tried to diminish the size of the lens first by crushing it so as to be able to extract it through the section. But these attempts were all unsuccessful. The section always remained too small for the cataract, which in its passage contused the lips of the wound, so that inflammation frequently ensued. *Jacobson*, who sought the remedy in another way, obtained better results. He placed the section in the sclera. He gave up the linear character of the section, and made a flap incision, skirting the lower margin of the cornea, but situated still in the sclera. With this he combined iridectomy. This method gave better results, and particularly a less frequent suppuration of the wound. The cause of this was regarded as consisting in the fact that the sclera, being a vascular tissue, is less disposed to suppuration than the non-vascular and hence more poorly nourished cornea.

Von Graefe now attempted to combine in a new method both advantages—namely, the linear character of the section, which ensures a good coaptation of the edges of the wound, and the position in the sclera, which affords protection against suppuration of the wound. It soon became apparent to him that a linear section, which should be of the necessary length and situated in the sclera, could not be performed with the lance-shaped knife. The lance knife must be pushed forward parallel with the plane of the iris, and therefore, as soon as it is desired to make a wound of any length at all, produces a section which is nearly concentric with the margin of the cornea, and hence has the character of a flap. Von Graefe accordingly devised the narrow or linear knife, which soon proved to be one of the most useful instruments in ophthalmology. With this knife he performed the section in such a way that at its centre it was in contact with the summit of the cornea, but at its ends was removed a considerable distance from the corneal margin. The point of entry is determined by means of a tangent ($t\ t_1$, Fig. 450). which is conceived to be drawn through the external margin of the cornea; the puncture (s) is situated in this and at that point of it where it is at a distance of one to one and a half mm. from the margin of the cornea. The point of emergence, s_1, lies directly opposite the point of entry. While the section is being performed, the edge of the knife, which at first was directed straight upward, is turned a little forward, so that the centre of the section gets to lie right behind the limbus. The new way of making the section had the conjunctival flap and iridectomy as its necessary concomitants. The iridectomy had to be performed as a regular thing, since otherwise the iris, owing to the peripheral situation of the wound, would certainly have become incarcerated in it. (In the old methods of extraction iridectomy was done only when there was some special necessity for it.) As cataract operations combined with iridectomy were called "modified" operations (so named in contradistinction to those that are "simple," i. e., performed without iridectomy), Von Graefe called his new method "*modified linear extraction.*" Later on, one made a virtue of necessity, and laid special stress upon the advantages of the iridectomy that was combined with the extraction. It prevented, they said, the incarceration of the iris, made it possible to open the capsule more thoroughly, facilitated the removal of the remains of the cataract, and afforded a protection from subsequent inflammation of the iris. Hence, people soon got to regard the excision of the iris as one of the additional advantages of the new method.

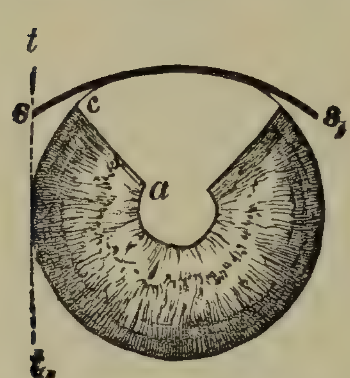

FIG. 450.—MODIFIED LINEAR EXTRACTION BY VON GRAEFE'S METHOD. Magnified 2 × 1.
The section, $s\ s_1$, lies in the sclera; the iris shows a large coloboma with very divergent limbs, $a\ c$.

The results of Von Graefe's linear section were, in fact, much better than those which the earlier methods had given. Suppuration of the wound, in particular, had become less frequent. But yet the method had its dark side, too. Its performance required more operative skill, and the delivery of the lens was made more difficult, owing to the slight tendency to gape that the wound possessed. Other disadvantages arose from the peripheral situation of the section, which brought the latter, espcially at its extremities, close to the zonula and the ciliary body. Prolapse of the vitreous was frequently met with, and also inclusion of the limbs of the coloboma in the wound. While suppuration of the wound proved to be less frequent, iritis and irido-cyclitis became proportionately more common, and in consequence sympathetic disease of

the other eye was observed more frequently than before. These facts induced operators to depart more and more from this section, which was felt to be too peripheral in its situation, and in particular led them to place the extremities of the latter nearer the cornea. If Von Graefe's original section, as Von Graefe himself gave it, was itself not a pure linear incision, this is still more the case with the section as it was subsequently performed. It had become a curved section, forming an arc of small altitude. Under this somewhat modified form, scleral extraction soon became the method that was generally employed.

Since with the introduction of the antiseptic method the danger of suppuration of the wound had been reduced to a minimum, operators no longer hesitated to make the section in the limbus or in the transparent cornea itself—as for example, is done in the corneal flap extraction above described. Other improvements that have been made have had regard to the *excision of the iris*. Operators had already learned by careful reposition of the iris to avoid the dangers arising from its incarceration. When this is done there is no need of making a large coloboma such as Von Graefe had described —indeed it is better to make the iridectomy as slender as possible.

When at length surgeons again adopted the corneal section, they took the last step and operated entirely *without iridectomy,* as Daviel and Beer had done in former times.

975. Removal of a Dislocated Lens.—A lens dislocated into the anterior chamber should always be removed. The lens if soft, can be needled and the fragments be removed by linear extraction. If the lens has a large hard nucleus, a sufficiently large incision should be made, through which the lens can be lifted out with a hook and spatula or wire loop. In order to prevent the lens from slipping back through the pupil, the latter should be contracted with eserine.

A lens dislocated into the vitreous causes trouble sooner or later. It should accordingly be removed early. In the case of a lens dislocated after cataract extraction it is sometimes possible to expel it by Knapp's method of making systematic pressure with the lower lid against the lower part of the sclera right toward the centre of the eyeball (De Schweinitz). Otherwise, especially in the cases of spontaneous dislocation, in which the vitreous is usually fluid, the lens may either be lifted out with a delicate wire loop or flat (Smith) spatula, or be drawn out with a bident or double hook (Agnew) or with a not too curved sharp Tyrrell's hook (Fig. 444, *d*). The hook, with which the lens can often be slid along the anterior wall of the vitreous chamber with but little disturbance of the vitreous, serves better than the loop, unless the lens has sunk to the bottom of the chamber, where the hook might readily become entangled in the tunics of the eye (Beard). In Paine's very serviceable operation the pupil is first dilated with atropine and a wide iridectomy made. The lids are lifted away from the eye by raising the speculum or by Smith's method (§ 969). Then the depth of the eye is fully illuminated with an electric light, placed a little above and to one side of the eye, and with a loupe, so that the operator can look down to the very bottom of the vitreous and see the lens there. He then engages the lens with a Stevens hook, draws it up to the incision, slips a Smith spatula behind it as a support, and either draws the lens out with the hook or expels it by gentle pressure on the cornea.

A subluxated lens, such as is found after reclination as practiced in India, often floats up behind the pupil after a regular cataract incision is made, and may then be removed by slipping a spatula behind it and then sliding it up along this by pressure made on the cornea (Colonel Smith).

III. Posterior Sclerotomy

976. In posterior sclerotomy (sclerotomia posterior) an opening is made in the posterior division of the sclera so as to enter the vitreous chamber.

The section should be meridional—i. e., run from behind forward, since this is the direction of most of the scleral fibres, and hence such sections gape the least, and besides with the incision in this direction the fewest chorioidal vessels are encountered, since these too have mainly a meridional course (Fig. 31). The position of the section must be chosen, so that neither an ocular muscle nor the ciliary body is injured. For the

latter reason the section should not extend farther forward than at most to a point six mm. from the corneal margin.

In order to cover in the section, a quadrantal flap of conjunctiva is first formed and laid back, then the puncture is made in the area thus laid bare. and the conjunctiva is replaced.

The indications for sclerotomia posterior are:

1. *Detachment of the retina*—A broad Graefe knife is thrust into that spot of the sclera which corresponds to the most prominent part of the detachment. As soon as the knife has penetrated the sclera and chorioid and into the subretinal space, it is turned a little, so that the wound is thus made to gape. We then observe the conjunctiva lifted up by the subretinal fluid which escapes from the wound, so as to form a yellowish vesicle. As soon as the fluid ceases to escape, the knife is withdrawn again.

2. *Glaucoma,* when the anterior chamber is obliterated, and hence iridectomy has become technically impossible (cases of glaucoma malignum and glaucoma absolutum). After the operation the anterior chamber is usually restored, so that an iridectomy can be performed later. Posterior sclerotomy is also done in cases of great tension,to be followed in half an hour by an iridectomy which is then done under more favorable conditions. The operation is performed in the same way as in detachment of the retina, except that instead of subretinal fluid some vitreous is withdrawn. Owing to the greater consistence of the vitreous it is usually necessary to make the section somewhat longer.

3. A meridional section of pretty great extent is made when *extraction of a foreign body* or of a *cysticercus* from the vitreous, is in question. In this case it is well to add a small cross-cut at the end of the meridional incision, so as to give the latter a T-shape (Weeks). See § 978.

4. To *divide a membranous cataract* by scleronyxis (see § 964).

977. Posterior Sclerectomy.—In detachment of the retina in myopic eyes, Müller *exsects* from the sclera in the posterior half of its temporal portion a piece eight to ten mm. broad and twenty mm. long, and shoves it beneath the edge of the wound so that there is a duplication of the sclera. The chorioid is not removed but simply punctured to let out the subretinal fluid. The operation is done with the idea of preventing the stretching and pull which the elongation of the eye produces.

Parker and others after laying back a conjunctival flap over the most dependent portion of the detached retina make an opening in the sclera with a two or three mm. *trephine* and allow the subretinal fluid to escape. The flap is replaced and the patient kept in bed ten days. Thompson and Curtin follow the trephining either immediately or later by withdrawal of the subretinal fluid with an aspirating needle.

IV. Removal of Foreign Bodies from the Eye

978. The removal of foreign bodies (including entozoa) presupposes a careful preliminary examination to determine the precise location of the foreign body. In some cases this can be done with the ophthalmoscope. Metallic foreign bodies can be located by radioscopy, and the presence of a magnetic metal can be detected by the magnet (see § 857).

If the wound is still gaping and is large enough, we can pass a well-disinfected instrument in through it, and endeavor to grasp the foreign body. If the wound is not adapted for this procedure, either because it has already closed up or because it presents unfavorable conditions on account of its size or position, it is better to make a new wound, located in the cornea or the sclera, according to the situation of the foreign body. In placing it in the sclera, the region of the ciliary body must be avoided; the section must lie behind the latter, and is best made in a meridional direction (i. e., running from before backward), since such wounds gape the least (see also § 976). Through the wound we enter with the instruments in search of the foreign body; but the operation of grasping and extracting

it is often attended with great difficulty, and very frequently miscarries. The best prospect of success is afforded by foreign bodies in the anterior chamber, since we can be guided by sight in taking them out; also by fragments of iron, for whose extraction we employ an electro-magnet.

The attempt to remove the foreign body which has penetrated into the eye is often beset with great difficulties, and very frequently is unsuccessful. No fixed rules can be laid down for the procedures to be employed for this purpose, as almost every individual case has its peculiarities and calls for an operation devised specially for itself.

The extraction of a chip of iron from the vitreous by means of a *bar magnet* introduced through an incision on the sclera was first performed by MacKeown (1874). Hirschberg constructed the first available *electro-magnet*, which is the form now commonly employed. We introduce the blunt point of this either through the wound itself, in case this is large enough and is still open, or we make an incision in a suitable situation in the cornea or sclera, according to the location of the foreign body. Now very powerful electro-magnets are used (Haab, and others). Mellinger's inner pole magnet is an electromagnet in the form of a ring big enough to contain the patient's head. An iron rod brought into the centre of the ring becomes magnetic and can be used to withdraw the foreign body. Giant magnets are not introduced into the eye, but are merely applied to it exteriorly, as they are able to attract even small fragments of iron when at some distance from them. They have the following advantages over the small magnets: (1) It is not necessary to make a wound in the sclera; (2) the situation of the fragment need not be known precisely; (3) even very small fragments can be removed in this way.

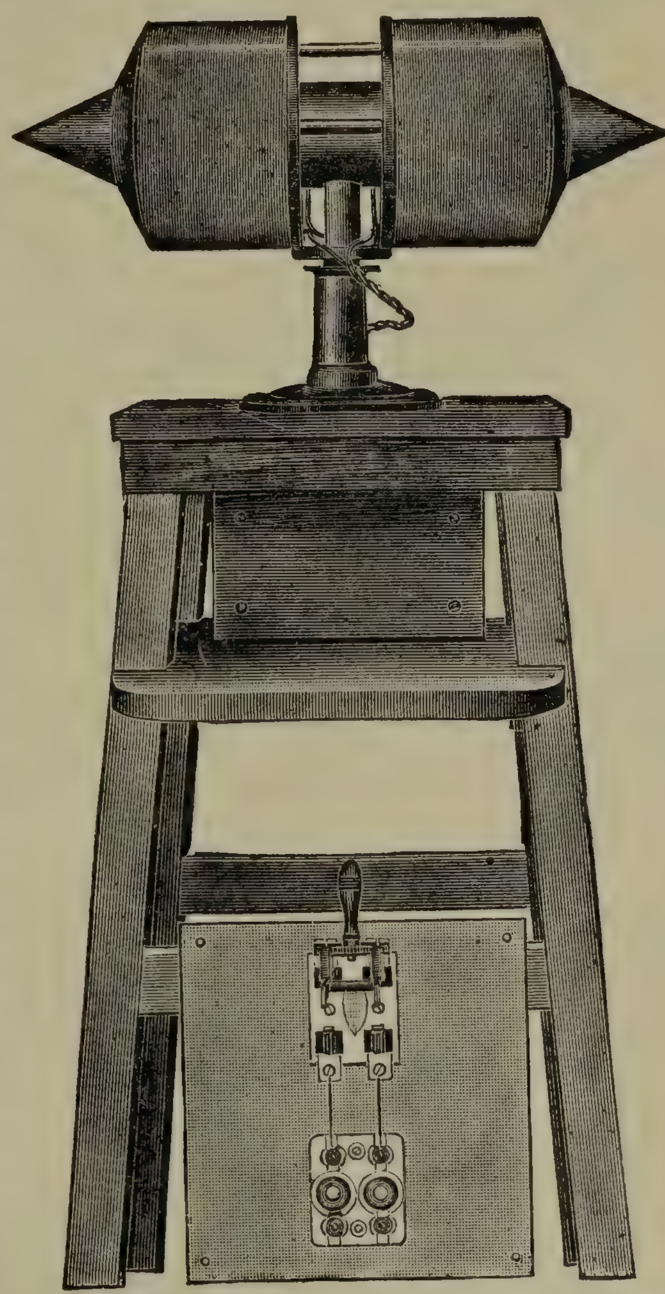

Fig. 451.—Haab's Giant Magnet. (After Norris and Oliver.)

If the wound of entry is sufficiently large, the magnet should be applied there. Otherwise it is better to place the tip of the magnet against the centre of the cornea, so as to bring the fragment against the posterior surface of the iris. When this latter bulges, the patient's head is so turned that the direction of the magnetic attraction shall be parallel with the plane of the iris. The foreign body is thus drawn down into the pupil and anterior chamber, from which it may then be removed by an incision made at the margin of the cornea (anterior operation). This is the operation of election when the foreign body is in the lens or iris. When it is in the vitreous, many prefer to withdraw it either by the giant magnet or the hand magnet through a T-shaped incision in the sclera as close as possible to the site of the foreign body as previously determined by radioscopy. If the situation of the foreign body is determined accurately, so that the minimum of traumatism is produced in removing it, and if conjunctival flaps are used, this posterior method appears to give good results. In certain cases when the foreign body does not yield to the magnet, Jackson connects the latter with a pair of sharp scissors, which, being thus rendered magnetic, is introduced into the wound and is used both to tunnel a way to the foreign body and to withdraw the latter when detached.

CHAPTER XXXVII

OPERATIONS UPON OCULAR MUSCLES AND ORBIT

I. Squint Operations

(*a*) *Tenotomy*

979. Technique.—Tenotomy is performed upon the internal or external rectus; less often, upon the other ocular muscles.

Tenotomy of the internal rectus by Von Graefe's method is performed as follows: An incision about 10 mm. long is made through the conjunctiva at right angles to the tendon and right over the insertion of the latter. A buttonhole is made in Tenon's capsule above or below the margin of the tendon and close to its insertion, and through this hole a squint hook is inserted beneath the tendon and raised so as to lift the latter away from the sclera. The tendon is then divided close to the hook with the scissors.

After dividing the tendon we must see whether there are not some strands of tendon still remaining at its upper or lower border. A squint hook (Fig. 452, *a*) is accordingly passed in beneath the tendon and is carried upward and downward so as to explore all parts, the intention being to catch up upon the hook any fibres that may chance to be intact and then to divide them.

After the division of the tendon has been completed, a test must be made of *effect* of the operation, which should be neither excessive nor insufficient. We first (1) make the eye that has been operated upon turn toward the side of the divided muscle. If the tendon has been cut through completely, there must be a considerable diminution of the motility inward. If the eye can be turned inward as well as it could before the operation, this proves that some strands of the tendon have remained undivided. As in this case the result of the operation would be nil, these fibres must be sought out with the hook and divided. On the other hand, the diminution in motility may be too great, in case we have not only divided the tendon, but have also loosened up too much its connections with Tenon's capsule. In this event the effect of the operation must be decreased, the tendon which has slipped back too far being re-attached further forward with stitches. (2) We determine the convergence near point. If this has been carried out beyond 13 cm. from the base line, the effect of the operation is excessive. The working capacity of the internus is then so greatly weakened that we should have to fear the subsequent development of a divergent strabismus. In this case too, the effect of the operation has to be reduced again. 3. Subject to the above limitations, we try to push the operation until we have produced an undercorrection of several degrees. Whether we have succeeded in this or not we determine by making prismatic measurements of the deviation for distance and near with the screen, parallax, and, if binocular vision is present, with the Maddox rod and

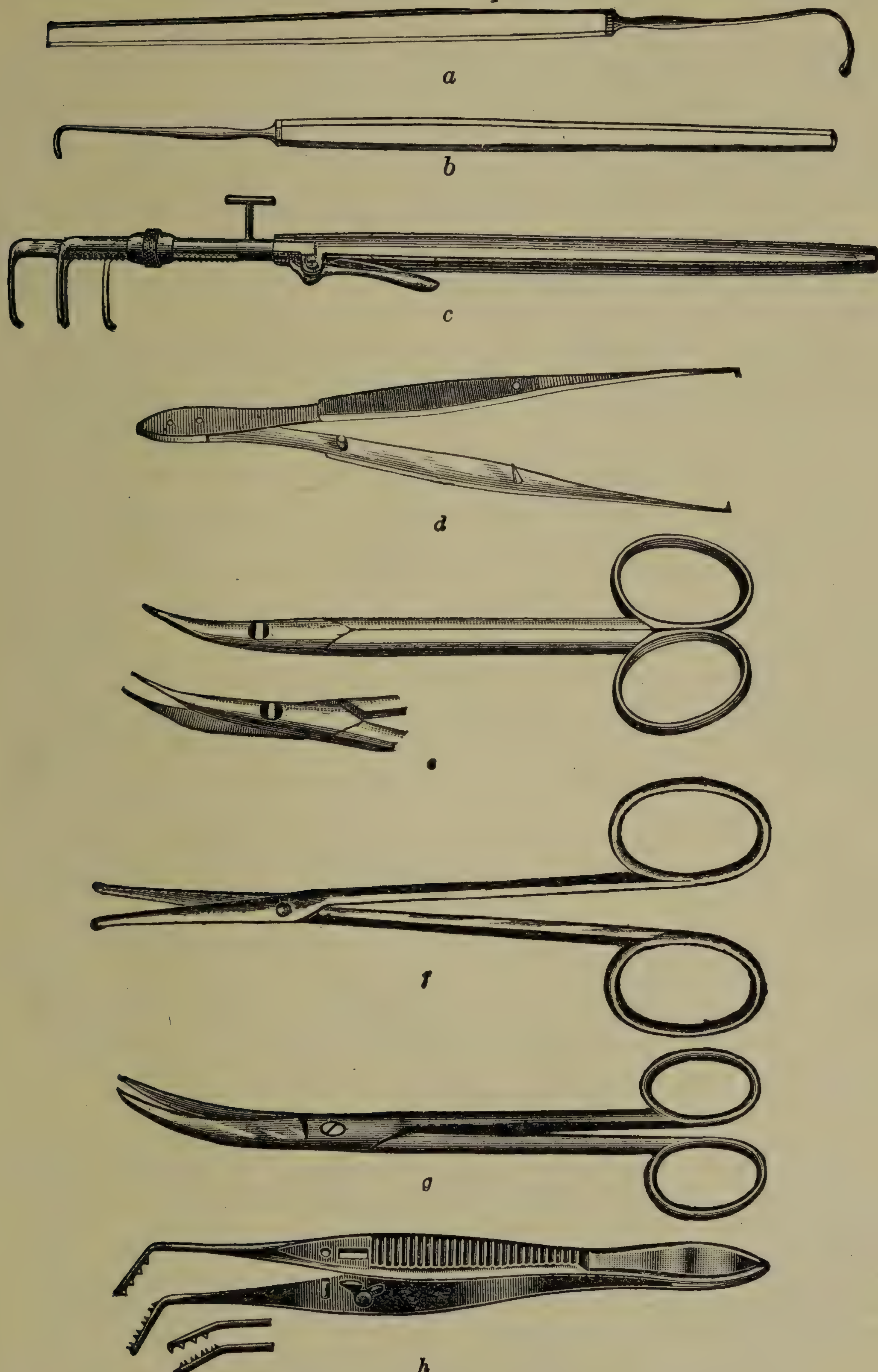

Fig. 452.—Instruments for Tenotomy, Advancement, and Enucleation.

a, squint hook for picking up the tendon and putting it upon the stretch. *b*, Stevens's tenotomy hook, used especially in performing partial tenotomies. *c*, Bruns's modification of Clark's hook for tucking-advancement. *d*, strabismus forceps for taking up conjunctiva and tendon in the button-hole operation. *e*, Steven's tenotomy scissors, with cutting edges near the end. *f*, straight probe-pointed scissors. *g*, heavy scissors for dividing the optic nerve in enucleation. *h*, Prince's forceps for holding the tendon securely in advancement. Similar in principle is Reese's forceps used in resection. (Fig. 454, B, C, D) Other instruments used are a spring speculum or Desmarre's retractor, for holding the lids open, and a fixation forceps (Fig. 388). A delicate needle holder and curved needles are also required for stitching the conjunctiva, and, in the case of advancement, for suturing the tendon.

phorometer, or red glass (see §§ 304-305). By these tests we can measure to within 0.5 *d* of the actual deviation. The deviation is determined by these means before the operation and repeatedly during the operation, which is carried on by cautious division of the lateral attachments of the tendons until a satisfactory result is secured.

Tenotomy of the external, superior, and inferior recti is performed in an analogous fashion. We must simply bear in mind that the insertion of these muscles lies farther from the cornea than does that of the internus (see § 54).

In tenotomy of the *external rectus*, we usually wish to secure a very thoroughgoing effect, and unless this is accomplished and a moderate primary over-correction obtained, the after results are apt to be disappointing. It is usually well to get as the immediate result of the operation a slight limitation in outward movement, but this should not exceed 1 or 2 mm. In tenotomy of the *superior* and *inferior recti* great care must be taken not to produce even a slight over-correction and in no event to make very extensive division of the lateral bands attaching the tendon to the sclera. Otherwise a complete paralysis of the muscle may be produced which is pretty hard to remedy. This paralysis may occur long after the operation. Tenotomy of the inferior rectus should in general be avoided, as the results of operation on this muscle are particularly uncertain and paralysis of it causes greater discomfort to the patient than that of any other muscle, since it gives rise to confusing diplopia in looking down, i. e., in the habitual direction of the gaze.

When, by testing in the manner given, the effect of the operation is deemed satisfactory, the operation is finished by uniting the conjunctival wound with a suture.

The operation can be rendered nearly painless if, besides instilling cocaine and adrenaline before the operation, some cocaine or procaine solution is injected beneath the conjunctiva at the spot where the tendon is divided. This, however, sometimes puffs up the tissues enough to obscure the proper relations of the parts.

The squint operation as first performed by Dieffenbach in 1839 was a division not of the tendon but of the muscle itself, i. e., was a *myotomy*. This often produced complete paralysis of the muscle with corresponding excessive over-effect. Böhm later introduced tenotomy and Von Graefe showed how to regulate the effect.

Some use Critchett's *subconjunctival* method. In this the tendon is not exposed by a long conjunctival incision, but a buttonhole is made in the conjunctiva and capsule above or below the tendon and just beyond its insertion. (Thus in a tenotomy of the internal rectus it would be made at a point six or seven mm. to the inside of the cornea and on a level with the lower corneal margin.) Through this hole a hook is inserted beneath the tendon, and on this the tendon is divided by scissors passed beneath the conjunctiva. No sutures are necessary. The translator prefers the open method described in the text.

In *Snellen's* subconjunctival tenotomy a buttonhole is made through conjunctiva, capsule, and tendon in the middle of the latter and just beyond its insertion. Starting from this hole the tendon is divided upward and downward by the scissors carried underneath the conjunctiva. This method was used by Stevens in his partial tenotomies (see § 982).

When we wish to secure a *large effect*, especially in operating on the external rectus, we may divide the tendon two or three mm. beyond the insertion. As it is possible in this way to cause a complete paralysis of the muscle, it is well to combine the tenotomy with the insertion of a stitch (see § 981). Panas reinforced the effect of a tenotomy of the internus by pulling the eye forcibly outward with a strabismus hook inserted under the tendon. Gruening reinforced the effect of a bilateral tenotomy of the externi by inserting a stitch through each tendon stump and tying the two stitches tight over the nose so as to place both eyes for twenty-four hours in a position of extreme convergence. This is an application of the thread operation devised by Von Graefe to increase the effect of an advancement.

When a tenotomy is done merely to *relieve a temporary strain* on the opposing muscle (as in advancement of the latter when we wish the stitches to be subjected to no extra pull), it should involve simply the insertion without going into the lateral attachments or producing any real impairment of rotation in the muscle tenotomized.

It is possible to tenotomize the *superior oblique* by dividing the reflected tendon through a cutaneous incision in the brow. Tenotomy of the *inferior oblique* is done by dividing the tendon of origin of the muscle through a cutaneous incision 15-20 mm. long, made at the lower inner angle of the eye just below the lacrimal sac. The incision is carried down to the bone, and the orbital septum is divided just where it joins the lower and inner margin of the orbit. A squint hook is swept inward along the floor of the orbit with its point directed upward and inward until it engages the tendon. The latter, which is more or less buried in fat and fascia, is completely divided with the scissors. The tendon usually retracts, but to prevent possible readhesion two or three mm. are exsected. The effect is a complete paralysis of the muscle.

980. Method of Action.—Tenotomy of a rectus acts by displacing the insertion of the divided muscle to a spot situated farther back. The divided tendon glides back upon the sclera and forms a new attachment to the latter. Owing to the fact that the insertion of the muscle now lies farther back, the effect of the latter upon the eyeball is permanently impaired.

How does tenotomy of a muscle *weaken* the latter? Let us assume that a tenotomy has been performed upon the right internus for right convergent strabismus. The right external rectus, which was stretched and elongated as a result of the convergent squint, now tends to return to its normal length. Hence, after the division of the tendon of the internus, the externus draws the eye outward, and thus diminishes the strabismic deviation. In proportion as the eye thus rolls outward, the tendon of the internus glides backward over the sclera. This retraction is increased by the elastic contraction which every divided muscle shows: hence, the tendon of the internus lies farther back upon the sclera, and becomes attached there anew. The insertion of the muscle is thus approximated to its point of origin at the optic foramen, and the muscle is shortened. Before being divided, the muscle in its relaxed state had a certain length, from which by its contractile force it was able to shorten down to a certain minimum. After the division this minimum remains the sâme, but the length of the muscle in the state of relaxation is less; hence, the difference between the state of rest and of maximum contraction is disminished. But this difference corresponds to the power of adduction of the eye, which, accordingly, is permanently reduced after the tenotomy. We can readily convince ourselves that, after an operation, the eye cannot be brought inward as far as it could before; indeed, it is from this fact that we determine whether the operation has been successful.

It is hence clear that the correction of the faulty position due to the squint is purchased at the expense of the adduction. In fact, the loss in the motility inward is always greater than the gain in the position of the eye. The former loss is of no

moment in cases in which the adduction is greatly increased. Here, even if this is reduced by the operation to a point somewhat below its mean value, this reduction would still fail to become noticeable except when the eyes were in the extreme lateral position. But the case is different when the attempt is made to correct a marked strabismic deflection by a very thoroughgoing tenotomy of the internus, with which there would necessarily be associated a considerable diminution of the power of adduction. We would then, it is true, have the eyes in a correct position while the gaze is directed straight forward; but as soon as the patient wished to look toward the side where the tenotomy had been done (e. g., in the case of right convergent strabismus, to the left) the eye that has been operated on would not be able to follow the movement properly. In this direction of the gaze divergent strabismus would appear just as in the case of a paralysis of the internus. We should not therefore try to correct a marked strabismic deviation by making the tenotomy more extensive, but should increase the effect by a simultaneous advancement of the external rectus. It is still better to distribute the operation between the two eyes (see § 991).

In tenotomy the setting back of the tendon is mainly due to the fact that after the latter is divided its antagonist draws the eye toward its own side. The *effect* of a tenotomy, therefore, depends essentially upon the condition of the antagonist of the contracted muscle. The more powerful this is, the greater will be its ability to bring the eye into correct position after the division of the contracted muscle. But for the force of the muscles we have a sure gauge in the amount of the lateral excursion (abduction and adduction). If the muscular power is normal, the outer rim of the cornea should reach the external canthus in abduction, and the inner rim of the cornea the caruncle in adduction. We should determine the abduction and adduction in this way and also the absolute or relative near point of convergence (§ 308) before every squint operation; since by these means we can predict approximately the effect of the tenotomy.

981. Restriction of Effect.—If the effect of a tenotomy should turn out to be too great, there are the following means at our command to *diminish it*:—

1. When the suture is applied that is designed to close the wound in the conjunctiva, a wide and deep grasp is taken upon the conjunctiva, so that the needle is passed through Tenon's capsule as well. Then, when the knot is drawn tight, the tendon is drawn forward a little along with the conjunctiva.

2. If it is apparent that the divided muscle has suffered too great impairment of its action, its end must be grasped and attached again farther forward by means of stitches. This event occurs when the connection of the muscles with Tenon's capsule has been loosened to too great an extent, or when the operation has been done for a very slight strabismic deviation. When the operation itself is such as to be likely to produce an excessive effect — e. g., in a tenotomy made beyond the insertion and also in tenotomy of the superior and inferior recti—, it is well to insert a stitch before the tenotomy is made. If then the immediate effect of the operation is excessive, the excess can be remedied at once by drawing up the tendon with the stitch until the desired effect is secured. Otherwise, the stitch, untied and with ends left long, may be left in the wound for twenty-four hours to be drawn up in case the tendon slips.

982. Partial Tenotomy and Tendino-extension.—Differing in method of action from a total tenotomy are the various forms of partial tenotomy. In these the intention is to divide most of the fibres of the tendon in such a way that the tendon without losing its primitive attachment shall elongate somewhat, and thus become more slack.

Von Graefe in 1861 did partial tenotomy by making a central buttonhole in the tendon, carried nearly to the edge of the latter; and also by making a cross-cut in the tendon involving one edge and nearly the whole width. Stevens, adopting Von Graefe's first procedure, and operating subconjunctivally after Snellen's method (§ 979), made very extensive use of partial tenotomy in heterophoria. Other methods consist in making two cross-cuts in the tendon, one on each edge, opposite each other, and each

involving one-third the width of the tendon (Ziegler); a pair of such cross-cuts (Abadie); a pair of such cross-cuts combined with a central buttonhole (Verhoeff); two cross-cuts, one on each edge, but not opposite (Todd).[1] As a further modification, Ziegler combined a pair of cross-cuts according to his original method with a paring down of the central bridge of tendon, continued until the desired effect was attained.

Partial tenotomies have been done in various ways in order to correct a cyclophoria.

According to the translator's experience, the central buttonhole operation produces no material nor permanent effect unless the tenotomy is made practically total, i. e., severs the whole insertion. The graduation of the effect in this case is gotten by cautious division of the lateral attachments until the desired results as determined by repeated testing is obtained. With the other methods of partial tenotomy he has had no experience.

Partial tenotomy is done in low degrees of squint and in heterophoria of moderate amount. It seems to the translator that it should be restricted to tenotomies of the superior and inferior recti. In esophoria and esotropia of low degree, advancement (resection, or tendon tucking) of the external rectus should be done instead of a partial tenotomy of the internal rectus; and when tenotomy of the external rectus is indicated at all, it had better be complete.

983. Results.—The immediate result of tenotomy is greater usually than the final result. The more solid the union which the divided tendon forms again with the sclera, the stronger is the action which it can exert, and thus the effect of the operation diminishes in the succeeding four or six weeks.

As regards the *final result,* cases behave differently. The most frequent outcome is that the effect of the operation increases somewhat in the first few days, then diminishing again, and ultimately becoming less than it was directly after the operation. Sometimes the diminution proceeds so far that the effect of the operation almost completely disappears and the operation has to be repeated. This is particularly apt to be the case in exotropia, but it also occurs in esotropia when it has been operated on simply by advancement. In an operation by tenotomy the reverse frequently occurs—i. e., the effect slowly but steadily increases until finally a very disfiguring exotropia supervenes. This may take place even years afterward (and may occur without operation at all—as a result of the tendency of the eyes to become more divergent during the growing period see § 331). Unfortunately, it is impossible, either before or directly after the operation, to foresee with certainty which of these occurrences is going to ensue, so that we can not at once take measures to combat them.

Over-effect after tenotomy may be due to mangling of the tendon which makes it curl up and adhere to the conjunctiva instead of the sclera or which produces other untoward results. It is also apt to occur when a muscle is tenotomized for the second time. *Re-operation* on the same muscle should be avoided when possible, as the results are always uncertain.

Among the unpleasant results that sometimes accompany tenotomy is *sinking in of the caruncle,* which looks as if it had been drawn far back. This occurs only after tenotomy of the internus, and is due to the fact that the muscle as it retracts draws the conjunctiva of the inner half of the eyeball after it. This can be prevented if the conjunctival wound is closed with a suture and the conjunctiva thus kept in place.

Impairment of the cosmetic result may also be produced by dilatation of the palpebral fissure, giving the eye a staring aspect. This is usually ascribed to the fact that the divided rectus fails to retract the eye as usual, so that the eye protrudes somewhat and pushes the lids apart. The real reason for the dilatation, however, at least in most cases, is the retraction of the lid caused by the operation. For as the divided tendon recedes it pulls the lid back, too, by means of the slip of fascia attaching it to the latter. This retraction is particularly marked after tenotomy of the superior and infer-

[1] These methods are not given in chronological order. As Ziegler points out, Abadie followed Von Graefe, and Stevens, Abadie—these being succeeded in turn by Ziegler, Verhoeff and Todd.

ior recti. Per contra, after advancement of these muscles the palpebral fissure is *contracted* (see § 66). If the dilatation of the palpebral fissure is disfiguring it can be relieved by a tarsorrhaphy.

During the after treatment of a squint operation there is formed not infrequently a *nodule of granulation,* growing out of the sclera at the site of the conjunctival wound. Subsequently this becomes constricted at its base and ultimately falls off. It may also be snipped off readily with the scissors.

Serious accidents, such as suppuration of the wound, exudation into Tenon's space with protrusion of the eyeball, and actual panophthalmitis, can occur only when the wound has been infected during the operation. Such accidents are extremely rare. If we should perform the operation with a sharp pair of scissors, and at the same time are dealing with an unruly patient, it may happen to us to perforate the sclera. If the operation has been performed aseptically, this accident will, as a rule, pass over without producing any bad results. In general, the squint operation, if carefully performed, may be said to be perfectly free from danger; and it is one of the operations for which patients (especially those of the female sex) are most grateful.

(b) *Advancement of an Ocular Muscle*

984. Technique.—The term advancement may be applied in a general sense to any operation designed to enhance the action of an ocular muscle. A great many advancement operations have been proposed, of which only one or two, that have seemed to the translator the most serviceable, will be mentioned. They aim to effect their object either by attaching the tendon further forward, so as to increase the arc of contact of the muscle with the eyeball and also put the muscle itself more on the stretch (advancement proper); or by shortening the tendon and so putting the muscle on the stretch. Shortening the tendon may be effected either by cutting a piece out of it (resection) or by making a permanent fold in the tendon (tucking or folding operations). In many of the operations two of these methods are combined.

In all methods of operation the tendon should be fully exposed. This is usually done by a transverse incision not less than twelve mm. in length over the insertion of the tendon. In Worth's advancement a curved incision with its convexity directed toward the cornea and with its proximal point close to the latter is made (Fig. 453 F). In Landolt's advancement a semioval flap of conjunctiva with its apex close to the cornea is laid back. In advancements of the superior and inferior recti the translator believes that the best exposure is secured by reflecting a quadrantal conjunctival flap, the base of which, seven or eight mm. long, lies athwart the insertion of the tendon and the sides of which run out along the sides of the tendon for about ten mm. The tendon should be completely freed from any attachments to the sclera or other part, that might prevent its assuming the position required. Especially should the attachments to Tenon's capsule be divided by incisions carried well back along either margin of the tendon.

985. Advancement Proper.—In this the tendon is divided at its insertion, brought forward into a pocket made between the conjunctiva and sclera, and sewed to the latter close to the cornea. In doing this it is important not only that the conjunctiva should be dissected freely to form the pocket, but that all adventitious tissue in the latter that might interfere with a firm attachment of the tendon should be removed (Worth). Of the many methods employed, the following, which give excellent results, may be mentioned.

(*a*) *Worth's operation.*—A curved incision with its convexity toward the cornea is made through conjunctiva and Tenon's capsule (Fig. 453 F). The external lip of the

wound, including conjunctiva and capsule, is pushed back so as to expose the insertion of the tendon. A Prince's forceps is introduced with one jaw beneath the tendon, the other outside the outer lip of the wound so as to include in one grasp the tendon near its insertion and the cut edge of the capsule and conjunctiva. The tendon being thus securely held, is divided with its attachments close to its insertion. A single-armed suture is passed in through conjunctiva, capsule, and tendon, at *a* and out through capsule and conjunctiva at *b*. A similar suture is passed in at *c* and brought out at *d*. The ends at *c* and *d* are then crossed so as to make a half hitch, and the end with the needle is entered again at *e*, carried under the tendon to the cornea and brought out at *g*. The same is done with the upper stitch which is brought out at *h*. Before the stitches are passed forward to the cornea the outer lip of the wound, including the strip of conjunctiva, capsule, and tendon in the grasp of the forceps, is excised. The end at *g* is tied to the long end left at *c* and the end at *h* to the end at *a*. Conjunctiva, capsule, and tendon are thus brought forward together.

(*b*) A method which the translator has found to give excellent results is the *single-stitch operation* of Oliver (Fig. 453 *A*).

After exposing the tendon, a buttonhole is made in Tenon's capsule just above the tendon. A squint hook is passed through the hold and under the tendon, hugging the insertion. As the point of the hook emerges from beneath the lower side of the tendon, a buttonhole is cut over it in the capsule, so that the point comes through. A Prince's forceps (Fig. 452, *h*) is then substituted for the hook and is locked. The tendon is now divided right in front of the forceps, i. e., at the very insertion. Then the tendon is lifted away from the sclera and its lateral and inferior attachments are thoroughly divided, so that it can play freely backward and forward. Both needles of a double-armed thread are passed through the tendon well back and from within outward (at *g*, *h*) so as to include a good bit of its breadth in a loop, the bight of which is on the under surface of the tendon; then are carried under the conjunctiva and through the episclera, emerging close to the cornea some millimetres apart (at *i* and *l*). Then the small portion in the grasp of the forceps is excised, (or, if a greater effect is desired, a larger portion of the tendon is resected) and the two ends of the looped thread are gradually drawn tight so as to slide the tendon forward under the conjunctiva. The two ends are then tied together with a half hitch, and the result tested. If this is insufficient the knot is tightened, and when the result is satisfactory is tied securely.

(*c*) In *Landolt's advancement* two single-armed sutures are passed through the tendon from without inward, one coming out beneath one edge of the tendon, the other beneath the other edge. According to the effect desired, the sutures are either entered close to the insertion of the tendon and the latter divided there, or they are inserted further back and the portion of tendon in front of them is resected. Each suture after emerging beneath the edge of the tendon is carried forward and passed for several millimetres through episcleral tissue so as to emerge, respectively above and below and close to the cornea. Each suture is tied separately thus advancing the tendon to the corneal margin.

In *Wootton's* modification of this operation (Fig. 453 B), three double-armed sutures are passed, one through the middle of the tendon and one at each edge. The lateral sutures are applied before dividing the tendon, the two needles of each suture being passed from within outward so as to leave a bight on the under surface of the tendon. The tendon is then divided and lifted up, and the two needles of the central suture passed from without inward, first through the conjunctiva (*a*) then through the tendon. One needle of each lateral suture (*b*, *b*) and both needles of the central suture (*c*, *c*) are then passed through episcleral tissue.

Prince's method is shown in Fig. 453 C. Verhoeff's in Fig. 453 D and E.

986. Resection.—Of the resection operations the best is that devised by *Reese*.

In this, after the tendon has been exposed (Fig. 454 A) and separated from the conjunctiva and from the fascial attachments on either margin, the Reese muscle forceps (which resemble a Prince's forceps) is passed with one jaw over and one jaw under

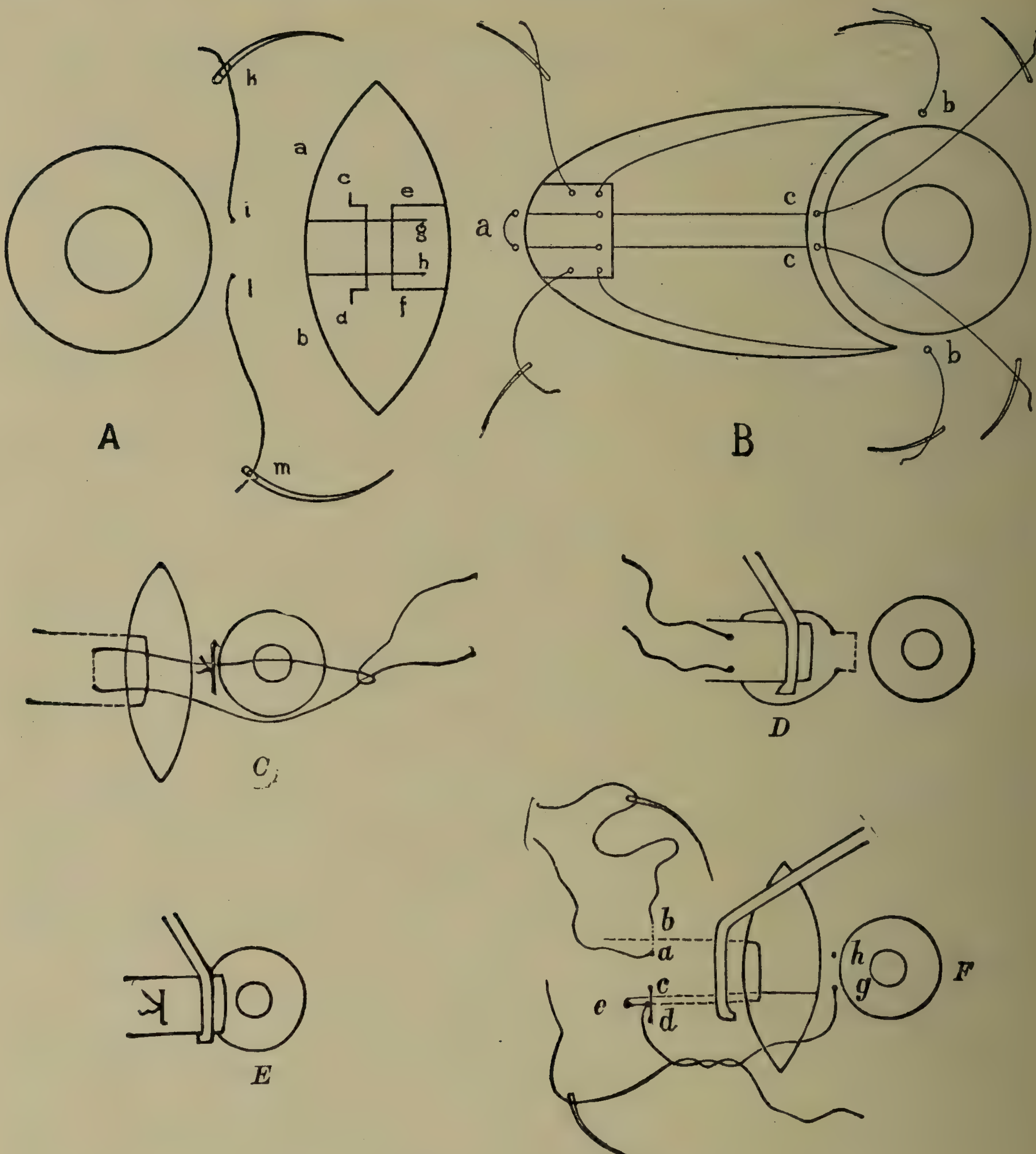

Fig. 453.—Advancement Operations. In part after Haab.

A. *Single stitch advancement.*—*ab*, inner lip of the wound in the conjunctiva; *cd*, tendon stump; *ef*, detached tendon; *g*, *h*, points of emergence of the two branches of the loop, the bight of which is on the under surface of *ef*; *i*, *l*, points of emergence of threads after passing over *ef* and *cd* and under *ab*; *h*, *m*, free extremities of threads, afterwards tied. See for description § 985.

B. *Wootton's modification of Landolt's advancement.* (see § 985).

C. *Prince's operation.*—A thread, designed to act as a pulley, is passed through the conjunctiva and episclera at the corneal margin and in a direction tangent to the latter. The tendon is then grasped and separated in the manner described in §984, and the two needles of a double-armed thread are passed from within outward through tendon, capsule, and conjunctiva, thus forming a loop on the under side of the tendon. One of the free ends is laid across the pulley stitch, which, as shown in the figure, is then tied over it. Traction is made until the tendon is in the proper position, and the free ends are then tied, one being under, the other over the pulley stitch.

D, E. *Verhoeff's operation.*—The tendon is grasped with a Prince's forceps and detached from its connections as described in § 984. A double-armed stitch is introduced into the sclera (previously exposed) at a distance of 1 mm. from the cornea and is passed for some 6 or 8 mm. through the superficial layers of the sclera in a direction at right angles to the axis of the tendon (vertical dotted line in Fig. D). The needle is reintroduced at the point of exit and carried a short distance in the sclera horizontally outward toward the tendon. The same is done with the other needle. Both needles are then passed through the tendon (Fig. D), the tendon is brought forward with the forceps (Fig. E) to the proper point, and the stitches are tied.

F. *Worth's operation.* (§ 985).

the tendon, three mm. from the insertion of the latter, and is clamped (Fig. 454 B). The tendon is divided one mm. from its insertion so as to leave a stump. Both needles of a heavy double-armed suture are introduced through the tendon from beneath, so as to form a loop, 2 mm. broad on its under surface, and are brought out through the conjunctiva at the outer lip of the wound (Fig. 454 C and D). To reinforce this central suture, single-armed lateral sutures are introduced, one above, the other below. These are entered in the conjunctiva at the outer lip of the wound a little above and below the points of emergence of the central suture and are then passed down through the upper

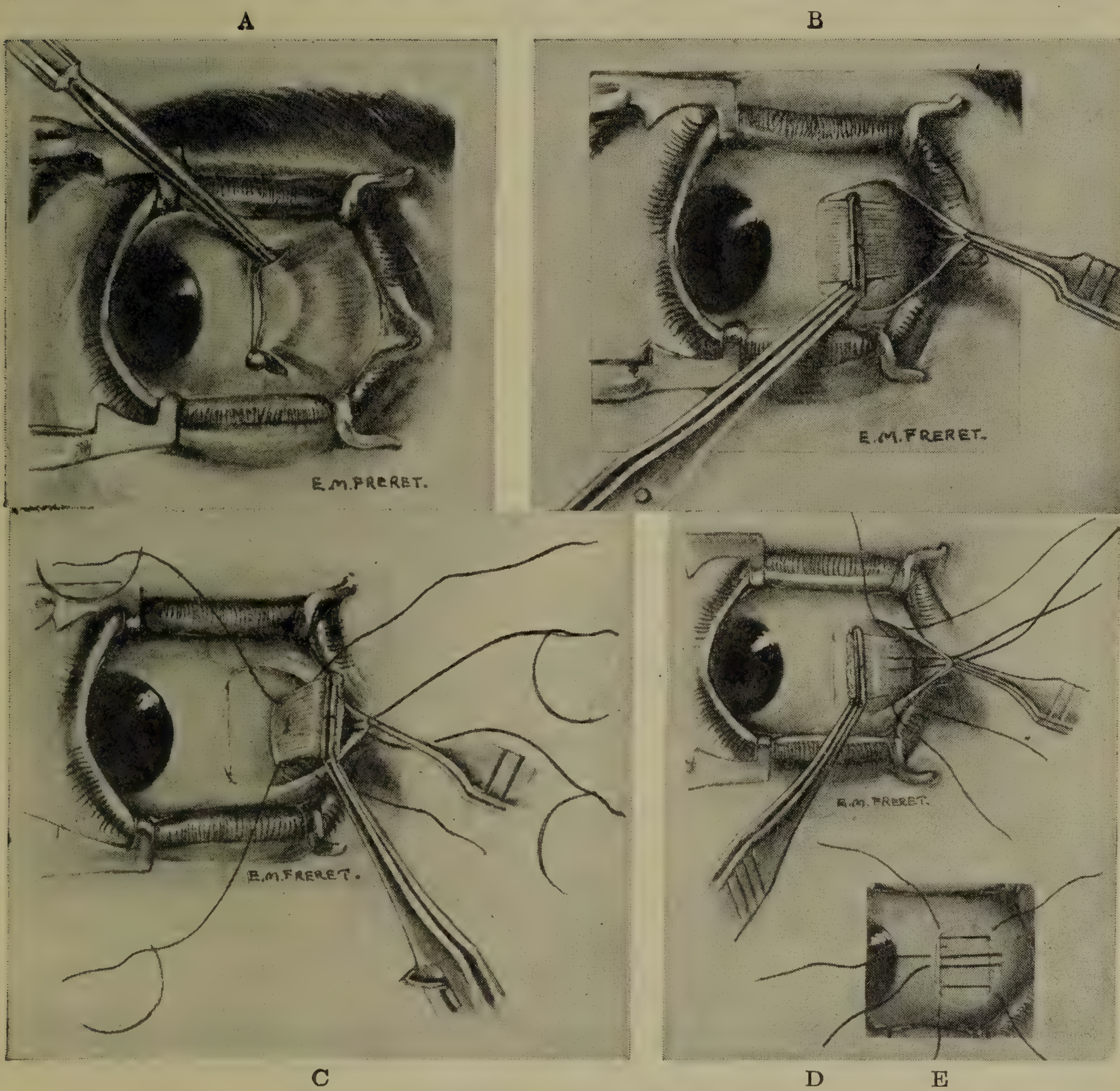

Fig. 454.—Resection of Tendon (after Reese).

and lower margins of the tendon, respectively (Fig. 454 C and D). First the central suture, then the lateral sutures are passed beneath the tendon stump, then made to transfix the latter, then brought out through the conjunctiva on the inner lip of the wound (see Fig. 454 E). All three sutures are tied thus not only advancing the shortened tendon up to the original insertion but also closing in the wound with conjunctiva.

The tendon stitches after an advancement or resection should be left in at least a week, and it is expedient to bandage both eyes for five or six days. Worth leaves his stitch in ten days and keeps both eyes bandaged all this time. Reese leaves his central stitch in 10 to 14 days.

987. (3) **Tendon Folding.**—In one variety of this operation the tendon is shortened by making a permanent tuck in it with some suitable instru-

ment (Savage, Valk, Todd). A combination of tucking and advancement, devised by *Bruns*, gives excellent results.

In this, the modified Clark hook (Fig. 452, *c*) is used. This hook has a middle prong which can be set below the level of the side prongs or raised above the latter to any desired extent. The tendon is exposed in the usual way, and the hook with its middle prong depressed is inserted in such a way that the middle prong is beneath, the side prongs above the tendon. The instrument is swept along the tendon to free it from any adhesions to the sclera. Then, keeping the hook six or seven mm. out from the insertion of the tendon, the operator by turning a screw in the handle of the instrument gradually raises the middle prong, carrying a fold of tendon with it. When this tuck is raised to the desired height, the centre of its base is transfixed by a needle in the middle of a long slender thread. The needle being drawn well through is cut off, leaving the tendon thus transfixed by two threads, each of which is then tied down over the corresponding edge of the tuck. This keeps the latter from flattening out when the hook is removed. (The threads may cause necrosis if tied too tight.) Through an eye in the centre prong of the hook a thread is carried, so that when the hook is withdrawn, the thread remains behind in the bight of the tuck. This thread, being armed at each end with needles, is passed forward into the episcleral tissue near the cornea and tied, thus laying the tuck flat and advancing it at the same time. By the amount of this advancement the effect of the operation can be regulated.

In *O'Connor's* operation the tendon is split by blunt dissection into a central and two outer strands. The central strand is separated from the insertion and laid back. Then a catgut loop is passed round each outer strand and tied, the result being that the strand is pursed up and shortened in much the same way that a saddle girth is cinched.

988. Tendon Transplantation.—Sometimes in complete paralysis of a muscle, the attempt is made to replace the lost power of the latter by connecting its tendon with that of an adjacent active muscle. This has been done with good results, especially in paralysis of the external rectus. The tendons of the superior and inferior recti are split and the outer sections of each after being separated from their own insertions are brought down and sewed into the tendon of the external rectus at its insertion.

989. Mode of Action.—By advancement, the insertion of the muscle is brought nearer the cornea, and thus there is given to it more power over the eye. The farther back the threads are passed through the muscle, and the farther forward the anterior end of the latter is attached, the greater is the effect of the operation. Later on, however, the effect diminishes, for which reason we gauge the operation so as to get a primary over-correction.

In resection and tendon-tucking the shortening of the tendon renders the muscle taut and thus increases its effectivenesss.

Worth finds that in the operation as he performs it no primary over-correction is required—the primary effect in this case being also the permanent effect. This seems also to be the case with the Bruns tucking-advancement.

Advancement not only reinforces the action of the muscle operated on, but, if extensive, also hampers the action of the opponent—sometimes to a marked degree. This is of particular importance in advancement of the superior rectus, which may so restrict the action of the inferior rectus as to produce considerable and annoying diplopia in the lower part of the field of fixation.

(*c*) *Muscle Operations in General*

990. Conditions Affecting Muscle Operations.[2]—There are certain conditions that have to be weighed in performing muscle operations.

1. Effect of Anæsthetic.—A general anæsthetic often disturbs the motor relations considerably. For example, a marked esotropia may disappear entirely under ether,

[2] In what follows the term "advancement" is used generically to cover advancement proper, resection and tendon-tucking.

so that if we judged by the appearances we should refrain from doing a sufficiently effective operation. Whenever possible, therefore, we ought to operate with a local anæsthetic, since with this measurements can be made during and directly after the operation and moreover give reliable indications. With some adroitness we can use local anæsthesia and apply these measurements even with children as young as six. If compelled to use a general anæsthetic, we should measure the deviation before administering the latter, compare this measurement with the appearances presented under complete anæsthesia, and make a corresponding allowance in operating. Thus, a patient with marked esotropia, but whose eyes looked straight under ether, would have to be put in a condition of well-marked divergence by the operation.

2. **Effect of Age.**—Since in some cases an esotropia tends to diminish up to the age of ten or twelve, some prefer to wait till then before operating—particularly, as the operation can then be done under a local anæsthetic. But by far the better plan is to do the primary operation, at least, early—as soon, in fact, as it is evident that other forms of treatment are no longer effective. The possible danger of a subsequent divergence need not bar an operation for esotropia, since such a divergence if it does occur can usually be rectified with ease. Only, in very young children we prefer as a safeguard to leave a slight amount of undercorrection, and incline to remedy a marked deviation by an advancement rather than a tenotomy.

3. **Effect of Glasses and Orthoptic Treatment.**—In gauging the effect of an operation we must always remember that the result may be materially modified by glasses. For this reason the tests during and directly after the operation should when possible, be made with the patient's glasses on. We may also in the case of an incomplete or excessive effect modify the result somewhat by changing the strength of the glasses, but especially we can modify it by orthoptic treatment (training of vision in an amblyopic eye and binocular training with the amblyoscope, red glass, prisms, and bar reading). Such orthoptic treatment should both precede the operation and should be resumed as soon as the bandage is removed.

991. Choice of Operation in Comitant Squint and Heterophoria.—The operations indicated in the different varieties of comitant deviation are as follows (translator):

If one of the two functions, convergence or divergence, is markedly excessive and the other nearly normal, reduction of the excessive action by tenotomy.

If one of the functions is deficient and the other normal, or but slightly excessive, reinforcement of the deficient function by advancement.

If one function is markedly excessive and the other deficient, advancement combined with tenotomy.

In applying these rules we must remember three things. First, a tenotomy acts by producing an actual insufficiency of the muscle whose over-stimulation we are aiming to correct. Hence, if pushed too far it may produce a detrimental weakness of action. This is particularly seen in tenotomy of the internal recti, which, if excessive, impairs the power of convergence. Second, a considerable advancement of one muscle may hamper the action of the antagonist. This is particularly seen in advancement of the superior rectus which may produce an uncomfortable limitation of movement and troublesome diplopia in the lower field. Third, in ordinary esotropia and exotropia the deviation really affects both eyes (see § 338). Hence, in the case of large deviations it is better to divide the operation between the two eyes.

It is clear from the above that the operation selected must depend not simply on the sort of deviation present—i. e. whether an esotropia or an exotropia—but on the essential character of the deviation and its amount. To ascertain these points we must before deciding on the operation determine, first, the amount of deviation for distance and for near points; second, the extent of rotation of the eyes, singly and in combination, in each of the six chief directions of the gaze; third, the screen deviation in each of these directions; fourth, the convergence near-point, absolute and relative; fifth, in cases in which we can determine it, the diverging power measured by prisms, base in (cf. § 310).

1. In *esotropia,* operation is indicated in all cases in which thoroughgoing and protracted orthoptic and refractive treatment has failed to produce an adequate correction. In the comparatively rare cases in which in spite of this treatment the condition is still mainly a convergence-excess (marked by excessive convergence and excessive adduction of one or both eyes, normal rotation outward, and a considerably greater deviation for near than for distance) tenotomy of one or both interni is proper. In cases of comparatively slight deviation (15-20 *d* or less) the operation may be limited to the deviating eye; otherwise, should be done on both eyes. If there is marked deviation in distant vision, advancement of the external rectus should be done and, if the deviation is considerable, should be divided between the two eyes. In high degrees of squint the advancement of the externus is better combined with a simultaneous tenotomy of the internal rectus, this being especially indicated in cases in which the deviation for near is even greater than that for distance and the adduction of the eye operated on and the converging power are excessive.

In esotropia especially of young children a moderate degree of convergent deviation may be left. In any event, we should be careful not to produce a crippling of the internal rectus (cf. § 979) nor a definite over-correction.

The same principles apply to *esophoria,* in which operation may be done whenever the symptoms are considerable and apparently can not be relieved by correction of the refraction and by orthoptic and general treatment. In esophoria, except in the very rare cases of a pure convergence-excess not relived by glasses, tenotomy should not be done but rather advancement of the externi.

2. In *exotropia* due to insufficiency of convergence (with deviation notably greater for near than for distance and remote or negative convergence near-point), advancement of one internal rectus or of both, depending on the amount of deviation, is indicated. The like is true of exotropia following a tenotomy of the internal rectus. Here the tenotomized muscle should be picked up, cleared of its attachments, and advanced. The single-stitch operation is an effective means of doing this. In cases of exotropia where there is a marked deviation for distance as well as for near, the advancement of the internus will in general have to be supplemented by a free tenotomy of the externus; and in the quite frequent cases of pure divergence-excess (marked deviation for distance with practically normal

relations for near) a tenotomy of one or both externi without advancement of the interni should be done.

Similarly in *exophoria*, tenotomy of the externi, advancement of the interni, or advancement combined with tenotomy is done, according as the condition is one mainly of divergence-excess, convergence-insufficiency, or a combination of the two.

Before correcting a lateral squint, whether convergent or divergent, it is important, especially in children, to ascertain whether there is any considerable vertical deviation present. Such vertical deviation due usually to congenital anomalies, is often the cause of the lateral squint (see §336), in which case the latter can not be relieved unless a *correction of the vertical deviation is first effected* by operation. When this correction has been secured the lateral squint may in some cases disappear of itself and in any event will yield as it would not otherwise to operative or orthoptic treatment.

Bilateral Operation.—When in a lateral squint or heterophoria the amount of deviation makes it advisable to divide the operation between the two eyes, the two operations may be done simultaneously or an interval of some weeks may be placed between them. The latter course may be adopted if the patient has no binocular vision. Otherwise, especially in the case of a double tenotomy, for exotropia, the translator believes that it is generally better to operate on both eyes at the same time, approximating to the correction as nearly as possible, and modifying the result later, if necessary, by an advancement. So also an advancement may be done on both eyes at the same time, the result being modified later by a tenotomy. But it would be unwise to do a combined advancement and tenotomy on both eyes at once.

3. *Hyperphoria* or *hypertropia* of such a degree as to require operation is usually non-comitant and should be treated according to the rules laid down in § 992. For a comitant hyperphoria or hypertropia the best operation is a tenotomy of the superior rectus so graduated as not to produce over-correction.

992. Operations for Paralytic Squint.—The operations done to relieve paralytic squint are:

1. Advancement of the Paretic Muscle.—This operation is highly satisfactory in cases of traumatic (including post-operative) paralysis, in which the muscle has the normal nerve power, but is ineffective because of faulty placement. In cases of true paralysis, unless the paralysis is quite incomplete, it at most produces a cosmetic improvement in the primary position, but does not increase the rotating power so that in the outlying direction of the gaze the deviation is still apparent.

In paralysis of an oblique, advancement of the muscle itself is technically impracticable, hence, Landolt advises advancement of the corresponding rectus in the same eye, e. g., of the inferior rectus in the case of paralysis of the superior oblique. The translator prefers tenotomy of the associate or of the direct antagonist (see 2 and 3 below).

2. Tenotomy of the Direct Antagonist.—As a remedy for true paralysis this is usually ineffective and if pushed too far does harm by producing the effect of two paralyses instead of one. It may, however, be used with advantage to supplement advancement of a paretic muscle when the antagonist to the latter is much contractured, as evidenced by the persistence of the deviation and the diplopia far into the field of action of the latter. Furthermore, the diplopia and deviation in an incomplete paralysis of the superior oblique may be materially relieved by tenotomy of the inferior oblique in the same eye. This operation is indicated if there is evidence of overaction of the latter muscle (up-shoot of the deviating eye in adduction).

3. **Tenotomy of the Associate.**—This aims to obviate the deviation and diplopia by limiting the movement of the sound eye in the same way that the paralysis limits the movement of the other. Suppose, for example, that the right superior oblique is paralyzed. This muscle normally moves the right eye down, abducts it (carries it to the right), and rotates its vertical meridian inward (to the left); and if it is paralyzed, the movements of the right eye are restricted in all these senses. Diplopia, therefore, results, which we can prevent if we can restrict the movement of the left eye in the same sense and to the same extent. This we can do by tenotomy of the left inferior rectus, which moves the left eye down, adducts it (moves it to the right), and rotates its vertical meridian outward (to the left). Moreover, as this parallelism of action of the superior oblique of one eye and the inferior rectus of the other holds good for all directions of the gaze, the operation affords complete compensation for the paralysis. A paralysis of the right inferior oblique would similarly be compensated for by a tenotomy of the left superior rectus, and one of the right superior rectus by tenotomy of the left inferior oblique. This latter operation is particularly indicated in congenital paralysis of the superior rectus, in which it gives excellent results, as it does also in some cases of traumatic paralysis of this muscle. In paresis of the externus, advancement of this muscle, combined with tenotomy of the internus in the other eye, is indicated, and will usually have to be supplemented by tenotomy of the internus in the same eye. So in paresis of an internus, its advancement combined with tenotomy of one or both externi is usually required.

4. **Transplantation.**—This (see § 988) has been done especially in paralysis of the external rectus.

II. Enucleation of the Eyeball

993. Simple Enucleation.—Enucleation consists in shelling the eyeball out of Tenon's capsule, the conjunctiva and all the tissues adjoining the eyeball being left behind. This operation was suggested by Ferrall in 1841 and about the same time by Bonnet upon the basis of his studies upon Tenon's capsule (which hence is also called Bonnet's capsule).

In Bonnet's operation, the recti muscles are allowed to retract after having been severed from the eye, so that they exert little or no effect on the conjunctival bed and hence on the artificial eye. The mobility of the latter is much increased if the muscles are advanced to the edge of the conjunctival wound. This is well effected in De Schweinitz's modification, which is as follows:

The lids are separated by a spring speculum. The conjunctiva is divided with the scissors by a circular cut made all round the cornea and close to it. The incision is carried into Tenon's capsule exposing the recti tendons. Each of the latter is picked up, separated from the sclera close to its insertion, and brought forward to the edge of the conjunctival wound to which it is attached by a suture. Then the speculum is pushed into the orbit so as to make the eye protrude, and the eye is drawn forward and turned outward, The enucleation scissors (Fig. 452, g) is now introduced close to the nasal side of the eyeball till its points touch the optic nerve which is felt as a hard cord. The blades of the scissors are now opened and the nerve divided. As soon as this is done the eye can be pulled out of the orbit (luxated) in front of the lids. Then the two oblique muscles and remaining structures attached to the eyeball are divided as close to the latter as can be, and the eye is removed. A wound cavity is now presented, which is bounded behind by Tenon's capsule, in front by the detached conjunctiva

of the eyeball. Through the margin of the latter, which corresponds to the limbus of the conjunctiva, interrupted sutures are passed which are made to include Tenon's capsule. A firm pressure bandage is applied to press the conjunctiva against Tenon's capsule and to check the bleeding.

In Arlt's or the so-called Vienna operation, either the external or internal rectus was first divided, the stump grasped with forceps so as to steady the eye, and each of the other recti in succession divided with the scissors without being picked up on the hook.

994. **Enucleation with Implantation in Tenon's Capsule.**—In order to make a better and more movable support for an artificial eye, many after enucleation implant in the cavity between Tenon's capsule and the conjunctiva a glass, gold, or silver ball or in some cases a mass of fat. The fat often shrinks, and the balls are often expelled after a time, so that the operation is not permanently successful. Expulsion of the ball may be prevented if the conjunctiva alone is sewed over the ball, the muscles being allowed to retract (Verhoeff, Sweet). If a glass ball is used it should be lead-free, as otherwise it is attacked by the orbital juices. In successful cases the result of implantation is very satisfactory.

In Lagrange's *anaplerosis* a mass of fat taken from the temple is pushed through a subcutaneous tunnel into the depth of the orbit, where it is held in place, while still connected with its original site by a pedicle.

995. *Healing* takes place after enucleation without suppuration and by primary union. The cavity which remains after the removal of the eyeball is lined by Tenon's capsule, the raw, inner surface of which becomes attached to the raw surface of the detached bulbar conjunctiva.

Enucleation, if performed in an aseptic manner, is an operation perfectly devoid of danger. For purposes of anæsthesia we may employ either general narcosis or local anæsthesia applied deep in the orbit (see § 893). The hæmorrhage ordinarily is slight, so as to require no other measures for its arrest than the application of a pressure bandage upon the closed lids. In case the bleeding is more profuse, a tampon of iodoform gauze must be introduced into the orbit itself. Under normal conditions the operation wound heals within less than a week. Purulent inflammation (phlegmon) of the orbital tissue occurs after enucleation only when the wound has been infected. (For meningitis after enucleation, see § 758.)

The *prothesis* (*artificial eye*) should not be inserted sooner than fourteen days at earliest after the operation. It consists of a shell of glass, which is made in imitation of the anterior division of the eyeball, and which is retained in place behind the lids. After an enucleation which has healed in the normal way there is found a cavity clothed with conjunctiva, which, behind the upper and lower lid, is converted into a deep furrow corresponding to the fornix conjunctivæ. It is into this furrow that the upper and lower rims of the artificial eye are inserted. The deeper the furrow is, the better will it keep the artificial eye in place. For this reason we take care in operating to preserve the bulbar conjunctiva as much as possible. In cases in which we are compelled to remove part of the conjunctiva, the portion which remains may be drawn into the cavity by cica-

trization, so that the fornix is made proportionately shallower. In this way it may become impossible for an artificial eye to be worn. In such cases a new cul-de-sac may be fashioned, deep enough for the reception of the artificial eye (see § 916). The artificial eye moves conjointly with the other eye, although its excursions are smaller; for the ocular muscles, although detached from the eyeball, still retain their connection with Tenon's capsule. They move the latter in the same direction that the other eye is moving in, and with Tenon's capsule they move both the conjunctiva which lines it and the artificial eye which rests upon the conjunctiva.

The *artificial eye* should be taken from the orbit every night and be well cleaned. In time it loses its lustre, and must then be replaced by a new one. It not infrequently happens that the conjunctiva is thrown into a state of catarrhal inflammation by the mechanical irritation which the artificial eye sets up. Then the wearing of the latter must be restricted to a few hours each day, or given up altogether for a while, and the conjunctival catarrh must receive appropriate treatment. But the opposite of this state of things also occurs—namely, that, through wearing an artificial eye, troubles that were formerly present are made to disappear. This is the case when, after an enucleation, the lids sink back and an entropion develops, in consequence of which the cilia, which are directed inward, irritate the conjunctiva. By the insertion of an artificial eye the lids receive support; the entropion disappears, and with it vanishes the condition of irritation in the conjunctiva.

The artificial eye may be worn not only in an empty orbit but also over the eyeball. The only prerequisite is that the eyeball shall be diminished in size, either as a whole through atrophy or phthisis, or at least in its anterior division through applanatio corneæ, or as a consequence of ablation of a staphyloma of the cornea. An artificial eye when resting upon the natural eyeball has a particularly deceptive and natural appearance, and also moves quite perfectly with the eye which lies beneath it. Unfortunately, the stump of an eye does not always bear an artificial eye over it. It may become irritated by the latter so as to get inflamed and painful. In such cases either the artificial eye must be laid aside or the too painful stump must be enucleated.

In an empty orbit the ordinary shell-like artificial eye sinks too deep and does not move very well. Hence, for such cases, eyes have been manufactured which are convex posteriorly (reform eyes of Snellen).

996. Indications.—The indications for enucleation are:

1. *Malignant tumors* in the eyeball, and also those on or behind it, provided they can not be removed radically by a less thoroughgoing operation with retention of the eyeball. In tumors which develop in the posterior section of the eyeball (gliomata of the optic nerve and sarcomata of the chorioid) there is a possibility that the new growth is in process of transmission backward along the optic nerve. In such cases, therefore, the optic nerve is not divided close to the eye, but as far back as possible. After the enucleation has been performed, the cross section of the piece of nerve attached to the eyeball is examined. If it should prove to be attacked by the new growth, the portion of the optic nerve which has been left in the orbit must also be sought for and exsected.

2. *Injuries.* Enucleation is performed at once (primary enucleation) when such a considerable injury is present that the eye is lost beyond peradventure. This is the case in extensive laceration of the anterior portion of the eyeball, with escape of a part of the contents of the globe. By enucleation in such a case we spare the patient the panophthalmitis that otherwise awaits him, or the tedious and painful process of shrinking of the eye.

If the injury is of such a character that the preservation of the eye, at least so far as its form is concerned, is not altogether out of the question, we try first to save the eye by initiating that form of treatment which is indicated by the nature of the injury. If, nevertheless, inflammation develops, and the vision of the eye is absolutely abolished, the indication then is to perform enucleation (secondary enucleation) in order to prevent sympathetic inflammation of the other eye. Enucleation should also be performed upon those eyes which have been blinded by inflammation in consequence of an unsuccessful cataract operation.

3. *Irido-cyclitis, atrophy of the eyeball,* and *phthisis bulbi* furnish an indication for enucleation whenever sympathetic ophthalmia threatens to occur, or has already broken out. And even a condition of painfulness in the eye which can not be relieved in any other way demands the performance of enucleation, provided all hope for retaining or restoring a serviceable degree of vision has disappeared.

4. *Glaucoma absolutum,* when it is associated with continual pain, and when other less radical operations have either been already performed without success or are impracticable.

5. *Ectasia of the eyeball.* When the eyeball is very much increased in size either by large staphylomata of the cornea or sclera or by hydrophthalmus, it torments the patient by giving rise to continual attacks of irritation, by preventing the closure of the lids, and by producing disfigurement. Enucleation is then indicated, provided the eyeball can not be diminished in size in any other way (e. g., by a staphyloma operation).

6. *Hæmorrhage* which comes from an eye that has been operated upon or that has been ruptured, and which can be arrested in no other way.

7. *Cosmetic considerations* sometimes furnish the indication for the removal of a blinded and very disfiguring eye, so as to allow an artificial eye to be worn in its stead.

997. Substitutes for Enucleation.—Not infrequently simple enucleation is replaced by other operations either because some condition is present like panophthalmitis or orbital cellulitis in which the performance of enucleation may, according to the current belief, produce a septic meningitis; or because of the desire to get a better and more movable stump for the artificial eye. These substitutes have been used in all the conditions given in § 996 except in the case of malignant tumors, in which obviously nothing can replace enucleation.

1. **Partial enucleation.**—In this operation devised by Lister, the cornea is removed, the eye scooped out, and all the sclera cut away except a frill about the optic nerve. Lister believes that since the optic-nerve sheath is not opened, the danger of a septic meningitis is averted. The operation, it will be seen, is the direct converse of that of Dimitry (see below).

2. **Evisceration of the Eye.**—This operation (also called *exenteratio bulbi*) is performed as follows (A. Graefe). The cornea with an adjacent zone of sclera is removed by incising the sclera near the limbus with a knife, and then detaching it by a circular cut with the scissors. Then the contents of the eyeball which has thus been opened are scooped cleanly out with a sharp spoon, so that the inner surface of the sclera lies exposed. Lastly, the opening is closed again by means of sutures passing through the conjunctiva and the cut edges of the sclera. In this way a stump is obtained which, however, shrivels

up so much in the course of time, that it scarcely has any value as a support for the artificial eye. Hence, the attempt has been made to get a stump that shall remain large permanently, by introducing into the empty scleral capsule a chemically indifferent, sterilized foreign body, such as a glass or gold ball, and allowing it to remain there permanently (*Mules's* operation). Unfortunately the balls inserted in Mules's operation are not infrequently extruded after a time. To prevent the forward push that leads to this extrusion, *Dimitry* cuts out a portion of the sclera at the back part of the eye, including the head of the optic nerve, and then inserts a gold ball. The results of this operation are good and permanent.

Evisceration has been done particularly in cases of panophthalmitis—being in this regarded (probably without good reason) as safer than enucleation (§ 758). In other conditions it seems to hold no advantages over enucleation with implantation in Tenon's capsule. This is particularly the case if sympathetic ophthalmia threatens, since the protection afforded against this by even a carefully performed evisceration is not as complete as that afforded by enucleation.

3. **Optico-ciliary Neurotomy.**—In the endeavor to be as conservative as possible, observers have also tried to replace enucleation by the division of the nerves going to the eye. This operation is optico-ciliary neurotomy (Boucheron, Schöler). First the conjunctiva over the internal rectus and then the muscle itself are divided. From the wound thus made the scissors are passed backward to the optic nerve, which is divided as far back as possible. It is then possible to rotate the eyeball so far outward that its posterior segment together with the stump of the optic nerve shall appear in the wound. The portion of the optic nerve still attached to the sclera is then removed close to the latter, so that, provided the nerve was divided well back the first time, a long piece of it is resected. Next, the posterior section of the eyeball as far forward as the equator is freed from all tissues attached to it, in doing which most of the ciliary nerves are divided. Then the eyeball is returned to its place in Tenon's capsule, and fixed there by uniting the divided ends of the internal rectus and the divided conjunctiva with sutures. After the operation is completed a pressure bandage is applied.

Neurotomy is suggested as a substitute for enucleation when we are dealing with eyes that should be removed on account of threatening sympathetic inflammation or on account of pain. Neurotomy, however, has not proved perfectly reliable. Sympathetic inflammation has repeatedly occurred after it, so that when there is a danger of such inflammation absolutely nothing else but enucleation is indicated. Pain, too, sometimes recurs after neurotomy. Added to this is the fact that this operation is more difficult to perform than enucleation and requires a considerably longer period of healing. It is, therefore, indicated only when there is no danger of sympathetic inflammation, as for example in absolute glaucoma, and when the patient is unwilling to consent to an enucleation.

III. Removal of Orbital Tumors

998. In the removal of retrobulbar tumors the eyeball is in the way and prevents access to the back part of the orbit. If, nevertheless, we wish to perform extirpation of the tumor with preservation of the eye, there are two ways open to us. In cases in which marked exophthalmus has been present for quite a long time, the eye muscles and the optic nerve are very much stretched. Then it is usually easy, after dividing the conjunctiva on the temporal side and also the external rectus, to shove the eye toward the nose far enough for us to be able to get into the deeper parts between the eye and the temporal wall of the orbit. To get the lids out of the way and to secure more room, the external commissure may be divided and the lids turned back. When the eye can not be displaced sufficiently in this way we procure access to the back part of the orbit by making a *temporary resection of the outer wall of the orbit* (Wagner, Krönlein).

We first make an incision through the skin at the orbit's outer margin. This incision begins at the linea semicircularis of the frontal bone, passes in a slightly convex curve downward in front of the outer border of the orbit, and turns outward at the level of the upper border of the malar bone (Fig. 455, *ab*). Where the section runs over the outer margin of the orbit, it is carried through the periosteum down to the bone and then the periosteum is detached from the outer wall of the orbit backward and downward as far as the inferior orbital fissure. Then we divide the periosteum at the two places where the outer margin of the orbit is to be chiselled through; i. e., above the zygomatico-frontal suture (*c*), for one, and just above the place where the zygomatic arch is given off, for the other. Starting from these two points, we chisel through the orbit's outer wall, converging the two lines of incision (the two heavy black lines in Fig. 455) as we go backward so that they meet in the outer end of the inferior orbital fissure. The triangular portion thus defined in the orbital wall is then swung outward, and thus the back part of the orbit is exposed. When the operation is completed the displaced segment of bone is swung back into position again.

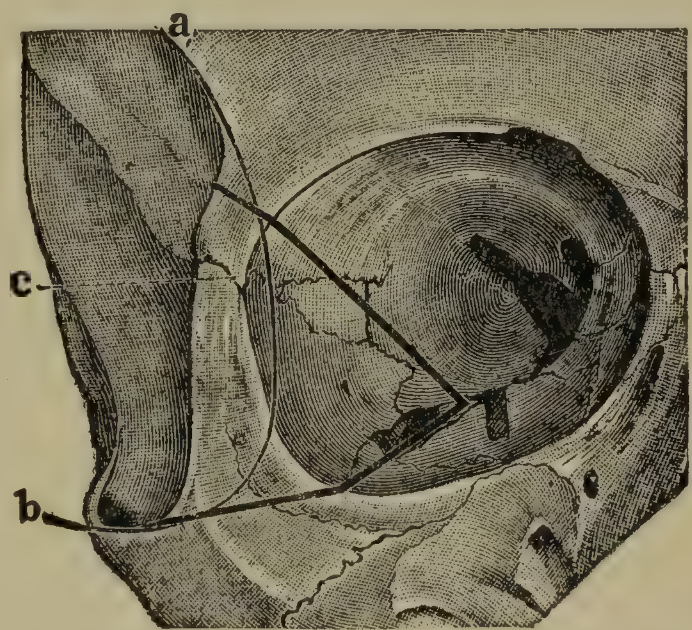

Fig. 455.—Temporary Resection of the Outer Wall of the Orbit. (After Haab.)

Krönlein's operation is also done to expose the sclera in doing Müller's operation for detachment (§ 977), and sometimes to gain access to the ophthalmic veins when these are ligated (§ 881).

999. Exenteration of Orbit.—We ought not to push too far our efforts to preserve the eye. If after extensive extirpation, the eyeball would have to be left denuded, it would subsequently be destroyed by suppuration. After removal of the entire retrobulbar tissue the eye would sink back deep into the orbit, would be fixed there by scar tissue, and would be quite useless. In such cases it is better to sacrifice the eye in the beginning, even if it should be normal, since by so doing the extirpation of the tumor can generally be done more quickly and with more precision. In such cases and also when the eye itself is involved, we remove the eye and the retrobulbar tissue in one mass—*exenteration of the orbit.* We begin by splitting the outer commissure of the lids out as far as the external margin of the orbit. By this procedure the lids are made freely movable, and can be turned back the one up, the other down, so as to admit as free access as possible to the orbit. Then the soft parts behind the everted lids are divided with the scalpel down to the bony margin of the orbit. From this as a starting point the periosteum is detached from the bone all round down to the apex of the orbit. The entire contents of the orbit now form a wedge which lies free in the latter, and is attached only at the optic foramen by means of the optic nerve and the ophthalmic artery. This pedicle is now divided and the bleeding cut surfaces are cauterized with Paquelin's thermo-cautery or with the galvano-cautery, since ligation of the ophthalmic artery is technically impossible. Next, all shreds of tissue still attached to the bone are removed, so that the latter is completely denuded. Then the orbit, after suitable irrigation with a disinfecting fluid, is packed with tampons of iodoform gauze, and a light pressure bandage is applied over it.

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